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An Examination of the Mechanisms of Action in Cognitive Behavioral Therapy for Bulimia Nervosa

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Cognitive-behavioral therapy (CBT) for bulimia nervosa (BN) has received considerable empirical support for its efficacy. However, few investigators have examined the mechanisms proposed to account for the reduction of BN symptoms during CBT. The current study examined the associations between therapist interventions, client mechanisms, and symptoms during treatment in a sample of 56 clients undergoing CBT for BN. Results suggested that behavioral interventions were most associated with symptom change during treatment whereas relational interventions were most associated with change in client mechanisms such as client engagement. Additionally, some changes in BN symptoms were mediated by changes in proposed client mechanisms whereas others were directly associated with therapist interventions. Implications of these findings for CBT theory of BN treatment and CBT treatment process research methodology are discussed.

A number of controlled treatment studies have documented the efficacy of cognitive-behavioral therapy (CBT) for bulimia nervosa (BN; see Lewandowski, Gebing, Anthony, & O’Brien, 1997; Wilson & Fairburn, 1998, for reviews), and a recent meta-analysis indicates CBT as the treatment of choice for BN (Whittal, Agras, & Gould, 1999). Despite this widespread support for its efficacy, few studies have examined how CBT works or identified the mechanisms of action of CBT for BN (Craighead & Agras, 1991; Wilson, 1999; Wilson & Fairburn, 1993). As noted by Wilson and Fairburn (1993) “there has been little experimental work of relevance to its action, and the findings

Scott Baldwin is now at the University of Memphis.

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of treatment studies provide only indirect evidence regarding mechanisms” (p. 265).

CBT approaches to treatment are based on theoretical models that include several types and levels of cognition. Distinctions are drawn between how information is organized, processed, and its content (Vitousek & Hollon, 1990). In terms of thought content, an additional distinction is drawn between dysfunctional beliefs and automatic thoughts. Automatic thoughts are situation-specific, moment-to-moment, unplanned thoughts that often reflect processing biases and dysfunctional content. In contrast, dysfunctional beliefs are more stable, cross-situational beliefs that are posited to give rise to automatic thoughts and processing errors, and to influence the attention to, interpretation of, and behavioral response to stimuli (Clark & Beck, 1999). Several BN models identify the strong endorsement of dysfunctional attitudes about body appearance as a critical factor in the precipitation and maintenance of BN symptoms (M. Cooper, 1997; Fairburn, 1997; Spangler, 1999, 2002). According to these models, strong belief in such dysfunctional convictions (e.g., my relationships depend upon how I look) leads to valuing thinness, overt body criticism, body dissatisfaction, and attempts to control and alter body shape and weight via dietary restriction and rigid eating rules. The restrictive eating pattern results in both physiological and psychological deprivation, which increases susceptibility to binge eating. Binge eating activates dysfunctional beliefs about the body and increases anxiety about predicted weight gain. Purging follows binge eating as an attempt to compensate for the calories consumed during binge eating and to reduce anxiety. Lastly, a rededication to restrictive eating follows the binge-purge episode in an attempt to regain a sense of self-control and as a behavioral recommitment to dysfunctional beliefs about the importance of body appearance and thinness (see Fairburn, 1997; Spangler, 1999; Wilson, Fairburn & Agras, 1997, for expanded descriptions of this cognitive-behavioral model of BN).

Based on this model of BN, Fairburn (Fairburn, 1981; Fairburn, Marcus, & Wilson, 1993) developed a CBT protocol that is divided into three phases, each of which focuses on specific variables in the model (described above). The primary goal of Phase 1 is to reduce and eventually eliminate all forms of excessive dietary restriction. The primary goal of Phase 2 is to modify client dysfunctional beliefs about body shape and weight. The final phase focuses on maintenance of treatment gains and relapse prevention (see Fairburn, 1997; Spangler, 1999; Wilson et al., 1997, for detailed descriptions of CBT for BN). A number of interventions or components of CBT are employed by therapists to attain these treatment goals. Phase 1 interventions include explaining the CBT model of BN and thereby providing a rationale for interventions, self-monitoring of eating pattern and of weight, psychoeducation, prescription of regular eating, meal planning, devising alternatives to binge and purge behaviors, stimulus control, and exposure to feared foods. Phase 2 interventions include self-monitoring of thoughts and beliefs (particularly body-related), evaluation of thoughts and beliefs, and enhancing cognitive
flexibility and problem-solving skills. Phase 3 interventions include reviewing changes made during treatment, distinguishing between a lapse and relapse, identifying high-risk situations, and developing a relapse plan. Additionally, the CBT protocol for BN incorporates relational and structural elements throughout all phases of treatment. The relational interventions include collaboration, empathy, involvement, and feedback to convey understanding. The structural interventions include agenda-setting, homework assignment, pacing the session, and summarizing.

Thus, within this CBT approach to the treatment of BN, distinctions can be drawn between treatment mechanisms and manipulations. According to the terminology of Hollon and Kriss (1984), a treatment mechanism is defined as a client variable that directly produces a clinical outcome of interest. In BN treatment, mechanisms would be characteristics of clients that change during treatment—for example, dietary restraint or beliefs about the body—that produce reduction in BN symptoms such as binge eating, purging, or body weight and shape concern. A treatment manipulation is defined as an intervention introduced by the therapist as a catalyst for client change. Hollon and Kriss (1984) note that several types of therapist characteristics can be classified as treatment manipulations, including types of therapist activities (e.g., meal scheduling, cognitive restructuring), quality of delivery, and therapist interpersonal style in which manipulations are delivered and embedded. To ascertain how a treatment works, both client mechanisms and therapist manipulations must be investigated: “Mechanism variables can be likened to a ‘toothed cog’ interfacing with treatment manipulations on the one hand, and outcomes on the other” (Hollon & Kriss, 1984, p. 43). We thus propose a three-stage model wherein treatment manipulations influence treatment mechanisms, which, in turn, influence symptoms.

According to CBT theory and practice, several mechanisms of CBT for BN are possible. One hypothesized mechanism of CBT for BN is change in client cognition, in particular dysfunctional beliefs about appearance. According to CBT theory, as dysfunctional beliefs about the importance of body appearance are reduced, the desire for thinness, negative automatic thoughts about the body, level of body concern, and engagement in dietary restriction are consequently reduced. Additionally, client change in restrictive eating behaviors is another central hypothesized mechanism of CBT. Specifically, reducing dietary restraint is hypothesized to directly reduce binge eating. It is further predicted that as clients eat more regularly and binge eating is reduced, purging is also reduced since purging is thought to be a consequence of perceived binge eating. Table 1 lists some of the primary hypothesized mechanisms of CBT for BN.1

As outlined by Spangler (2003), there are multiple BN treatment manipulations that are proposed to influence change in the client mechanisms of

1 There are additional plausible mechanisms of CBT for BN. See Wilson and Fairburn (1993) and Mizes (1985) for a discussion of other possible mechanisms.
**TABLE 1**

**HYPOTHEZIZED TREATMENT MANIPULATIONS, MECHANISMS, AND SYMPTOMS IN CBT FOR BN**

<table>
<thead>
<tr>
<th>Treatment Manipulations</th>
<th>Treatment Mechanisms</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Behavioral interventions</td>
<td>Client dietary restriction</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Cognitive interventions</td>
<td>Client body-related beliefs</td>
<td>Binge eating</td>
</tr>
<tr>
<td>Relational interventions</td>
<td>Client engagement in treatment</td>
<td>Shape and weight concern</td>
</tr>
<tr>
<td>Structural interventions</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

dietary restraint, beliefs about appearance, and engagement in treatment. In the Fairburn treatment protocol, psychoeducation, cognitive-behavioral explanation of BN, and relational interventions provided by CBT therapists are thought to contribute to client hope, motivation, and client engagement in treatment. Psychoeducation and specific behavioral interventions such as prescription of regular eating, meal planning, and exposure to feared foods are predicted to contribute to changes in client dietary restriction and eating habits during treatment. Monitoring of cognition and therapist interventions to explore and modify cognition, as well as body-related behavioral experiments and psychoeducation, are hypothesized to serve as catalysts of change in client beliefs about and investment in body appearance during treatment. In contrast, Phase 3 interventions are hypothesized to impact the maintenance of cognitive and behavioral changes after (rather than during) treatment, and are thus predicted to be related more to long-term rather than acute treatment outcome. Table 1 lists the primary hypothesized treatment manipulations of CBT for BN.

In the few studies that have attempted to examine mechanisms of action in CBT for BN, a common approach has been to compare changes in client cognitive, behavioral, and BN symptom variables occurring during CBT versus other forms of treatment (Fairburn et al., 1991; Jones, Peveler, Hope, & Fairburn, 1993; Wilson et al., 1999) or dismantled versions of CBT (Fairburn et al., 1991; Thackwray, Smith, Bodfish, & Meyers, 1993; Wilson, Rossiter, Kleifield, & Lindholm, 1986). These studies are based on the premise that if CBT works as hypothesized, it should evidence more or quicker change in particular client cognitive, behavioral, and symptom variables as compared to other forms of treatment that are assumed to work through alternative mechanisms. Consistent with prediction, CBT for BN has been shown to have a significantly

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2 Body-image exposure is one form of body-related behavioral experiment that could be used to challenge beliefs about the body. Anxiety models of BN (e.g., Mizes, 1985) emphasize the importance of body exposure for treatment effectiveness. However, since we did not single out body exposure as a required intervention in the study treatment protocol, we cannot address the unique effect of a body exposure intervention on client mechanisms or symptoms.
more rapid time course in decreasing binge eating and purging than alternative forms of treatment such as interpersonal psychotherapy (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Fairburn et al., 1991; Jones et al., 1993), supportive-expressive therapy (Garner et al., 1993), and psychodynamic therapy (Walsh et al., 1997; Wilson et al., 1999), suggesting the possibility of different mechanisms of action. Additionally, CBT tends to produce greater amounts of change in client cognitive variables such as body image, eating attitudes, and attitudes toward body shape and weight than other forms of treatment or dismantled (purely behavioral) versions of CBT (Fairburn et al., 1991; Garner et al., 1993; Jones et al., 1993; Rosen, 1996; Thackwray et al., 1993; Whittal et al., 1999).

However, although these findings are consistent with theory about how CBT for BN works, they are not sufficient to determine whether CBT effects change through the cognitive and behavioral methods hypothesized, since the changes that occur in client cognitive and behavioral variables may not have been produced by therapist cognitive or behavioral interventions. Additionally, the proposed mechanisms of change may have no effect on the reduction of BN symptoms. It is possible that clients treated with CBT may change their eating behaviors and beliefs about the body, but the improvement in BN symptoms could result from other factors. Furthermore, different treatments could affect BN symptoms via similar or different pathways regardless of mean levels of particular client cognitive or behavioral variables posttreatment (cf. Hollon, DeRubeis, & Evans, 1987; Wilson & Fairburn, 1993). Thus, a comparison of levels of BN symptoms or particular cognitive or behavioral variables posttreatment does not indicate the client mechanisms that are associated with symptom recovery, nor does it indicate the therapist manipulations that are associated with changes in client mechanisms. Specific and repeated measurement of proposed client mechanisms and therapist manipulations must be conducted during treatment in order to study the associations between these factors and symptom change.

The purpose of the present study was to examine the association during treatment between BN symptoms (e.g., vomiting, body weight and shape concern) and the hypothesized mechanisms and manipulations of CBT for BN. For treatment manipulations, we hypothesized that during treatment: (a) therapist behavioral interventions (e.g., meal scheduling) would be most strongly associated with changes in client dietary restriction and vomiting; (b) therapist cognitive interventions (e.g., cognitive restructuring) would be most strongly associated with changes in client body-related beliefs and client shape and weight concerns; and (c) therapist relational and structural interventions would be most strongly associated with changes in client treatment engagement. For treatment mechanisms, we hypothesized that client change in dietary restriction and client level of engagement in treatment would be most strongly related to changes in vomiting during treatment, and that client change in body-related dysfunctional beliefs during treatment would be most strongly related to changes in shape and weight concern during treatment.
Method

Overview

The present study was part of a larger randomized controlled trial comparing the efficacy of CBT and interpersonal psychotherapy (IPT) in the treatment of BN (i.e., Agras et al., 2000). There were two treatment sites (Stanford and Columbia) and a quality-control center (Oxford). The current study was limited to data from the 56 patients receiving CBT at the Stanford site. Acute outcome findings from the larger study indicated that CBT was significantly superior to IPT at the end of treatment in the percentage of participants recovered, percentage remitted, and percentage meeting community norm criteria for eating attitudes and behaviors. Greater detail regarding treatment outcome is provided by Agras et al. (2000).³

Participants

Clients. Participants were 56 female outpatients meeting DSM-III-R criteria for BN. Participants were recruited via advertisements and referrals from clinics. Potential participants were initially screened by telephone, which was followed by a screening appointment. Diagnoses were assessed using the SCID I and II for DSM-III-R (Spitzer, Williams, & Gibbon, 1987), and the Eating Disorder Examination (EDE; Cooper & Fairburn, 1987; Fairburn & Cooper, 1993). Assessors were supervised weekly on-site. Additionally, 20% of the EDE tapes were audited at the Oxford quality-control center and feedback faxed to the assessor and the site supervisor. The principal reasons for screening out participants were that the participant did not meet the binge/purge severity criteria for the diagnosis of BN, participant was already involved in another form of treatment, or participant disinterest in the study. Participants were also excluded from the study if they had comorbid psychosis, current anorexia nervosa, or were pregnant. Additionally, participants could not be currently participating in any other type of psychotherapeutic or psychotropic treatment. The sample ranged in age from 18 to 44 (mean = 28.5, SD = 7.3). Ethnicity was 80% Caucasian, 11% Hispanic, 5% African American, 2% Asian, and 2% American Indian. Patients reported a mean duration of binge eating of 10.8 years, and a mean duration of purging of 10.1 years.

Therapists. Four doctoral-level psychologists administered CBT at the Stanford site. All therapists were experienced in the treatment of eating disordered clients and were trained by Fairburn in the use of the Fairburn CBT protocol for BN (i.e., Fairburn et al., 1993) prior to seeing study clients. Two

³Wilson et al. (2002) report some mediator data from the larger data set. The Wilson et al. (2002) study, however, differs from the current study in that the Wilson study compares CBT and IPT on time course of effect and examines the relationship between some mediators and posttreatment outcome. Additionally, the Wilson et al. study does not examine the relationship between therapist manipulations, client mechanisms, and symptom change during treatment as described herein.
levels of ongoing monitoring of the therapy were established. Therapists were supervised weekly by an on-site supervisor (WSA). Additionally, audiotapes of a random sampling of therapy sessions were rated for protocol adherence by Oxford on an ongoing basis with ratings and feedback faxed to therapists.

**Treatment**

Treatment was administered on an outpatient basis consisting of 19 individual sessions conducted over 20 weeks. Each session was 50 minutes in length and occurred twice weekly for the first 2 weeks, weekly for the next 12 weeks, and then at 2-week intervals for the last 6 weeks. The CBT treatment was manualized (Fairburn et al., 1993) and utilized the treatment phases and principles as described in the introductory section. Briefly, the protocol consisted of three overlapping phases. The main goal of Phase 1 was to reduce dietary restraint by educating clients about BN and the processes that maintain the disorder. Clients were helped to increase the regularity of their eating, and to develop alternative ways to cope with the desire to binge eat and purge. The second phase, beginning about the ninth session, focused on identifying and changing dysfunctional thoughts and beliefs about body appearance and weight. The final three therapy sessions focused on maintenance of change and relapse prevention. Detailed descriptions of the treatment protocol are provided by Fairburn et al. (1993), Spangler (1999), and Wilson et al. (1997).

**Measures**

*Eating Disorder Examination.* The Eating Disorder Examination (EDE; Cooper & Fairburn, 1987; Fairburn & Cooper, 1993) is an interviewer-based measure used to assess the severity of eating disorder symptomatology. The scale assesses the frequency of binge eating and purging, and also yields dimensional measures of degree of dietary restraint, and shape, weight, and eating concerns. The EDE has been shown to have moderate to excellent interrater reliability (Fairburn & Cooper, 1993; Wilson & Smith, 1989), high internal consistency (Cooper, Cooper, & Fairburn, 1989), and discriminant and concurrent validity (Fairburn & Cooper, 1993).

In the present study, self-report adaptations of the EDE items regarding vomiting, dietary restriction, and body shape and weight concern were administered on a biweekly basis during treatment. Vomiting, rather than binge eating, was used as a self-report measure of symptom improvement during treatment since clients can vary widely in their definitions of what constitutes a “binge.” In contrast, vomiting is discrete and less subjective in its definition (Black & Wilson, 1996; Fairburn & Beglin, 1994; Whittal et al., 1999). Moreover, comparisons of interviewer- and self-administered versions of the EDE result in little discrepancy between the two instruments in their assessment of self-induced vomiting, dietary restraint, and weight concern (Black & Wilson, 1996; Fairburn & Beglin, 1994); in contrast, significant discrepancies
between the interviewer- and self-administered versions of the EDE are found on binge-eating items.\(^4\)

**CBT Coding Scale for Bulimia Nervosa.** The CBT Coding Scale for Bulimia Nervosa (CCS-BN; Spangler, 1998) was used to assess therapist use of specific CBT treatment components (i.e., treatment manipulations) and the treatment mechanisms of client engagement and client change in body-related beliefs during treatment. The CCS-BN is an observer-coded scale consisting of distinct therapist and client sections.

The therapist section is divided into subscales that assess therapist use and quality of execution of behavioral, cognitive, structural, and relational interventions on a Likert scale ranging from 0 (not executed) to 6 (very well executed). The behavioral subscale (6 items; alpha = .80) assesses the extent to which therapists probe for problematic behaviors (e.g., binge eating and vomiting), plan and practice alternative behaviors during the session, attempt to teach behavioral skills to the client, schedule or structure activities with the client (e.g., meal planning), and help to identify cues and/or consequences for specific behaviors (e.g., purging). The cognitive subscale (15 items; alpha = .87) assesses the extent to which therapists inquire about cognitions during sessions, and the quality of their use of various techniques for restructuring cognitions such as empiricism, identifying cognitive errors, examining evidence, generation of alternative explanations, practicing rational responses, etc. The structural subscale (2 items; alpha = .99) assesses the degree to which therapists set and follow an agenda during the session. The relational interventions subscale (7 items; alpha = .97) assesses the degree to which therapists display empathy, warmth, understanding, interpersonal effectiveness, collaboration and involvement during sessions. Items from each subscale were averaged to retain a common metric across subscales. The therapist portion of the scale has been shown to be composed of separable therapist subscales or factors (as described above) using both exploratory and confirmatory factor analyses, and to possess adequate interrater reliability (Spangler, Beckstead, Hatch, Radpour-Wiley, & Agras, 2001). In the current study, interrater correlations for the therapist subscales of cognitive, behavioral, relational, and structural interventions were .69, .64, .71, and .79, respectively, \(p < .01\) for all.

The client portion of the scale assesses (among other things) the degree of client change in body-related dysfunctional beliefs during a given session (4 items; alpha = .89), and the degree of client engagement (5 items; alpha = .91) in a given session using a Likert scale ranging from none (0) to extremely (6). The client mechanism of change in body-related dysfunctional beliefs was assessed using this scale, which focuses on change during a given session in the proposed mechanism of dysfunctional beliefs about the body

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\(^4\)We also assessed for other forms of purging, namely laxative and diuretic use, but the frequency of their use was very low across the sample and thus we did not include them in the current analyses.
(such as beliefs about the perceived consequences of attaining a particular body weight or body shape) as opposed to changes in body-related symptoms such as body weight-shape concern or body criticism, which were assessed using client self-report EDE items (e.g., "How dissatisfied have you felt about your shape?"). Confirmatory and exploratory factor analyses of the client portion of the CCS-BN have documented the distinctiveness of changes in the symptoms of body shape and weight concern from changes in dysfunctional beliefs about the body during CBT sessions for BN (Spangler et al., 2001). To assess change in body-related dysfunctional beliefs, coders rated the degree of client change in the believability of a given body-related belief (e.g., the success of my relationships depends upon my physical appearance) that clients verbalized during a given session. The client engagement scale assessed the level of client motivation, willingness, and involvement in therapy activities.

Two doctoral students in clinical psychology served as raters of the audio-taped sessions included in the current analysis. Raters received 5 months of training in general and BN-specific CBT protocol and practice including reading the CBT protocol for BN and other primary CBT texts. Prior to rating study tapes, each coder independently rated 10 CBT pilot sessions, afterward discussing any discrepancies to ensure that raters interpreted items similarly. To prevent drift, raters met periodically with the trainer (DLS) to rate and discuss tapes on an ongoing basis. Ratings were based on entire sessions rather than segments thereof. In the current study, Sessions 4, 10, 14, and 16 were rated for all clients to allow for examination of the relationship between therapist (i.e., manipulation), client (i.e., mechanism), and symptom variables during treatment.

In summary, the therapist portion of the CCS-BN was used to assess therapist manipulations. The client portion of the CCS-BN was used to assess the client mechanisms of change in dysfunctional body-related beliefs and change in level of client engagement. The self-report EDE was used to measure the client mechanism of changes in dietary restraint and the symptoms of vomiting, weight concern, and shape concern.

Data Analyses

Hierarchical linear growth curve models were used to model the relationships between the hypothesized treatment manipulations, treatment mechanisms, and symptoms during treatment. "Growth" in this type of analysis refers to changes, either increases or decreases, in the level of a particular dependent variable over time. A hierarchical linear model is a multilevel technique, which combines between and within individual variability in the growth curves into a single regression equation. In the current study, level one of each model examined changes in the dependent variable within clients across sessions and how those changes are related to other time-varying covariates (e.g., treatment manipulations). In level two of the analyses, between client variables (e.g., therapist) were added to the model to determine their
association with any change in a given dependent variable across the trajectory of sessions.\(^5\)

Given the hypothesized distinct relationships between treatment manipulations, mechanisms, and symptoms, analyses were divided into two sets. The first set of analyses examined the relationships between treatment manipulations and treatment mechanisms across the trajectory of sessions. This first set of analyses consisted of three separate analyses for each hypothesized treatment mechanism (i.e., dietary restraint, body-related beliefs, and client engagement). In Model 1 of each analysis, session number was entered as the sole predictor to determine whether or not significant change in each treatment mechanism had occurred across sessions. In Model 2, the four hypothesized therapist manipulations were entered (as a block) as predictors of change in each of the three hypothesized client mechanisms.

The second set of analyses examined the relationships between treatment manipulations and symptoms, and whether or not the proposed client mechanisms mediated the relationship between therapist manipulations and symptom change. To examine the associations between the treatment manipulations and symptoms, we conducted three separate analyses for each symptom (i.e., vomiting, weight concern, and shape concern). In Model 1 of each analysis session number was entered as the sole predictor to determine whether or not significant change in each symptom had occurred across sessions. In Model 2, the four hypothesized therapist manipulations were entered (as a block) as predictors of change in each of the three symptoms. To test whether the proposed client mechanisms mediated the relationship between therapist manipulations and symptoms, we next entered all three hypothesized mechanisms as a block (Model 3). In all models, session number, manipulations, and mechanisms were treated as fixed effects, whereas the intercept was included as a random effect.

Because the vomiting variable is a rare count variable, it is distributed as a Poisson random variable (Land, 1992; Osgood & Rowe, 1994). An assumption of Poisson regression methods is that the mean and the variance of the outcome variable be equal. In the present study, however, the variance was greater than the mean, as is the case in most data sets (i.e., the distribution was overdispersed). Ignoring overdispersion results in inconsistent estimates and incorrect \(p\) values (Cameron & Trivedi, 1990). Therefore, the model was estimated using a Poisson procedure that corrects for overdispersion (Bryk, Raudenbush, & Congdon, 1996; Christiansen & Morris, 1997). The link function for a Poisson model is a log-link. Thus, the results of the vomiting growth curve analysis can be interpreted as the log-odds of either increasing or decreasing vomiting behavior.

\(^5\)We included three therapist dummy variables (therapist number four was the reference category) in each model. None of the dummy variables was statistically significant in any model. Thus, these results are not included in the growth model tables.
Results

Relationships Between Treatment Manipulations and Treatment Mechanisms

Dietary restraint. Model 1 included only session number as a predictor of change in dietary restraint. The significant coefficient in Model 1 indicated that there was a significant decrease in dietary restraint across sessions (see Table 2). Model 2 included the hypothesized treatment manipulations as predictors of the change in dietary restraint across sessions. With the inclusion of these variables, the relationship between session number and changes in dietary restraint became insignificant, indicating that the change in dietary restraint across sessions was completely accounted for by the treatment manipulations occurring within each session. The only treatment manipulation that was significant in this model was therapist relational interventions. Specifically, increases in therapist relational interventions were associated with decreases in dietary restraint.

Body-related dysfunctional beliefs. Model 1 included only session number as a predictor of change in body-related dysfunctional beliefs. The significant coefficient in Model 1 indicated that there was substantial change in body-related dysfunctional beliefs across sessions. Model 2 included the hypothesized treatment mechanisms as predictors. With the inclusion of these variables, session remained significant, although its effect was reduced by approximately 50%, suggesting that the treatment mechanisms accounted for a substantial amount of session-related change in body-related dysfunctional beliefs. Across sessions, behavioral interventions had a significant negative relationship with changes in body-related dysfunctional beliefs, whereas relational interventions had a significant positive relationship with changes in body-related beliefs. None of the other treatment mechanisms had a significant relationship with changes in body-related dysfunctional beliefs.

Client engagement. Model 1 included only the session variable as a predictor of change in client engagement. The significant coefficient in Model 1 indicated that there was a significant increase in level of client engagement across sessions. Model 2 included the hypothesized treatment manipulations as predictors. Across sessions, therapist relational interventions had a significant positive relationship with client engagement, whereas all other treatment mechanisms were not significantly related. Thus, increases in client engagement were solely associated with increases in therapist relational interventions.

Relationships Between Symptoms, Treatment Manipulations, and Treatment Mechanisms

Vomiting. The results of the vomiting analyses are presented in Table 3. Model 1 included only session number as a predictor of change in vomiting frequency. The significant coefficient in Model 1 indicated that there was a significant decrease in vomiting across sessions. Model 2 included the hypothesized treatment manipulations as predictors. None of the treatment manipulations were
TABLE 2
GROWTH CURVE OF THERAPIST MANIPULATIONS PREDICTING CLIENT MECHANISMS

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Dietary Restraint</th>
<th>Body-Related Beliefs</th>
<th>Client Engagement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 1</td>
</tr>
<tr>
<td>Fixed effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>4.65 (0.21)**</td>
<td>5.74 (0.70)**</td>
<td>0.11 (0.11)</td>
</tr>
<tr>
<td>Session</td>
<td>-0.14 (0.02)**</td>
<td>-0.06 (0.03)</td>
<td>0.15 (0.01)**</td>
</tr>
<tr>
<td>Behavioral interventions</td>
<td>0.15 (0.15)</td>
<td>-0.49 (0.08)*</td>
<td></td>
</tr>
<tr>
<td>Cognitive interventions</td>
<td>0.23 (0.20)</td>
<td>0.10 (0.11)</td>
<td></td>
</tr>
<tr>
<td>Relational interventions</td>
<td>-0.54 (0.15)**</td>
<td>0.49 (0.08)**</td>
<td></td>
</tr>
<tr>
<td>Structural interventions</td>
<td>-0.01 (0.09)</td>
<td>0.09 (0.05)</td>
<td></td>
</tr>
<tr>
<td>Random effects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>1.09**</td>
<td>0.99**</td>
<td>0.37*</td>
</tr>
<tr>
<td>Residual</td>
<td>1.37**</td>
<td>1.27**</td>
<td>0.87**</td>
</tr>
</tbody>
</table>

Note. The results represent a set of nested multilevel growth curve models. Coefficients are provided with standard errors in parentheses.

* p < .05; ** p < .01.
MECHANISMS OF ACTION IN CBT

GROWTH CURVE OF MANIPULATIONS AND MECHANISMS PREDICTING CHANGES IN THE SYMPTOM OF VOMITING

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Model 1</th>
<th>Model 2</th>
<th>Model 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed effects</td>
<td>Intercept</td>
<td>2.09 (0.42)**</td>
<td>2.13 (0.42)**</td>
</tr>
<tr>
<td></td>
<td>Session</td>
<td>-0.09 (0.01)**</td>
<td>-0.04 (0.02)*</td>
</tr>
<tr>
<td></td>
<td>Behavioral interventions</td>
<td>-0.01 (0.10)</td>
<td>0.03 (0.11)</td>
</tr>
<tr>
<td></td>
<td>Cognitive interventions</td>
<td>-0.13 (0.13)</td>
<td>-0.08 (0.12)</td>
</tr>
<tr>
<td></td>
<td>Relational interventions</td>
<td>-0.11 (0.09)</td>
<td>0.20 (0.11)</td>
</tr>
<tr>
<td></td>
<td>Structural interventions</td>
<td>0.01 (0.05)</td>
<td>0.06 (0.05)</td>
</tr>
<tr>
<td></td>
<td>Client engagement</td>
<td>-0.42 (0.12)**</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Body-related cognition</td>
<td>-0.02 (0.10)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dietary restraint</td>
<td>0.10 (0.04)*</td>
<td></td>
</tr>
<tr>
<td>Random effects</td>
<td>Intercept</td>
<td>0.80**</td>
<td>0.80**</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>1.63</td>
<td>1.55**</td>
</tr>
</tbody>
</table>

Note. The results represent a set of nested multilevel growth curve models. Coefficients are provided with standard errors in parentheses. These analyses also included therapist assignment as a predictor. In no case was therapist assignment significant; thus, the coefficients and standard errors were not included in the table.

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

significantly associated with decreases in vomiting. Model 3 introduced the treatment mechanisms as predictors. With the inclusion of the treatment mechanisms, session number was no longer significant, indicating that all of the session-related changes in vomiting were accounted for by the predictors in Model 3. Across sessions, dietary restraint and client engagement were significantly associated with vomiting frequency. Increases in dietary restraint were associated with increases in vomiting frequency, whereas increases in client engagement in treatment were associated with decreases in vomiting frequency.

Weight concern. The results of the weight concern analyses are presented in Table 4. Model 1 included only session number as a predictor of change in weight concern. The significant coefficient in Model 1 indicated that there was a significant decrease in weight concern across sessions. Model 2 included the hypothesized treatment manipulations as predictors. Across sessions, increases in behavioral interventions were associated with significant decreases in weight concern. None of the other treatment manipulations were significantly related to decreases in weight dissatisfaction. Model 3 introduced the three hypothesized mechanisms as predictors. Of the mechanisms, decreases in dietary restraint were significantly associated with decreases in weight concern, and increased change in body-related dysfunctional beliefs was significantly associated with decreases in weight concern. Client engage-
TABLE 4
GROWTH CURVE OF MANIPULATIONS AND MECHANISMS PREDICTING CHANGES IN SYMPTOMS OF WEIGHT AND SHAPE CONCERN

<table>
<thead>
<tr>
<th>Predictors</th>
<th>Weight Concern</th>
<th></th>
<th>Shape Concern</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1</td>
<td>Model 2</td>
<td>Model 3</td>
<td>Model 1</td>
</tr>
<tr>
<td>Fixed effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>4.70 (0.19)**</td>
<td>5.66 (0.66)**</td>
<td>4.31 (0.75)**</td>
<td>4.58 (0.19)**</td>
</tr>
<tr>
<td>Session</td>
<td>-0.12 (0.01)**</td>
<td>-0.12 (0.03)**</td>
<td>-0.08 (0.03)**</td>
<td>-0.10 (0.01)**</td>
</tr>
<tr>
<td>Behavioral interventions</td>
<td>-0.31 (0.14)*</td>
<td>-0.43 (0.14)**</td>
<td></td>
<td>-0.27 (0.14)</td>
</tr>
<tr>
<td>Cognitive interventions</td>
<td>0.33 (0.18)</td>
<td>0.30 (0.17)</td>
<td></td>
<td>0.29 (0.19)</td>
</tr>
<tr>
<td>Relational interventions</td>
<td>-0.01 (0.13)</td>
<td>0.27 (0.17)</td>
<td></td>
<td>0.01 (0.14)</td>
</tr>
<tr>
<td>Structural interventions</td>
<td>-0.11 (0.08)</td>
<td>-0.08 (0.07)</td>
<td></td>
<td>-0.18 (0.08)*</td>
</tr>
<tr>
<td>Client engagement</td>
<td>-0.06 (0.17)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body-related cognition</td>
<td>-0.24 (0.12)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dietary restraint</td>
<td>0.22 (0.07)**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random effects</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intercept</td>
<td>0.99**</td>
<td>0.98**</td>
<td>0.98**</td>
<td>0.98**</td>
</tr>
<tr>
<td>Residual</td>
<td>1.20**</td>
<td>1.14**</td>
<td>1.07**</td>
<td>1.24**</td>
</tr>
</tbody>
</table>

Note. The results represent a set of nested multilevel growth curve models. Coefficients are provided with standard errors in parentheses. These analyses also included therapist assignment as a predictor. In no case was therapist assignment significant; thus, the coefficients and standard errors were not included in the table.

*p < .05; **p < .01.
ment did not have a significant relationship with changes in weight concern. The general pattern of effects of the treatment manipulations were unchanged by the inclusion of the mechanisms, although the session effect was reduced by 33% from Model 1, suggesting that the included treatment manipulations and mechanisms only partially accounted for the session-related changes in weight concern.

**Shape concern.** Model 1 included only session number as a predictor of change in shape concern. The significant coefficient in Model 1 indicated that there was a significant decrease in shape concern across sessions. Model 2 included the hypothesized treatment manipulations as predictors. Across sessions, only structural interventions had a significant relationship with changes in shape concern. Specifically, increases in structural interventions were associated with decreases in shape concern. Model 3 introduced the hypothesized treatment mechanisms as predictors. None of the mechanisms had a significant relationship with changes in shape concern. However, with the inclusion of the mechanisms, the treatment manipulation of behavioral interventions became significantly associated with changes in shape concern. Specifically, increases in behavioral interventions were associated with decreases in shape concern. Session number remained significant in Model 3, although its effect was reduced by about 30% from Model 1, suggesting that the included treatment manipulations and mechanisms partially accounted for the session-related changes in shape concern.

**Discussion**

This study examined the relationships between hypothesized therapist treatment manipulations and client treatment mechanisms in CBT for BN. Additionally, relationships between treatment manipulations, treatment mechanisms, and symptom change across therapy sessions were investigated. Overall, results provided mixed support for the hypothesized mediators of change in CBT for BN.

*Relationships between Therapist Manipulations and Client Mechanisms*

With regard to the hypotheses about the relationships between treatment manipulations and treatment mechanisms, the specific hypothesized relationship between therapist relational interventions and client engagement in therapy across sessions was supported. As predicted, increases in therapist relational interventions across sessions were associated with increases in level of client engagement across sessions, and accounted for nearly all of the change in client engagement during sessions. Increases in relational interventions were also significantly associated with increases in change in body-related dysfunctional beliefs and decreases in dietary restraint. Thus, therapist relational interventions were associated with all three client mechanisms. In addition, decreases in behavioral interventions were associated with increased change in body-related dysfunctional beliefs during treatment.
These associations between therapist manipulations and client mechanisms are largely consistent with cognitive-behavioral theory. Specifically, several CBT theorists posit that particular qualities of the interpersonal relationship between therapist and client are necessary (but not sufficient) elements of change during CBT (e.g., Beck, Rush, Shaw, & Emery, 1979; Burns & Auerbach, 1996; Newman, 1998), and that relational interventions are particularly important when addressing change in areas that the client might initially resist (Burns, 1989; Leahy, 2001), such as decreasing commitment to dietary restriction in persons with eating disorders (Vitousek, Watson, & Wilson, 1998).

The finding of increased client body-related dysfunctional belief change being associated with decreased behavioral interventions across sessions could be interpreted as either consistent or inconsistent with CBT theory. This finding is consistent with CBT theory of BN treatment in that Phase 1 is largely behavioral, focusing primarily on changing eating habits, whereas Phase 2 focuses primarily on body-related cognitive change (see Fairburn et al., 1993; Spangler, 1999; Wilson et al., 1997, for descriptions of CBT for BN). Thus, given the design of the CBT protocol used in the current study, it is not surprising to find that decreases in behavioral interventions across sessions are associated with increases in cognitive changes across sessions as this finding may simply reflect a shift from Phase 1 to Phase 2 of treatment. In contrast, it could be argued that behavioral interventions are often employed for the direct purpose of changing cognition, and thus the observed negative association between client cognitive change and behavioral interventions could be considered inconsistent with CBT theory. However, the behavioral interventions used in the current study primarily targeted eating behavior whereas the cognitive interventions primarily targeted body-related (rather than food-related) cognition. Thus, the behavioral interventions employed in the current study would not be expected to directly reduce body-related dysfunctional beliefs. Clearly inconsistent with prediction, however, was the lack of significant relationships between therapist behavioral interventions and changes in client dietary restraint across sessions, and between therapist cognitive interventions and change in body-related dysfunctional beliefs.

Relationships Between Therapist Manipulations and Client Symptoms

Some of the treatment manipulations also had direct associations with changes in client symptoms that were not mediated by the included client mechanisms. Increases in behavioral interventions were significantly associated with decreases in both weight and shape concern when controlling for treatment mechanisms. Additionally, increases in structural interventions were associated with decreases in shape concern.

These associations between treatment manipulations and symptoms suggest that some therapist manipulations may be directly associated with symptom change which is not mediated by the proposed treatment mechanisms. Consistent with CBT theory, structural interventions (e.g., agenda-
setting) were associated with increased focus on and change in key symptoms. Also supportive of the CBT use of specific behavioral interventions for symptom reduction, results suggested that the behavioral interventions were the therapist interventions primarily associated with changes in client symptoms.

The specific association of behavioral (but not relational) interventions with decreased symptoms also has implications for “common factor” theorists who hypothesize that client symptom reduction during treatment is primarily or exclusively a function of factors common across various forms of treatment such as therapist empathy, attention, and support (Beitman et al., 1989; Frank, 1985; see also Grencavage & Norcross, 1990, and Wampold, 2001, for a discussion of additional, nonrelational common factors). In contrast to the “common factor” hypothesis, the current findings suggest that “common,” relational interventions versus “specific,” cognitive-behavioral interventions predict different aspects of client change. Specifically, relational interventions were associated with greater change in client mechanisms (such as client engagement and client willingness to reduce dietary restriction) whereas specific, technical interventions (i.e., behavioral interventions) were associated with greater change in client symptoms. Overall, the current pattern of findings suggests that “common,” relational interventions are more associated with client motivation for change whereas “specific,” behavioral interventions are more associated with actual symptom change. Thus, the current pattern of findings is most consistent with theorists who propose that relational interventions are necessary but not sufficient for clinically significant change and recovery (e.g., Beck et al., 1979). The current findings are also consistent with the findings of other investigators who report that both “common” relational and “specific” cognitive-behavioral interventions significantly, but independently, predict client change during CBT (Burns & Nolen-Hoeksema, 1991; Burns & Spangler, 2000), as well as with findings that specific, cognitive-behavioral interventions alone (delivered via self-help without any relational interventions) result in significant symptom reduction (e.g., Schmidt, Tiller, & Treasure, 1993; Scogin, Jamison, & Davis, 1990; Scogin, Jamison, & Gochneaut, 1989; Smith, Floyd, Jamison, & Scogin, 1997). Taken together, these studies suggest that significant portions of client change are associated with specific, cognitive-behavioral interventions that cannot be accounted for by common, relational interventions.

We defined relational interventions as particular behaviors emitted by the therapist such as empathy, involvement, and understanding, which represent only some of the factors considered to be common across various forms of treatment (see Grencavage & Norcross, 1990, and Wampold, 2001, for a full discussion of types of common factors). Thus, our findings only extend to common, relational factors as defined herein. Other CBT researchers have explored the role of another common factor, namely, therapeutic alliance, in the treatment of BN. Consistent with our results, Wilson et al. (1999) found that therapeutic alliance was not predictive of symptom change in BN clients.
As predicted, increased change in body-related dysfunctional beliefs was associated with decreases in weight concern during treatment. This supports CBT theory which posits that change in client body-related dysfunctional beliefs leads to change in BN symptoms such as level of concern about the body (cf. Spangler, 2002). Also consistent with the CBT model were the findings that increases in dietary restraint predicted increases in weight concerns and in vomiting. These findings support the hypotheses of several theorists (e.g., Fairburn, 1981, 1997; Polivy & Herman, 1985) who propose that dietary restriction leads to binge eating and purging, and with the findings of a number of studies documenting the relationship between increased dietary restriction and increased binge eating and purging (e.g., Killen et al., 1994; Leon, Fulkerson, Perry, & Early-Zald, 1995; Patton, 1988; Wardle & Beales, 1988). Client engagement was also related to vomiting frequency. As predicted, increased client engagement in treatment was associated with decreased vomiting. The overall pattern of results for client engagement supports the purported importance of client motivation, hope, and involvement in treatment for change to occur as hypothesized by both CBT (e.g., Burns, 1989; Leahy, 2001) and common factor (Grencavage & Norcross, 1990; Wampold, 2001) theorists.

Both Fairburn (1997) and Wilson (1996, 1999) have underscored the need to improve the efficacy of CBT for BN, and have highlighted treatment mechanism research as the primary avenue for enhancing the efficacy of CBT. Wilson (1999) further notes that improving CBT may be the best method for increasing success with BN clients since administering alternative therapies has not resulted in gains in those who do not initially respond to CBT. Taken together, the current findings suggest that the behavioral, cognitive, relational, and structural mechanisms of CBT for BN are all associated with therapeutic change but with different variables. The behavioral interventions and relational interventions had the most wide-ranging associations where relational interventions predicted changes in client mechanisms and behavioral interventions predicted reductions in client symptoms. Contrary to prediction, changes in cognitive mechanisms (i.e., body-related dysfunctional beliefs) and symptoms were more consistently associated with behavioral and structural interventions as opposed to cognitive interventions. This pattern of findings suggests that increased utilization of structural interventions and body-related behavioral interventions (such as body image exposure) may enhance change of body-related dysfunctional beliefs in BN clients. Structural interventions (such as agenda-setting, pacing, summarizing) have perhaps been the least expanded upon both practically and empirically and thus greater attention to structural interventions may hold particular promise for improving CBT for BN. The current findings also suggest the importance of vacillating
between relational, technical, and structural interventions during treatment (Alford & Beck, 1997; Burns, 1989; Linehan & Kehrer, 1993). It may be that therapist flexibility across these types of interventions is more associated with recovery than skill at any one alone. BN treatment protocols could thus be enhanced by incorporating information on when and how to shift between or blend the various forms of intervention during a given treatment session, such as the strategy described by Burns (1989).

Future studies could also incorporate more fine-grained methodologies. In the current study, we attempted to extend beyond past studies of CBT treatment process by incorporating a more fine-grained measure of CBT (Spangler et al., 2001), by assessing change at several points throughout treatment, and by incorporating measures of both client mechanisms and therapist manipulations. However, even greater specificity in treatment process research is likely needed to fully understand the processes of change during treatment. For example, methodologies that allow for analysis of the differential response to interventions across individual clients could be particularly fruitful. A main idea of typical “mechanisms of action” research is that therapist manipulations cause change in client mechanisms and symptoms. This rationale of assuming that therapist manipulations (stimulus) alone have the properties to cause a client response (Bergin, 1997; Stiles & Shapiro, 1989) is more consistent with behavioral theory than cognitive theory. In contrast to stimulus-response theorizing, cognitive theory suggests that the meaning that each individual client assigns to a given treatment intervention would dictate her or his response to that intervention rather than the intervention per se. Thus in CBT treatment process research, greater attention could be paid to understanding clients’ interpretation and experience of specific interventions. Although it is typical of CBT therapists and researchers to assess client change in content of proposed dysfunctional beliefs and/or automatic thoughts, far less attention has been paid to assessing client interpretation of CBT interventions themselves. Were such assessments made, individual variability in interpretation of interventions could be considered as a potential mediator of change.

Measurement of client interpretations, whether about interventions or other relevant variables, is also in need of further consideration. In the current study, we attempted to capture clients’ changes in meaning associated with their own bodies using an observer-rated coding scale. In retrospect, this was likely a poor way to measure client change in beliefs about the body, which may explain why client change in body-related dysfunctional beliefs was not associated with therapist cognitive interventions as predicted. Future studies

7 When therapist manipulations are assumed to directly produce client changes, therapy takes on machine-like qualities. As Bergin (1997) argues, the idea that therapeutic intervention produces client change assumes that “people (personalities and psychological problems) are objects (or dependent variables) to be acted upon by therapeutic interventions (independent variables) . . .” (p. 83). If therapeutic change is merely the product of different manipulations, then clients are not meaningfully or purposefully changing their behavior but are changing as a result of a cause and effect chain begun by the therapeutic intervention.
could include more client-driven measures of beliefs about the body such as self-report measures of beliefs about the body (Cooper, Cohen-Tovee, Todd, Wells, & Tovee, 1997; Spangler, 2002; Spangler & Stice, 2001), attentional measures (Morris, Goldsmith, Roll, & Smith, 2001) or qualitative methods (Lincoln & Guba, 1985; Strauss & Corbin, 1990). Likewise, client-driven (rather than observer-rated) measures of therapist relational qualities may produce a different (and potentially more useful) set of findings since client and observer ratings of therapist relational qualities have been shown to differ and only client ratings have been reliably related to treatment outcome (Burns & Auerbach, 1996; Orlinksy, Grawe & Parks, 1994).

Timing of variable measurement is also at issue in treatment mechanism research. Several studies of CBT for depression suggest that a significant amount of change in CBT happens in early sessions (Illardi & Craighead, 1994) or happens rapidly in a single session (Tang & DeRubeis, 1999). A recent study suggests a similar rapid response to CBT is evident in the treatment of BN (Wilson et al., 2002). In the current study, we measured variables at Sessions 0, 4, 10, 14, and 16 and therefore may have missed important patterns of change occurring between those session points. Future treatment mechanism studies may particularly benefit from variable measurement at (ideally) every session or from at least oversampling earlier sessions as clients receiving CBT appear to have the greatest amount of change in early sessions.

Greater specification, both theoretically and empirically, of the timing and type of relationships between treatment manipulations, treatment mechanisms, and symptoms is also needed (cf. Spangler, 2003). For example, some therapist manipulations (e.g., relational interventions) may be expected to be related to client mechanisms (e.g., client engagement) within a given session whereas other therapist manipulations (e.g., behavioral meal planning) may be expected to be related to client mechanisms (e.g., dietary restraint) across several sessions. Currently, theory regarding the timing of client change during CBT is not at such a level of temporal precision. Furthermore, the expected type of relationship between therapist manipulations, client mechanisms, and symptoms is in need of further clarification. In the current study, we assumed a linear relationship between variables; that is, that more (or a greater dose) of a given therapist intervention should result in more change in a given client mechanism or symptom. However, as others have discussed (Stiles, 1988; Stiles & Shapiro, 1989), this “dose assumption” is often problematic when applied to psychotherapy research since clients who respond quickly to a therapist intervention may need a much lower dose of the intervention than clients who take more time to respond. Thus, the dose assumption can lead to a faulty conclusion that greater therapist intervention leads to symptom increase or that therapist intervention is unrelated to symptom change.

A final limitation of the current study concerns the directionality of effects. The significant associations observed between particular therapist manipulations, client mechanisms, and symptoms could be the result of several causal structures. For example, the observed association between increased therapist
relational interventions and increased client engagement could be due to: (a) increased therapist relational interventions encouraging client engagement; (b) increased client engagement eliciting greater therapist relational involvement, (c) circular causality wherein increases in therapist relational interventions lead to increases in client engagement and vice versa, or (d) a third variable (or common cause) leading the association between therapist relational interventions and client engagement, but that neither has direct effects on the other. Thus, the direction of all of the associations noted in the current study should be interpreted with caution until tests of the directionality of effects have been conducted (e.g., Burns & Spangler, 2000, 2001).

References


