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Changes in self-schema structure in cognitive therapy for major depressive disorder: a randomized clinical trial.

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Running head: CHANGES IN SELF-SCHEMA STRUCTURE

Changes in Self-Schema Structure in Cognitive Therapy for Major Depressive Disorder

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Abstract

Negative cognitive structure (particularly for interpersonal content) has been shown in some research to persist past a current episode of depression, and to potentially be a stable marker of vulnerability for depression (Dozois, 2007; Dozois & Dobson, 2001a). Given that cognitive therapy (CT) is highly effective for treating the acute phase of a depressive episode, and that this treatment also reduces the risk of relapse and recurrence, it is possible that CT may alter these stable cognitive structures. In the current study, patients were randomly assigned to CT + pharmacotherapy (n = 21) or pharmacotherapy alone (n = 21). Both groups evidenced significant and similar reductions in level of depression (BDI-II, HRSD), as well as automatic thoughts and dysfunctional attitudes. However, group differences were found on cognitive organization in favor of individuals who received the combination of CT + pharmacotherapy. The implications of these results for understanding mechanisms of change in therapy and the prophylactic nature of CT are discussed.

Key words: cognitive therapy; depression; cognitive change; schema

Changes in Self-Schema Structure in Cognitive Therapy for Major Depressive Disorder

Beck's cognitive model (Beck, 1967; Beck, Rush, Shaw, & Emery, 1979; Clark, Beck, & Alford, 1999; Dozois & Beck, 2008) states that depressed individuals differ from nondepressed persons in the content and process of their thinking, and that these differences are implicated in the etiology and pathogenesis of this pernicious disorder. An important assumption in Beck's theory is that vulnerable individuals develop a negative or depressive self-schema that, although dormant for a certain period in life, becomes evoked by salient negative life circumstances. Once activated, these schemas influence the content of cognition that becomes available and processed (e.g., negative automatic thoughts and dysfunctional attitudes). Thus, conceptually, the schema construct is believed to be stable (although not impermeable) whereas automatic thoughts and dysfunctional attitudes are comparatively more transient and state-dependent.

Depressive schemas have been defined in a variety of ways, with some researchers focusing on the content (e.g., core beliefs) and others paying attention to both the content and organization of cognition. Ingram and his colleagues (1984, 1990; Ingram, Miranda, & Segal, 1998), for example, argued that schemas refer to the organization, representation and storage of information in memory (i.e., cognitive structure) as well as its content (cognitive propositions). Indices of negative thinking, such as automatic thoughts and dysfunctional attitudes, on the other hand, are viewed as the ensuing byproducts or cognitive products that stem from the operations of the schema (see Dozois & Beck, 2008). Thus, a distinction is made between "surface" and "deep" cognitions (see Garratt, Ingram, Rand, & Sawalani, 2007).

Research on cognitive products in depression has demonstrated that they generally dissipate in a concurrent fashion with improvement from an episode of depression (e.g., Bhar et al., 2008; DeRubeis et al., 1990; Dozois, 2007; Jarrett, Vittengl, Doyle, & Clark, 2007; Oei & Sullivan, 1999; Westra, Dozois, & Boardman, 2002) with few consistent differences between individuals

treated with cognitive therapy and those treated with pharmacotherapy (e.g., DeRubeis et al., 1990; see Garratt et al., 2007, for review). In contrast, a well-organized network of negative information may be stable despite symptom improvement. Dozois and Dobson (2001a) evaluated the structure of the self-schema using a computerized task in which participants rated self-referential adjectives on a grid, based on their self-descriptiveness and valence. On this task, the manner in which individuals organize adjective content is believed to provide information about the degree of schema consolidation or interconnectedness. A sample of depressed females was administered this task along with measures of attention to and recall of positive and negative interpersonal information. Participants were retested 6 months later when half of the sample had improved clinically and the other half continued to meet diagnostic criteria for depression. Information processing biases (attention and memory) were evident only while depression lasted, but negative cognitive organization remained stable across time in those individuals who no longer met diagnostic criteria for major depression. This finding was replicated in a subsequent study which also demonstrated that the stability of negative cognitive organization was specific to interpersonal self-referent content (Dozois, 2007).

Negative (especially interpersonal) content appears to be well-organized/ interconnected both in and out of episode, has demonstrated sensitivity and specificity to depression (Dozois & Dobson, 2001b; Dozois & Frewen, 2006) and may therefore represent a cognitive vulnerability factor for depression consistent with Beck's theories (Beck et al., 1979; Dozois & Beck, 2008). One important question is whether cognitive organization can change with cognitive therapy (CT), a treatment that is designed specifically to alter these "deep" cognitions (see Garratt et al., 2007). Three lines of evidence converge with the proposition that CT may in fact alter these cognitive structures: (1) the prophylactic effects of cognitive therapy; (2) priming studies

indicating that there are cognitive differences between patients treated with CT and those treated with pharmacotherapy (PT); and, (3) neurobiological data on the differential neural responses to CT and antidepressant medication. Each of these literatures is reviewed briefly in turn.

The Short- and Long-Term Efficacy of Cognitive Therapy

A number of randomized clinical trials have indicated that CT yields comparable results to antidepressant medication for the acute treatment of depression, with both active treatments producing superior results than placebo control conditions (see Hollon, Haman, et al., 2002; Hollon, Thase, & Markowitz, 2002). CT also appears to be as effective as antidepressant medication for the treatment of severe depression (DeRubeis et al., 2005; Hollon et al., 2005; DeRubeis, Gelfand, Tang, & Simons, 1999). In addition to the efficacy of CT for symptom reduction, are compelling data that suggest that this form of treatment also has an added prophylactic benefit relative to PT (Dobson et al., 2008; Evans et al., 1992; Hollon, DeRubeis, & Evans, 1996; Hollon, Thase, et al., 2002). In fact, numerous studies have now shown that CT yields approximately half the relapse rates that are evident with antidepressant medication. In their meta-analysis of the efficacy of CT, for example, Glogcuen, Cottraux, Cucherat, and Blackburn (1998) reported that the average risk of relapse (based on follow-up periods of 1 to 2 years) was 25% after CT, compared to 60% following PT. Some evidence even suggests that patients who receive CT alone are no more likely to relapse after treatment than are those individuals who continue to receive medication (Dobson et al., 2008; Hollon, Shelton, Salomon, & Lovett, 2001; Hollon, Thase, et al., 2002). Hollon and DeRubeis (DeRubeis et al., 2005; Hollon et al., 2005) compared CT to antidepressant treatment (SSRIs) in patients with severe depression. Both interventions resulted in equal outcomes of remission in the acute phase of treatment, but the risk of relapse at 1-year follow-up was favourable for individuals treated with

CT compared to those treated with continuance medication (Hollon et al., 2005). In addition, the durability of this prophylactic effect appears to last for several years (Paykel et al., 2005). The reasons for differential outcome between CT and PT are, however, not yet understood and need to be explored (Dobson & Ottenbriet, 2004).

Differential Responses to Priming

Aside from the fact that the relapse data indicate that CT likely produces different cognitive change than PT are other studies which have assessed this possibility more directly. Segal and Gemar (1997) investigated prime-target relatedness on a modified Stroop task following CT. Patients who had improved after treatment demonstrated less interference for negative adjectives whereas individuals who remained depressed continued to display high levels of cognitive interference for negative self-descriptive material. No relationship was found between post-treatment status and positive interference scores. While this study implies that patients successfully treated with CT may experience a shift in the organization of their negative self-structures, the interconnectedness of the self-schema was inferred from reaction times. Nonetheless, this research represents an important step toward assessing the structural properties of schemas in depression.

Segal, Gemar and Williams (1999) also addressed this idea of cognitive product change by comparing patients who had successfully completed either CT or PT. Following treatment, participants were administered the Dysfunctional Attitude Scale (DAS; Weissman & Beck, 1978), a self-report measure of negative beliefs and attitudes concerning self. They were subsequently induced into a dysphoric mood state and then administered a parallel form of the DAS. Individuals who received antidepressant medication showed more elevated DAS scores than did those who received CT, suggesting that the latter treatment modality may alter certain

crucial cognitive patterns. Segal et al. (2006) also found that these mood-linked changes in dysfunctional attitudes were predictive of relapse 18 months later. Thus, it is conceivable that CT does exert significant change on an individual's core negative structures, change that seems to lead to lasting therapeutic gains (also see Teasdale et al., 2002; Williams, Teasdale, Segal, & Soulsby, 2000).

Neuroimaging Studies

In addition to results of differential mood-reactivity are neuroimaging data on changes in CT. Goldapple et al. (2004) examined the neural responses to CT in unmedicated depressed outpatients who received CT and compared these findings with an independent sample of individuals who had been treated with paroxetine. Goldapple et al. (2004) found different pre- vs. post-treatment changes in the metabolic activity (PET scans) of individuals treated with CT compared to those treated with antidepressant medication.

Current Study

It is clear that CT has both short and long-term benefits for patients with clinical depression. Notwithstanding the efficacy of CT, the nature of cognitive changes when patients successfully complete CT is not well understood. Therefore, the primary objective of this research was to test the cognitive mechanisms underlying the efficacy of cognitive therapy. In addition to assessing change in dysfunctional beliefs and the frequency of negative automatic thoughts, we also investigated whether cognitive therapy alters cognitive organization or structure. These ideas were tested in a real-world "effectiveness" sample of patients referred to a tertiary care clinic.

We predicted that cognitive products would improve significantly following both cognitive therapy (CT) and guideline-based pharmacotherapy (PT). In contrast, we hypothesized that cognitive organization for negative content would be less interconnected in individuals who

completed CT compared to patients who were treated with antidepressant medication alone. In addition, we expected that both CT and PT would be associated with increased organization for positive content, consistent with previous research (e.g., Dozois, 2007; Dozois & Dobson, 2001a).

Methods

Participants

Participants were selected from successive referrals to the Mood Disorders Program of St. Joseph's Healthcare in Hamilton, Ontario, between November, 2004 and June, 2007. To be eligible for this study, individuals were required to be between the ages of 18 and 65 years, to have attained a minimum of 8th-grade education, and to have sufficient verbal skills (assessed via the Shipley Vocabulary test) to complete the questionnaires and cognitive tasks. Participants met diagnostic criteria for a current episode of Major Depressive Disorder (MDD), according to the Structured Clinical Interview for DSM-IV Axis I Disorders-Research Version (SCID-I, Version 2.0; First, Gibbon, Spitzer, & Williams, 1996). Individuals with comorbid conditions (e.g., anxiety disorders) were not excluded provided that depression was the main presenting problem. Exclusionary criteria consisted of a current diagnosis of Alcohol Dependence or Abuse, Substance Dependence or Abuse, prior or current diagnoses of Bipolar Disorder, Schizophrenia, Schizoaffective Disorder, or Schizophreniform Disorder, evidence of active psychosis, or cognitive impairments (e.g., Delerium, Dementia). Participants were excluded if they had electroconvulsive therapy within one year prior to study entry (see Calev et al., 1991; Rami-Gonzalez et al., 2003) or received any formal cognitive-behavioral interventions in the past. Patients who were considered refractory (i.e., they had not responded to at least 2 therapeutic

trials of antidepressants belonging to a difference class or to an empirically-supported treatment) were also excluded (Burrows, Norman, & Judd, 1994; Souery et al., 1999).

A total of 42 individuals were randomly assigned to treatment conditions and completed the study trial. The average age of these participants was 46.50 (*SD* = 10.40). The sample composition was mainly Caucasian (98%) and predominantly female (74%). Fifty-five percent of participants were married or in a common-law relationship, 24% divorced or separated, 19% single and 2% widowed. The sample was highly educated on average (M = 15.57 years; *SD* = 3.06) and most individuals (64%) were employed outside of the home (an additional 5% were retired, 2.5% were students and 2.5% were homemakers). Fourteen percent of the sample was on disability and 12% was unemployed.

Measures

Beck Depression Inventory-II (BDI-II). The BDI-II (Beck, Steer, & Brown, 1996) is the most widely used index of self-reported depressive symptomatology. The BDI-II consists of 21 items, with total scores ranging from 0-63. Numerous studies have supported the psychometric properties of the BDI-II (see Dozois & Covin, 2004; Dozois, Dobson, & Ahnberg, 1998). Internal consistency in this study was excellent ($\alpha = .89$).

Hamilton Rating Scale for Depression (HRSD). The HRSD (Hamilton, 1960, 1967) is the most frequently used clinician-rating instrument of depressive severity and is commonly used in treatment outcome research (e.g., Elkin et al., 1989). The HRSD is reliable, valid, and sensitive to treatment change (Dozois & Dobson, 2002; Nezu, Ronan, Meadows, & McClure, 2000). The internal consistency (Cronbach's alpha) in this study was .85.

Beck Anxiety Inventory (BAI). The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) is a 21-item self-report measure that assesses the severity of anxiety symptoms.

Each item is rated on a 4-point Likert-type scale (0 – 3), with total scores ranging from 0 to 63 (Beck & Steer, 1990). Numerous studies have supported the reliability and validity of this instrument (e.g., Beck et al., 1988). Coefficient alpha in this study was .93.

Automatic Thoughts Questionnaire - Negative (ATQ-N). The ATQ-N (Hollon & Kendall, 1980) is a 30-item measure that assesses the frequency of negative automatic thoughts. Each item is scored from 1 (*not at all*) to 5 (*all the time*), with higher scores indicative of more frequent negative thoughts. This instrument has excellent psychometric properties and differentiates between depressed and nondepressed groups. The internal consistency (Cronbach's alpha) in this study was .97.

Automatic Thoughts Questionnaire - Positive (ATQ-P). The ATQ-P (Ingram & Wisnicki, 1988) follows the same format as the ATQ-N, but measures the frequency of positive automatic thoughts. Previous research has supported the psychometric properties of this instrument (Burgess & Haaga, 1994; Ingram, Kendall, Siegle, & Guarino, 1995). Cronbach's alpha was .93 in this study.

Dysfunctional Attitudes Scale (DAS). The DAS (Form A; Weissman & Beck , 1978) is the most frequently cited cognitive measure related to depression, has been used extensively in the evaluation of treatment outcome (Dozois, Covin, & Brinker, 2003), and exhibits excellent psychometric characteristics (see Nezu et al., 2000). The internal consistency in this study was excellent ($\alpha = .95$).

Psychological Distance Scaling Task (PDST). The PDST (Dozois, 2002, 2007; Dozois & Dobson, 2001a, 2001b) was used to assess cognitive organization. On this task, a square grid is presented to participants on the computer monitor. In the middle of this grid is a horizontal line, anchored with the statement *not at all like me* on the left and *very much like me* on the right. A

vertical line is also shown in the middle of the grid with the anchors *very positive* at the top and *very negative* at the bottom. Adjectives are presented one at a time in the center of the grid, and respondents are instructed to move the mouse to the position on the screen that best characterizes the word's degree of self-relevance and degree of valence. There were 4 practice trials and 80 experimental trials.

The stimuli for the PDST were comprised of 80 adjectives (20 interpersonal positive, 20 interpersonal negative, 20 achievement positive, 20 achievement negative). All four word lists were statistically equivalent on the average frequency of word use in the English language, word length, emotional intensity, and imaginability (see Dozois, 2007, for additional information). The x/y coordinate point for each adjective was recorded by the computer to calculate the average interstimulus distances.[1] The manner in which individuals organize adjective content on the PDST is assumed to reflect the degree of schema consolidation or interconnectedness of self-relevant information (e.g., Bower, 1981). Greater distance among adjectives is believed to indicate less interconnectedness or consolidation of information, whereas less distance is thought to reflect greater interconnectedness or consolidation. The psychometric properties of the PDST have been supported in previous studies (Dozois, 2002, 2007; Dozois & Dobson, 2001b; Dozois & Frewen, 2006).

Procedure

After obtaining informed consent, participants were assessed using the SCID-I for the purpose of confirming eligibility. SCID-I interviews were audiotaped in order to establish interrater reliability. Interrater reliability, assessed via blind review of a random set (20%) of the audiotapes, was excellent (6 = 1.00 for MDD and 6 = .96 for other diagnoses). Eligible participants completed the symptom and cognitive questionnaires and the cognitive-

organizational tasks. Participants were then randomly assigned by a research assistant to cognitive therapy plus pharmacotherapy (CT+PT; *n* = 25) or pharmacotherapy alone (PT; *n* = 23).[2] A total of 42 individuals completed treatment (see Figure 1).[3] After treatment, participants were readministered the SCID-I, questionnaires, and cognitive task. Twenty percent of the SCID-I interviews were again examined for interrater reliability of status (remitted, still depressed) at the end of treatment. Interrater agreement for this follow-up assessment was 100%.

The trial of CT consisted of 15 individual sessions (1 hour each week), administered according to the empirically-supported protocol outlined by Beck and his colleagues (Beck et al., 1979; Beck, 1995; see Dobson, 2008). The treatment techniques used during these sessions included self-monitoring, behavioural activation, psychoeducation, and cognitive restructuring (the session-by-session protocol, based on efficacy studies, is available from the first author). The treatment was delivered by two licensed masters-level therapists (S.C. & K.M.) each with several years experience in the delivery of CT. The therapists were supervised by two Ph.D. psychologists (D.D. & P.B), both certified in CT with the Academy of Cognitive Therapy.

All patients were already receiving pharmacotherapy at the beginning of the study. Participants were treated by the psychiatrist (I.P.S.), who was blind to randomization, and received both medication and between 8 and 15 sessions of clinical management. Patient charts were reviewed weekly to ensure that a therapeutic dose was obtained before participants entered the study and to ensure maintenance. Pharmacological treatment followed the most current Canadian treatment guidelines available during the study period. Specially, medications were adjusted to adhere to the clinical guidelines for the treatment of Major Depression as outlined by Canadian Network for Mood and Anxiety Treatment (CANMAT; Kennedy et al., 2001). Subsequently medications were adjusted as clinically indicated. Eligible participants received

antidepressants such as SSRI's, SNRI's, or Tricyclics combined with various augmenting strategies whenever required (see Table 1 for a summary of medications used in the study). The specific dose of medication was monitored throughout the study using a compliance tool developed for the study. Participants in the PT group were instructed not to receive psychotherapy during the duration of the trial. At the completion of the study, participants assigned to the PT group were given the option of CT.

Results

Sample Characteristics

Initial analyses were conducted between individuals in CT+PT and PT to ensure that the groups did not differ on sociodemographic variables or patient-related variables (e.g., severity of depression, past episodes of depression). There were no significant between-group differences on age, $t(40) = -.66$, education, $t(40) = -.20$, gender, $\chi^2(1) = .12$, marital status, $\chi^2(3) = 1.54$, ethnicity, $\chi^2(1) = 1.02$, or employment status, $\chi^2(5) = 5.79$ (all *p's* > .05). Patient-related variables are presented in Table 2. Participants were statistically comparable on all variables.

Symptomatology

Table 3 presents the symptom scores for the BDI-II, HRSD and BAI at initial assessment and post-treatment. A 2 Group (CT+PT, PT) by 2 Time (pre-treatment, post-treatment) split-plot repeated measures and analysis of variance (ANOVA) with BDI-II as the dependent variable revealed a significant effect of time, $F(1,40) = 44.22$, $p < .001$ ($\eta_p^2 = .53$), but the main effect of Group, $F(1,40) = .003$, $p = .96$ ($\eta_p^2 = .00$), and the Group x Time interaction, $F(1,40) = 2.07$, $p = .16$ ($\eta_p^2 = .05$), were not significant. Similar findings were obtained on the HRSD (Group, $F[1,40] = .39$, $p = .54$, $\eta_p^2 = .01$; Time, $F[1,40] = 79.34$, $p. < .001$, $\eta_p^2 = .67$); Group x Time, $F[1,40] = 2.54$, $p = .12$, $\eta_p^2 = .06$) and the BAI (Group, $F[1,40] = .77$, $p = .39$, $\eta_p^2 = .02$; Time,

$F[1,40] = 15.50$, $p. < .001$, $\eta_p^2 = .28$; Group x Time, $F[1,40] = 1.37$, $p = .25$, $\eta_p^2 = .03$). Thus, there were no significant group differences in depressive or anxious symptomatology at pre- or post-treatment but both groups improved significantly over the course of treatment. There was also no significant between-groups difference on the number of individuals who remitted at the end of treatment as indexed by the SCID, $\chi^2(1) = 1.11$, $p = .29$.

Cognitive Products

A 2 (Group) by 2 (Time) mixed ANOVA, with ATQ-N as the dependent variable, revealed a significant effect only of time, $F(1,40) = 18.50$, $p < .001$ ($\eta_p^2 = .32$). A 2 x 2 ANOVA conducted with ATQ-P scores as the outcome variable also revealed only a significant main effect of time, $F(1,40) = 27.37$, $p < .001$ ($\eta_p^2 = .41$). As demonstrated in Figure 2, negative automatic thoughts decreased and positive automatic thoughts increased significantly over the course of treatment, with no significant differences between treatment groups.

A split-plot ANOVA was also used to examine changes in dysfunctional attitudes as a function of treatment condition. There was a significant effect of Time, $F(1,40) = 19.16$, $p < .001$ ($\eta_p^2 = .32$) but the main effect of Group and the Group x Time interaction were not significant. As shown in Figure 3, both groups evidenced a significant reduction in dysfunctional attitudes, with no statistically significant differences between groups.

Cognitive Structures

The interstimulus distances for positive and negative interpersonal and achievement content are presented in Table 4. These data were first analyzed for interpersonal and achievement content separately. A 2 Group x 2 Time x 2 Valence (positive, negative) mixed ANOVA was performed with the organization of interpersonal content as the dependent variable. The three-way interaction was marginally significant, $F(1,35) = 3.26$, $p = .08$ ($\eta_p^2 = .09$). Given a priori

hypotheses, we examined the between- and within-group contrasts. There were no significant differences between groups at pre-treatment for interpersonal positive, $t(39) = -.96$, $p = .34$, or negative, $t(39) = -.70$, $p = .49$, content. Group differences were obtained, however, post-treatment. Individuals treated with CT+PT showed significantly greater organization for positive content, $t(40) = -3.13$, $p < .01$, and less organization for negative content, $t(40) = 2.68$, $p < .01$, than those treated with PT alone. When within-subjects contrasts were inspected, individuals in the CT+PT group showed significant pre-post changes for both positive, $t(19) = 3.45$, $p < .01$, and negative, $t(17) = -4.28$, $p < .001$, content. In contrast, participants in the PT group showed a significant change only for positive content, $t(20) = 2.71$, $p < .05$ (negative, $t[40] = -1.95$, $p = .07$).

A 2x2x2 split plot ANOVA was also conducted using achievement content as the dependent variable. This analysis revealed a significant three way interaction of Group, Valence and Time, $F(1,30) = 4.67$, $p < .05$ ($\eta_p^2 = .13$). At pre-treatment, no significant between-group differences were obtained (positive: $t(37) = 1.14$, $p = .26$; negative: $t(38) = -1.46$, $p = .15$). Group differences were also not statistically significant at post-treatment for either valence (positive: $t(38) = -1.06$, $p = .30$; negative: $t(33) = 1.22$, $p = .23$). Paired t-tests, however, revealed a significant increase in the organization of positive content, $t(18) = 4.04$, $p < .001$ and a significant decrease in the organization of negative content, $t(14) = -2.61$, $p < .05$, for patients treated with CT+PT. Pre-post differences were not found for positive or negative content in the PT group ($t[19] = 1.60$, $p = .13$; $t[18] = .11$, $p = .91$, respectively).

Discussion

Cognitive therapy is a highly efficacious treatment for depression (Butler, Chapman, Forman, & Beck, 2006; Chambless & Ollendick, 2001; DeRubeis & Crits-Christoph, 1998), but

there has, and continues to be, controversy over what specific cognitive ingredients make this treatment so successful (e.g., Barber & DeRubeis, 1989; Garratt et al., 2007; Segal, 1988). This question is fundamental to both theory and therapy because the implications for the understanding of cognitive vulnerability and the delivery of treatment may vary contingent upon its answer. One possibility is that the organization or structure of the schema changes in some fundamental way (i.e., accommodation takes place; see Hollon, Evans, & DeRubeis, 1990). Another possibility is that negative thinking becomes deactivated with treatment. A third possibility is that individuals with depression eventually learn to develop healthier compensatory schemas (Garratt et al., 2007; Segal, 1988). In their review on mechanisms of change in cognitive therapy, Garratt et al. (2007) lamented that, although 14 years have passed since the last major review in this area (i.e., Whisman, 1993), not much has changed in our understanding of cognitive change in depression. In many ways, the current study provides at least an initial response to this call for research and for research that assesses deeper more structural properties of cognitive change (cf. Haubert & Dobson, 2007).

Consistent with the hypothesis that negative thinking (i.e., cognitive products) would dissipate as depression improved, for both CT+PT and PT groups, negative automatic thoughts (ATQ-N) and dysfunctional attitudes (DAS) declined and positive automatic thoughts increased (ATQ-P), with no differences between them. These findings are quite consistent with the extant literature which has compared self-reported cognition in patients treated with CT and PT (e.g., DeRubeis et al., 1990) and imply that both interventions are capable of deactivating negative thinking in depression.

In addition to altering cognitive products in depression, a particularly interesting finding was that the CT+PT intervention also seemed to alter conceptually deeper cognitive structures, an

effect that was, for the most part, unique to this treatment. This effect occurred despite the fact that these treatments showed equivalent changes in depressive symptomatology and cognitive products. Moreover, these results were obtained even though the patients treated in this study were complex cases. The individuals treated in this study had a long history of MDD and were referred to a tertiary care clinic because they had not responded to first line treatments. In addition, comorbidity was not an exclusionary criterion as is often the case in randomized clinical trials. As such, the findings from this study likely generalize to the realities of clinical practice.

The treatment groups did not differ at initial assessment. Individuals treated with CT+PT, however, evidenced significantly greater cognitive organization of positive interpersonal content and less well-connected negative interpersonal content than did individuals treated with PT alone. Moreover, individuals in the CT+PT group showed significant pre-post differences on positive and negative cognitive organization, whereas a shift in cognitive structure was not evident in the PT group. These results are intriguing in light of previous research which has shown that the organization of interpersonal negative content is stable despite the remission of depressive symptoms (Dozois, 2007; Dozois & Dobson, 2001a). It appears that cognitive therapy is able to modify these stable cognitive structures. Although between-group differences were not found for achievement content, individuals in CT+PT showed an increase in the organization of positive content and a significant decrease in the organization of negative content. Pre-post differences were not found for individuals treated with PT alone.

These findings have important implications for cognitive theory and therapy. First, together with previous research that supports the sensitivity and specificity of cognitive organization (e.g., Dozois, 2007; Dozois & Dobson, 2001a,b; Dozois & Frewen, 2006), these results suggest that

depressive schemas can be altered with cognitive therapy. Second, this study helps to elucidate possible mechanisms associated with the reduced risk of depressive relapse in cognitive therapy. Segal et al. (1999) found compelling evidence for differential mood-reactivity in patients treated with CT and PT. The present study contributes to this line of research by suggesting that cognitive therapy not only buffers the cognitive effects of an experimental mood induction (and likely naturalistic stress) but may also alter some of the deeper underlying cognitive structures that give rise to such mood-congruent activation. Major depressive disorder is not only debilitating in terms of the emotional, cognitive, behavioral, physiological, and interpersonal impairment (American Psychiatric Association, 2000) but is also highly recurrent (Coyne, Pepper & Flynn, 1999; Kessing, 1998; Solomon et al., 2000). Cognitive therapy is one of the most effective psychological treatments for depression, and yields lower relapse rates than antidepressant medication. However, the nature of the cognitive changes produced by this treatment is not understood sufficiently. The findings from this study help to provide some initial data regarding the changes in cognitive structure that take place in cognitive therapy.

There are limitations to the conclusions that may be drawn from the present study. This study did not pit CT alone against pharmacotherapy its combination. Instead, all patients were treated with evidence-based medication guidelines and only half of the sample also received 15 sessions of cognitive therapy. Utilizing depressed persons who were on a variety of antidepressant medications was an important first step in assessing cognitive change in CT and PT, more consistent with recent moves to “effectiveness” trials. Given that most patients referred for cognitive therapy are already on antidepressant medication, this study also increases the generalizability of these findings to everyday practice. This approach also allowed us to test these ideas in patients with a significant history of depressive illness that was often complex and

chronic. Arguably, it is most important to understand the effective ingredients of treatments in these kinds of "real world" samples. Notwithstanding its benefits, the design of this study does not allow us to conclude that the shift in cognitive structure was a result of cognitive therapy per se as the changes that took place may have been influenced by the synergy between cognitive therapy and pharmacotherapy. Thus, although promising, the results of this study are in need of replication.

A number of future research directions stem from this research. For example, an important issue is to ascertain what therapeutic dose is necessary for producing structural change in CT. Fava and his colleagues (Fava, Grandi, Zielezny, Canestrari, & Morphy, 1994; Fava, Grandi, Zielezny, Rafanelli, & Canestrari, 1996; Fava, Rafanelli, Grandi, Canestrari, & Morphy, 1998) have demonstrated that a few sessions of CT provided to individuals who were treated to remission using antidepressants, but who continued to show residual symptomatology, was effective in reducing the risk of relapse. The examination of CT following PT could also be investigated to determine whether structural change is obtained as efficaciously or effectively. It would also be important to ascertain whether structural change is possible in individuals who have chronic MDD. The implications for preventative interventions are also evident (e.g., does early intervention vis-à-vis selected or indicated prevention reduce the likelihood that individuals will develop a well-interconnected negative self-structure in the first place?). Determining whether changes in negative cognitive structures are predictive of future relapse prevention (e.g., Segal et al., 2006) is another important avenue of future research.

Notwithstanding its limitations, the results of this study are important conceptually and clinically as they speak directly to the axioms of the cognitive model of depression and may

facilitate our understanding regarding *why* cognitive therapy represents a prophylaxis against relapse and recurrence.

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Footnotes

[1]The formula for this index is:

$$\frac{\sqrt{\sum (X_1 - X_2)^2 + (X_1 - X_3)^2 + \ldots + (X_{19} - X_{20})^2 + (Y_1 - Y_2)^2 + (Y_1 - Y_3)^2 + \ldots + (Y_{19} - Y_{20})^2}}{n(n-1)/2}$$

where X is the adjective placement on the self-descriptiveness axis, Y is the adjective placement on the valence axis, and n is the total number of self-descriptive adjectives. These calculations involve taking the mean squared distances of every adjective-adjective combination within a particular adjective list (e.g., interpersonal positive), dividing by the total number of possible distances, and obtaining the square root of this number. As in previous research using this measure, interstimulus distances were assessed idiographically to examine the organization of the self-schematic content.

[2]Given the clinical reality that the vast majority of individuals with MDD are already on antidepressant medication by the time they reach an outpatient mental health clinic (Sirey et al., 1999), coupled with the fact that CT is most frequently used adjunctively, all participants in this study were treated to therapeutic dose with an SSRI an SNRI (e.g., Effexor), or a Tricyclic antidepressant. There are at least two advantages to such a design: (1) the service demands and ethical considerations associated with other control conditions are met and (2) every individual receives a veridical treatment, thereby reducing the risk of sample attrition (see Kazdin, 1998).

[3]Analyses were conducted to test whether there were any systematic differences between completers and participants who were excluded before randomization and between completers and individuals who dropped out of therapy. Participants were compared on sociodemographic variables, patient-related variables and self-report questionnaires. The only significant difference

obtained was for age, with completers (M = 46.25, SD = 10.60) significantly older than drop-outs (M = 36.83, SD = 9.52), $t(44) = 2.05$, $p < .05$.

Table 1

Medications provided by study condition

	CT+PT	PT
SSRI	10	9
Sertraline	2	3
Paroxetine	0	4
Citalopram	7	2
Escitalopram	1	0
SNRI (Effexor)	7	9
NDRI (Bupropion)	5	2
Tetracyclic (Mirtazapine)	1	0
Trazodone	3	0
Tricyclic (Amitriptyline)	1	0
MAOI (Moclobemide)	0	1
Mood Stabilizers	0	2
Lithium	0	1
Topiramate	0	1
Benzodiazepines	10	7
Clonazepam	7	1
Lorazepam	3	5
Zopiclone	0	1
Atypical Antipsychotics	3	1
Risperidone	1	1
Quetiapine	1	0
Olanzapine	1	0

Table 2

Patient-Related Variables

	CT+PT	PT	Statistic
	M (*SD*)/*n (%)*	*M* (*SD*) /*n (%)*	
Age of First Episode	27.81 (13.84)	24.67 (15.56)	*t* (40) = .69
Previous Episodes	6.00 (3.89)	5.83 (3.53)	*t* (45) = .87
Comorbid Axis I			
Dysthymia	0 (0%)	0 (0%)	---
Panic Disorder	2 (10%)	0 (0%)	Π^2 (1) = 2.10
Social Phobia	3 (14%)	0 (0%)	Π^2 (1) = 3.23
GAD	0 (0%)	2 (10%)	Π^2 (1) = 2.10
Specific Phobia	0 (0%)	1 (5%)	Π^2 (1) = 1.02
OCD	1 (5%)	0 (0%)	Π^2 (1) = 1.02
PTSD	1 (5%)	2 (10%)	Π^2 (1) = .36
Eating Disorders	0 (0%)	2 (10%)	Π^2 (1) = 2.10
Somatoform Disorders	0 (0%)	0 (0%)	---
Past Psychotherapy	7 (33%)	6 (29%)	Π^2 (1) = .11
Suicide Attempts	.33 (1.11)	.24 (0.52)	*t* (40) = .35
History of ECT	0 (0%)	0 (0%)	---

Note. GAD = Generalized Anxiety Disorder; OCD = Obsessive Compulsive Disorder; PTSD = Posttraumatic Stress Disorder; ECT = electroconvulsive therapy.

Table 3

Symptom Severity Scores Pre- and Post-Treatment

	CT+PT		PT	
	Time 1	Time 2	Time 1	Time 2
Variable	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
BDI-II	30.57 (9.75)	10.90 (12.29)	26.95 (10.52)	14.29 (10.34)
HRSD	20.38 (4.63)	6.43 (6.95)	19.05 (4.31)	9.33 (7.21)
BAI	19.14 (11.77)	10.09 (8.55)	14.71 (11.04)	9.81 (10.13)

Note. BDI-II = Beck Depression Inventory-II; HRSD = Hamilton Rating Scale for Depression; BAI = Beck Anxiety Inventory.

Table 4

Cognitive Organization Scores Across Time.

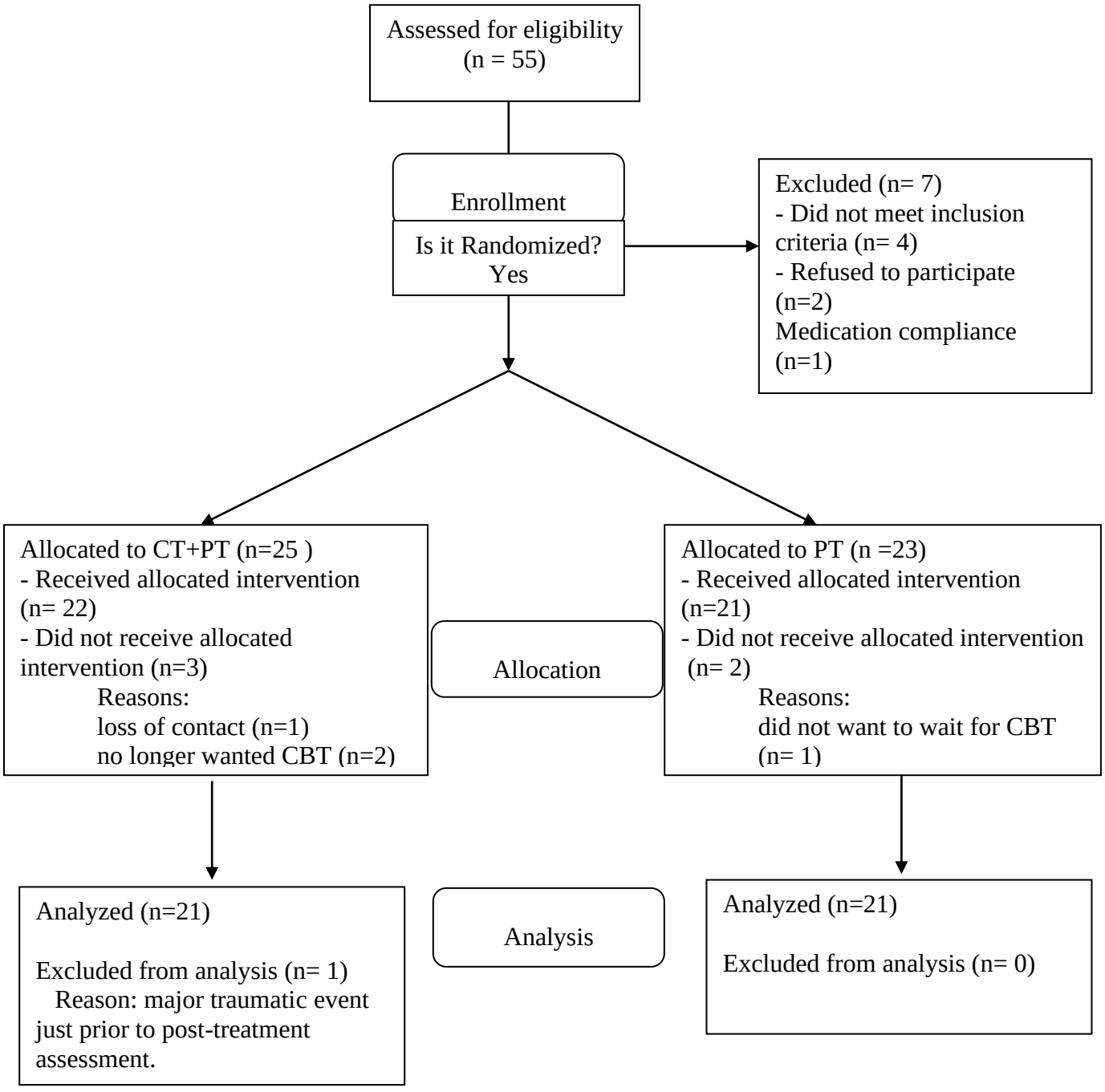
	CT+PT		PT	
	Time 1	Time 2	Time 1	Time 2
Variable	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
Interpersonal positive	.52 (.28)	.28 (.24)	.59 (.20)	.48 (.17)
Interpersonal negative	1.05 (.47)	1.65 (.56)	.96 (.36)	1.15 (.59)
Achievement positive	.95 (.46)	.53 (.36)	.80 (.35)	.66 (.36)
Achievement negative	.74 (.39)	1.25 (.61)	.99 (.65)	1.02 (.47)

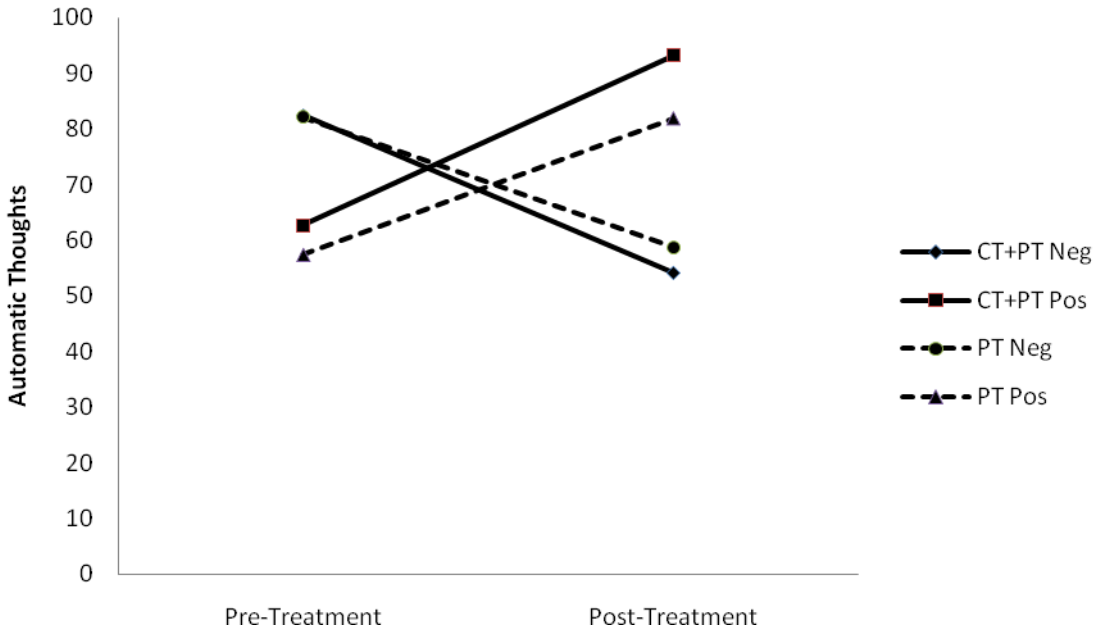
Figure Caption

Figure 1. CONSORT flow chart; CT+PT = Cognitive Therapy plus Pharmacotherapy; PT = Pharmacotherapy.

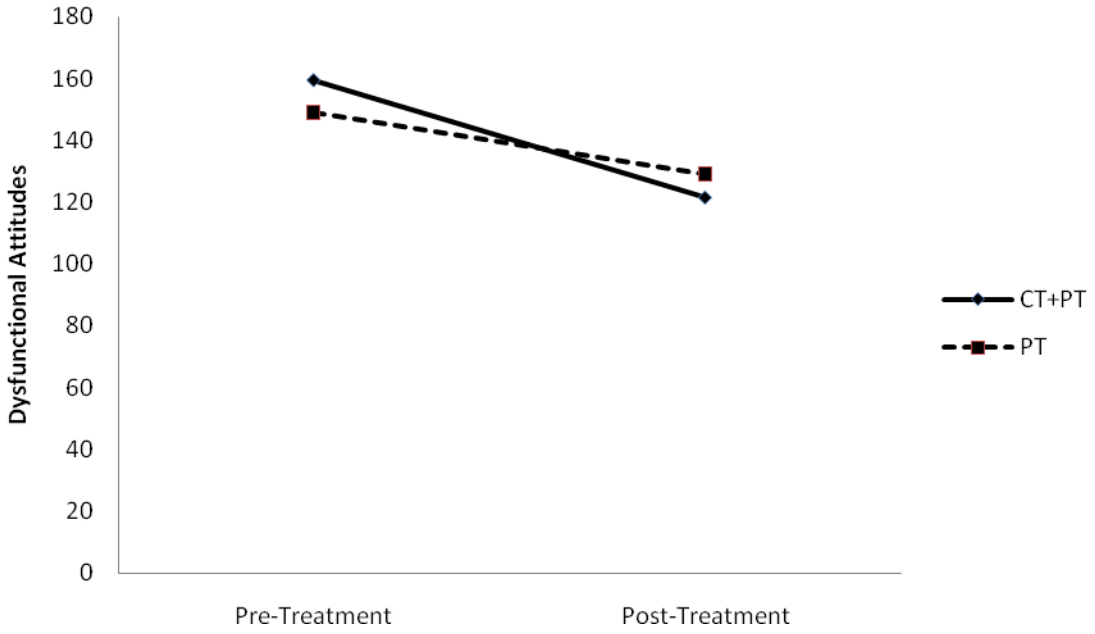
Figure 2. Changes in Positive and Negative Automatic Thoughts Before and After Treatment

Figure 3. Changes in Dysfunctional Attitudes Before and After Treatment.


Assessed for eligibility
(n = 55)
Enrollment
Is it Randomized?
Yes
Excluded (n= 7)
- Did not meet inclusion criteria (n= 4)
- Refused to participate (n=2)
Medication compliance (n=1)
Allocated to CT+PT (n=25)
- Received allocated intervention (n= 22)
- Did not receive allocated intervention (n=3)
Reasons:
loss of contact (n=1)
no longer wanted CBT (n=2)
Allocation
Allocated to PT (n =23)
- Received allocated intervention (n=21)
- Did not receive allocated intervention (n= 2)
Reasons:
did not want to wait for CBT (n= 1)
Analyzed (n=21)
Excluded from analysis (n= 1)
Reason: major traumatic event just prior to post-treatment assessment.
Analysis
Analyzed (n=21)
Excluded from analysis (n= 0)



Note. CT+PT= Cognitive Therapy + Pharmacotherapy; PT = Pharmacotherapy; Neg = Negative Automatic Thoughts; Pos = Positive Automatic Thoughts


180
160
140
120
100
80
60
40
20
0
Dysfunctional Attitudes
Pre-Treatment
Post-Treatment
CT+PT
PT

Appendix A

CONSORT Statement 2001 - Checklist

Items to include when reporting a randomized trial

PAPER SECTION **And topic**	Item	**Descriptor**	**Reported on Page #**
TITLE & ABSTRACT	1	How participants were allocated to interventions (*e.g.*, "random allocation", "randomized", or "randomly assigned").	2
INTRODUCTION Background	2	Scientific background and explanation of rationale.	3-8
METHODS Participants	3	Eligibility criteria for participants and the settings and locations where the data were collected.	8
Interventions	4	Precise details of the interventions intended for each group and how and when they were actually administered.	11-13
Objectives	5	Specific objectives and hypotheses.	7-8
Outcomes	6	Clearly defined primary and secondary outcome measures and, when applicable, any methods used to enhance the quality of measurements (*e.g.*, multiple observations, training of assessors).	9-11
Sample size	7	How sample size was determined and, when applicable, explanation of any interim analyses and stopping rules.	9, 35
Randomization -- Sequence generation	8	Method used to generate the random allocation sequence, including details of any restrictions (*e.g.*, blocking, stratification)	9, 11, 12
Randomization -- Allocation concealment	9	Method used to implement the random allocation sequence (*e.g.*, numbered containers or central telephone), clarifying whether the sequence was concealed until interventions were assigned.	11
Randomization -- Implementation	10	Who generated the allocation sequence, who enrolled participants, and who assigned participants to their groups.	12
Blinding (masking)	11	Whether or not participants, those administering the interventions, and those assessing the outcomes were blinded to group assignment. If done, how the success of blinding was evaluated.	13-15
Statistical methods	12	Statistical methods used to compare groups for primary outcome(s); Methods for additional analyses, such as subgroup analyses and adjusted analyses.	
RESULTS Participant flow	13	Flow of participants through each stage (a diagram is strongly recommended). Specifically, for each group report the numbers of participants randomly assigned, receiving intended treatment, completing the study protocol, and analyzed for the primary outcome. Describe protocol deviations from study as planned, together with reasons.	35
Recruitment	14	Dates defining the periods of recruitment and follow-up.	
Baseline data	15	Baseline demographic and clinical characteristics of each group.	9, 32
Numbers analyzed	16	Number of participants (denominator) in each group included in each analysis and whether the analysis was by "intention-to-treat". State the results in absolute numbers when feasible (*e.g.*, 10/20, not 50%).	35
Outcomes and estimation	17	For each primary and secondary outcome, a summary of results for each group, and the estimated effect size and its precision (*e.g.*, 95% confidence interval).	13-15
Ancillary analyses	18	Address multiplicity by reporting any other analyses performed, including subgroup analyses and adjusted analyses, indicating	13-15

		those pre-specified and those exploratory.	
Adverse events	19	All important adverse events or side effects in each intervention group.	35
DISCUSSION Interpretation	20	Interpretation of the results, taking into account study hypotheses, sources of potential bias or imprecision and the dangers associated with multiplicity of analyses and outcomes.	15-19
Generalizability	21	Generalizability (external validity) of the trial findings.	18
Overall evidence	22	General interpretation of the results in the context of current evidence.	15-19

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