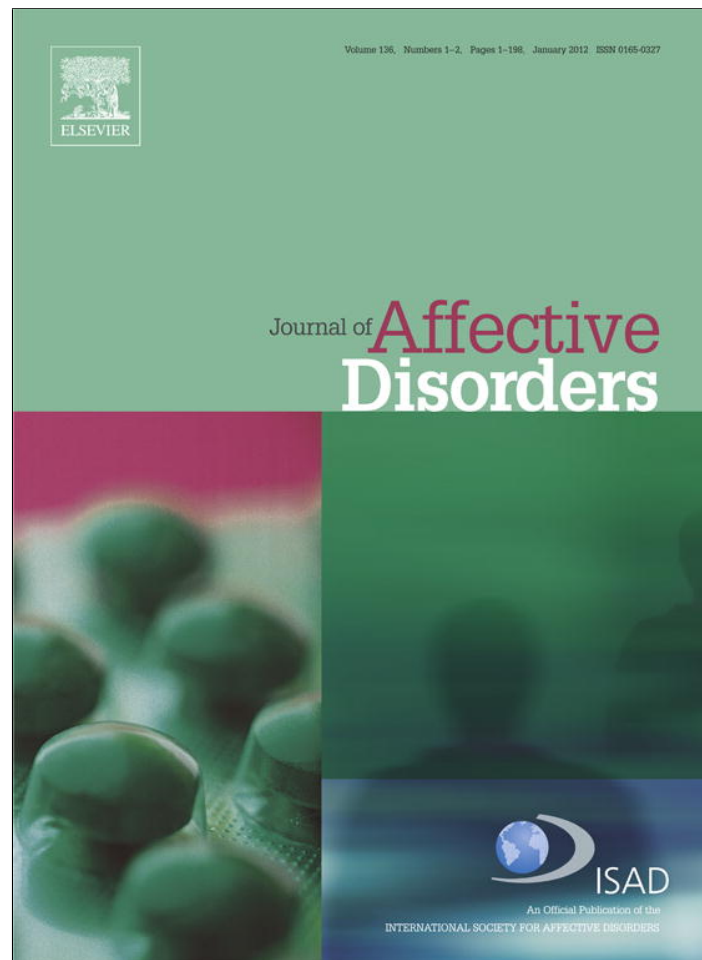


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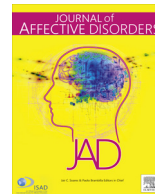
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Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research paper

Changes in causal attributions and relationship representations: Are they specific or common mechanisms in the treatment of depression?



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ARTICLE INFO

Article history:

Received 3 August 2015

Received in revised form

29 December 2015

Accepted 30 December 2015

Available online 31 December 2015

Keywords:

Psychodynamic therapy

Specific mechanisms

Common mechanisms

Relational representations

Attributional style

Depression

ABSTRACT

Background: The goal of the study was to examine two central theory-driven mechanisms of change, causal attributions and relational representations, to account for symptomatic improvement in psychodynamic treatment and supportive clinical management, combined with either pharmacotherapy or placebo, in a randomized control trial (RCT) for depression.

Method: We used data from an RCT for depression, which reported non-significant differences in outcome among patients ($N=149$) who received supportive-expressive psychotherapy (SET), clinical management combined with pharmacotherapy (CM+MED), or clinical management with placebo pill (CM+PBO) (Barber et al., 2012). Mechanism and outcome measures were administered at intake, mid-treatment, end of treatment, and at a 4-month follow-up.

Results: Improvements in causal attributions and in relational representations were found across treatments. Changes in causal attributions did not predict subsequent symptomatic level when controlling for prior symptomatic level. In contrast, decrease in negative relational representations predicted subsequent symptom reduction across all treatments, and increase in positive relational representations predicted subsequent symptom reduction only in SET.

Limitations: The study is limited by its moderate sample size. Additional studies are needed to examine the same questions using additional treatment orientations, such as cognitive treatments.

Conclusions: Findings demonstrate that changes in negative relational representations may act as a common mechanism of change and precede symptom reduction across psychodynamic therapy and supportive case management combined with either pharmacotherapy or placebo, whereas an increase in positive relational representation may be a mechanism of change specific to psychodynamic therapy.

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1. Introduction

In the last decades, empirical findings have consistently shown that different therapies often yield similar treatment outcomes, particularly in the treatment of depression. Repeated meta-analyses have shown that treatments of depression based on different theoretical principles are often found equally effective (Barber et al., 2013; Barth et al., 2013; Cuijpers et al., 2008; Leichsenring, 2001). Such similarities raise the question of whether mechanisms common or distinct to different treatments account for patient improvement (Gelfand and DeRubeis, 2014). If common

mechanisms exist, researchers should identify these to optimize treatments so that they include more strategies that trigger critical change processes (Coleman et al., 2010; Kazdin, 2007; Laska et al., 2014). If, however, distinct mechanisms account for patient improvement in different treatments, researchers must clarify these to further our understanding of the various causes of psychopathology and help us choose the most suitable treatment for each patient presentation (Barber and Muenz, 1996; DeRubeis et al., 2014).

The literature on common mechanisms focuses predominantly on therapeutic alliance (Castonguay et al., 2006). The scope of common mechanisms, however, has broadened in recent years to include theory-specific mechanisms that may change similarly across different treatment approaches (Crits-Christoph et al., 2013). The expansion of scope may be partially due to empirical evidence showing that many therapists are rather eclectic in their

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practice (Cook et al., 2010), even those in randomized controlled trials (RCTs) who are expected to adhere to a rather “pure/prototype” approach (Ablon and Marci, 2004; Barber et al., 2004, 2008). The expansion in scope may also be partially due to the possibility that techniques from distinct treatment orientations (e.g., identifying underlying dysfunctional interpersonal schemas in cognitive therapy vs. identifying core conflictual relationship themes in psychodynamic therapy) may work through a similar mechanism (DeRubeis et al., 2005). Despite the growing interest in common and specific mechanisms of change in different treatments for depression, few empirical studies demonstrate clearly which mechanisms may account for similar results across different treatments.

The current study aims to fill in some of the gaps by examining two theory-driven mechanisms of change in the treatment of depression. Both are considered to be key general psychological constructs for understanding the psychopathology and treatment of depression. The first mechanism focuses on changing dysfunctional causal attributions; the second on changing maladaptive relational representations. Both are described as important factors in the origin and persistence of depression (Lorenzo-Luaces et al., 2014; Luborsky and Mark, 1991), and are targeted in the practice of psychotherapy with the aim of assisting patients develop more adaptive perceptions, either of the world in general or of their interpersonal relationships. It is unclear, however, how common these factors are across various treatments, and especially whether they also apply to supportive clinical management (in which supportive techniques are allowed but techniques specific to a psychotherapeutic orientation are prohibited) and to supportive-expressive treatment (SET) of depression (Luborsky, 1984; Leichenring and Leibling, 2007). Changes in maladaptive relational representations, which are at the heart of SET, are expected to show some specificity to this treatment, whereas changes in dysfunctional attributions, which are not the focus of either supportive clinical management or SET (but rather of cognitive treatments), are expected to demonstrate no specificity to either treatment.

Depressive attributional or explanatory style is a form of inaccurate and maladaptive information processing (Abramson et al., 1978), considered to play a causal role in vulnerability to depression. It reflects a tendency to attribute bad events to internal, stable, and global causes, and good events to external, unstable, and specific causes (Peterson et al., 1982). According to the cognitive model, when maladaptive thinking improves, depressive symptoms are reduced (Beck et al., 1979). Consistent with this theoretical assumption, studies suggest that negative thinking can prospectively predict the onset, relapse, and recurrence of symptoms of depression (Mathews and MacLeod, 2005; Scher et al., 2005; Wenze et al., 2010). Empirically, some evidence supports the claim that causal attributions may change through treatment (DeRubeis et al., 1990; Lorenzo-Luaces et al., 2014; Shirk et al., 2013; Vittengl et al., 2015). However, the few studies that have examined the temporal relationship between changes in cognition and outcome have produced mixed findings (Lorenzo-Luaces et al., 2014), and it is still an open question whether changes in causal attribution precede those in depressive symptoms (Crits-Christoph et al., 2013). Even less is known about changes in negative cognitions and their relation to outcomes *outside* of cognitive treatment (Barber et al., 2005; Oei and Free, 1995; Quilty et al., 2008), although it has been argued repeatedly that effective treatment for depression should include changes in causal attribution owing perhaps to the nearly-universal depressive cognition in patients suffering from depression (Coleman et al., 2010; Garratt et al., 2007; Quilty et al., 2008). It is therefore an open question whether modification of maladaptive cognition is a necessary requirement for any successful treatment of depression (Dimidjian et al., 2006).

The second theory-driven mechanism of change on which we

focus concerns relational representations. Conceptualized in the context of the relational/interpersonal perspectives on depression, they explore patients' internalized representations of their relationships with significant others as a vehicle of therapeutic change (Bowlby, 2005; Freud, 1958; Luborsky, 1984; Luyten and Blatt, 2013; Mikulincer et al., 2013). According to these perspectives, relational themes are carried over from a patient's interpersonal experiences in childhood, and tend to be applied repeatedly later in life in different relationships, becoming rigid representations of others. Rigid, malevolent representations are considered to play a causal role in the origin and maintenance of depression. Based on this perspective, one of the main goals in the treatment of depression is to explore and rework these representations to develop more adaptive ways of perceiving and experiencing interpersonal relationships. Changes in interpersonal internal representations are expected to apply to real life interactions with others, and ultimately lead to symptom reduction (Book, 1998; Shedler, 2010). Most studies conducted so far examined change in relational representations in long-term dynamic treatments and produced mixed results (Blatt et al., 1996; Grenyer and Luborsky, 1996; Luborsky and Crits-Christoph, 1998; Wilczek et al., 2004). Much less is known about the change in relational representations and their associations with symptomatic change in short-term dynamic treatment.

Although both general causal attributions and relational representations form a central part of most theories on the causes of depression and on the mechanisms of change underlying symptom reduction, few studies have addressed the question whether changes in these mechanisms are treatment-specific or common across treatments, and whether improvement in these theory-driven mechanisms is associated with greater benefits in various treatments for depression. To address this issue, in the current study we first examined whether the two theory-driven mechanisms changed significantly over the course of treatment in different treatment conditions. We used data from an RCT for depression (Barber et al., 2012), comparing dynamic supportive-expressive therapy (SET) and supportive clinical management combined with pharmacotherapy (CM+MED) or with placebo (CM+PBO). In previous analyses on these data, no significant differences were found between the three treatment conditions in their efficacy, and patients in all treatment conditions experienced a significant reduction in depressive symptoms (Barber et al., 2012) and significant increases in quality of life and life-satisfaction (Zilcha-Mano et al., 2014b). Data from this RCT enable examining whether specific or common mechanisms are underlying similar outcomes in the treatment of depression. The most ideal design also includes a treatment condition aimed at working on attributional style, like cognitive-behavioral therapy. These data were not available in the current study.

We hypothesized that the two potential mechanisms of change, attributional style and relationship representations, show significant change in all three treatments because both are assumed to be central constructs in the psychopathology of depression. Although other specific and common mechanisms for placebo response (e.g., classical conditioning, in which individuals associate improvement in symptoms with taking a pill, or expectancy in which placebo instills a positive expectation of improvement), pharmacotherapy (a physiologic effect of the medication being studied on the target disorder, e.g., the effect of serotonin reuptake inhibition), and case-management can be proposed (Constantino et al., 2011; Imber et al., 1990; Rutherford and Roose, 2013; Stahl, 1998; Stewart-Williams and Podd, 2004; Vaswani et al., 2003; Zilcha-Mano et al., 2015), these were not measured in the original RCT.

The prediction that both general causal attributions and relational representations will change over treatment does not

necessarily mean that both are active mechanisms in bringing about symptomatic change in given treatments. Therefore, in the second stage, we examined whether change in each of the two mechanisms predicted subsequent change in symptoms across treatments. Given the emphasis of examining relationship patterns in SET but not in CM+PBO/MED, we hypothesized that changes in relational representations serve as a treatment-specific mechanism and predict subsequent symptomatic change in SET only. We also hypothesized that changes in general causal attribution do not predict subsequent symptomatic change in all treatment conditions given that none of these treatments specifically target causal attributions.

Finally, to unravel the temporal course of change in mechanism and outcome variables (Barber, 2009; Barber et al., 2013; DeRubeis et al., 2005), we used a statistical model that allowed testing whether changes in causal attribution or relational representations precede symptomatic change (see further elaboration in the Data Analytic Plan section below). In this way, we circumvented a limitation of many previous studies that did not adequately address the timeline challenge between mechanism and outcome (Zilcha-Mano et al., 2014a, 2014b).

In sum, we hypothesized that (a) changes in maladaptive relational representations which are at the heart of SET, show specificity to this treatment and therefore change across all treatments, but predict subsequent symptomatic levels only in SET, and (b) changes in dysfunctional attributions, which are not the target of either supportive clinical management or SET (but rather of cognitive treatments), demonstrate no specificity to either of these treatments and change across all treatments, but do not predict subsequent symptomatic levels in any of the three treatments.

2. Method

2.1. Participants

One hundred fifty-six depressive patients were randomly assigned to one of three treatments groups: SET, CM+MED, or CM+PBO. Only those who completed at least one mechanism and outcome questionnaire were included in the study, resulting in 149 participants. Patient characteristics (Table 1) show that differences between the treatments failed to emerge for any baseline demographic or clinical characteristic.

Table 1
Patients' demographic and clinical characteristics as a function of treatment condition.

Variable	MED (n=50)	SET (n=49)	PBO (n=50)	Total (n=149)	Statistical test	P Value
Demographics						
Age, y, mean (SD)	38.4 (12.7)	36 (12.1)	38.3 (12.0)	37.5 (12.2)	$F_{(2,146)}=0.60$.54
Education, y, mean (SD)	13.8 (2.9)	13.7 (2.1)	14.4 (2.8)	13.9 (2.6)	$F_{(2,143)}=0.79$.45
Female	56 (28)	63.3 (31)	62 (31)	60.4 (90)	$\chi^2=0.62$.73
Income > \$30,000	76 (38)	75.5 (37)	74 (37)	75.2 (112)	$\chi^2=0.05$.97
Single	48 (24)	52.1 (25)	48 (24)	49.3 (73)	$\chi^2=6.33$.78
Employed	40 (20)	36.7 (18)	44 (22)	58.4 (87)	$\chi^2=0.87$.64
Minority	40 (20)	59.2 (29)	50 (25)	49.7 (74)	$\chi^2=4.46$.10
Clinical features						
Chronic MDD	39.5 (15)	41.5 (17)	36.4 (12)	39.3 (44)	$\chi^2=2.03$.73
Age of onset, y, mean (SD)	22.4 (14.5)	24.1 (14.2)	21.1 (16.2)	22.6 (14.9)	$F_{(2,129)}=0.44$.63
Length of current MDD episode, mo, mean (SD)	53.0 (110)	26.7 (36.7)	34.6 (80.1)	37.6 (80.4)	$F_{(2,128)}=1.24$.29
Comorbidities						
Any Axis I disorder	56 (28)	63.3 (31)	72 (36)	63.8 (95)	$\chi^2=2.77$.24
Dystamia	18 (9)	8.2 (4)	12 (6)	12.8 (19)	$\chi^2=2.19$.33
Any Axis II disorder	44.9 (22)	51.1 (24)	42 (21)	45.9 (67)	$\chi^2=0.83$.66
Intake HRSD score, mean (SD)	19.5 (3.7)	21.1 (4.0)	20.3 (4.3)	20.3 (4.0)	$F_{(2,146)}=1.82$.16

Note: Values shown as % (n) unless otherwise noted. SET=supportive-expressive psychotherapy; MED=clinical management combined with pharmacotherapy; PBO=clinical management with placebo pill.

2.2. Treatments

All treatments were manualized and administered for 16 weeks. In the SET condition (n=49), patients received 20 sessions of time-limited manualized dynamic therapy for depression, which combines supportive techniques fostering a positive therapeutic relationship with a focus on understanding the patient's maladaptive relationship patterns (Luborsky, 1984; Luborsky and Mark, 1991). In the MED (n=51) and PBO (n=49) conditions, patients received supportive clinical management (CM) (Fawcett et al., 1987), and were treated with either Sertraline or placebo pill. Sertraline was initiated at 50 mg/day per os and raised in 50-mg increments to a maximum of 200 mg/day by week 4. Weeks 16–32 were defined as a follow-up period in which responders maintained their treatments and patients receiving placebo were switched to active medication. The study protocol from which these data were drawn was approved by the Institutional Review Board of the University of Pennsylvania. All participants provided written informed consent before any study procedures, in accordance with the US Department of Health and Human Services.

2.3. Measures

2.3.1. Causal attributions

The Attributional Style Questionnaire (ASQ; Peterson et al., 1982) was used to assess causal attributions. Participants were instructed to read scenarios with positive and negative outcomes, indicate their beliefs regarding the probable causes of the outcomes, and rate those causes on three 7-point scales: External/Internal, Unstable/Stable, and Specific/Global. Depressotypic attributional style was measured by summing scores separately for positive and negative items (Hollon et al., 1990; Teasdale et al., 2001). Cronbach alphas for both positive (.84–.93) and negative (.82–.93) attributions were satisfactory for all measurements points.

2.3.2. Relationship representations

The 101-item revised Central Relationship Questionnaire (CRQ-R; McCarthy et al., 2008), a self-report measure of the Core Conflictual Relationship Theme (CCRT; Luborsky and Crits-Christoph, 1998), was used to assess representations of the patients' wishes in interpersonal relationships, their perceptions of the others' responses to their wishes, and their own responses to others. Participants were instructed to rate on a 7-point Likert scale each of

four central relationships (with their romantic partner, mother, father, and same-sex best friend), based on three main relationship components: wishes (four W subscales, e.g., the wish to be intimate), responses from other (four RO subscales, e.g., the other is hurtful), and responses of self (five RS subscales, e.g., the self is autonomous). Scores for each interpersonal theme were aggregated across relationships, each patient having one score on each of 13 themes reflecting the patient's general representations of interpersonal relationships. Cronbach alphas for all themes in all measurement points ranged between .50 and .99.

Because of the low reliability of some of the subscales, we examined whether different subscales could be combined to create a factor solution that demonstrates a better fit to the data. Following Luborsky and Crits-Christoph (1998), we examined the relevance of a two-factor solution to the CRQ. We submitted the CRQ to a confirmatory factor analysis (CFA) based on its subscales, using the PROC CALIS procedure in SAS version 9.2. The hypothesized two-factor model fit the data well. The two factors accounted for 72% of the subscale variance and corresponded to the two theoretical factors of positive and negative representations. The same two factors were replicated across the four time points. The reliability of each factor was high ($\alpha=.98$ for the positive and $\alpha=.97$ for the negative representations factor). Therefore, we used the two factors in all analyses.

2.3.3. Outcome

We assessed the severity of depressive symptoms with the 17-item clinician-administered semi-structured interview version of the Hamilton Rating Scale for Depression (HRSD17; Hamilton, 1967). Total scores ranged from 0 to 52, with higher scores indicating greater severity of depression. Inter-judge reliability, as assessed by intraclass correlations, was .92.

2.4. Procedure

Assessments of each of the two mechanism variables (ASQ and R-CRQ) and of the outcome variable (HRSD) were collected at four time points: intake, mid-treatment (week 8), end of treatment (week 16), and follow-up (week 32).

2.5. Data analytic plan

Because of the hierarchical structure of the data (measures nested within individuals), general mixed models were estimated using PROC MIXED in SAS for multilevel modeling (Littell et al., 2006). This approach permits flexibility in the assumptions made about the covariance structure of repeated assessments.

To examine whether the two theory-driven potential mechanisms serve as mechanisms of change in the study, we followed a two-step approach (Barber et al., 2000; Gibbons et al., 2009b). First, we explored which mechanisms changed during treatment. Second, for mechanisms that changed during treatment, we examined whether earlier levels of the mechanism predicted later symptomatic levels, controlling for earlier symptomatic levels. Thus, we assessed whether patient scores in each mechanism variable predicted subsequent symptom levels.¹

To examine whether the two mechanisms changed over time, we used a general mixed-model approach for each mechanism. In

¹ Generally, to examine mechanisms of change one considers using mediation models (Baron and Kenny, 1986; Preacher et al., 2010). But because the theoretical question in this paper regards mechanisms of change in equally-effective treatments, a mediation model is not optimal: the treatment type does not serve as a main predictor in the model, whereas time does. Therefore, we conducted lagged analysis, a method used to examine temporal precedence between two variables (Collins and Sayer, 2001).

each model, at Level 1 (within-subject), time was entered as a predictor. At Level 2, treatment condition was added. Next, we examined whether levels of the mechanism variables predict subsequent levels of symptoms. To reduce the concern about reverse causation between mechanism and symptoms (Barber, 2009; DeRubeis and Feeley, 1990; DeRubeis et al., 2005), we entered the score of the outcome variable of the previous measurement point into the analyses. This allowed us to assess whether the levels of the mechanism variables predicted subsequent symptomatic levels throughout treatment and follow-up, controlling for prior levels of symptoms (Bolger and Laurenceau, 2013; Collins and Sayer, 2001). At Level 1 of the models (within-subject), we predicted the outcome at Time $t+1$ from each mechanism variable at Time t , and the symptomatic levels at Time t (Donegan and Dugas, 2012). To assess whether the type of treatment qualified these effects, we added an interaction with treatment condition. We ran all models twice: once across treatment conditions, to examine changes in the mechanisms across all three types of treatment, and once in the type of treatment entered into the model, to examine any possible differences between treatments.

Because previous findings showed that the decrease in HRSD in this dataset resulted in a linear trend over log of time (Barber et al., 2012), the current analyses used exponential time intervals ($T=0, 8, 16, 32$), so that changes in HRSD were constant between these time points (Zilcha-Mano et al., 2014a, 2014b).

3. Results

3.1. Preliminary results

Positive relational representations were significantly associated with positive and negative causal attributions across time points ($r_{(311)}=.26, p < .0001$ and $r_{(310)}=-.21, p < .0001$, respectively) and negative relational representations were significantly associated with positive and negative causal attributions ($r_{(311)}=-.22, p < .0001$ and $r_{(310)}=.38, p < .0001$, respectively). Table 2 presents the estimated marginal means for causal attributions and relationship representations specified for treatment condition and time point.

Table 2
Estimated marginal means for causal attributions and relationship representations specified by treatment group and time point.

	MED	SET	PBO
Positive relationship representations			
Time=0	5.00	4.78	4.95
Time=8	5.11	4.83	4.99
Time=16	5.21	4.87	5.02
Time=32	5.43	4.97	5.10
Negative relationship representations			
Time=0	2.83	3.03	2.84
Time=8	2.79	3.02	2.83
Time=16	2.75	3.00	2.83
Time=32	2.66	2.97	2.82
Positive causal attributions			
Time=0	4.91	4.95	4.63
Time=8	4.83	4.99	4.71
Time=16	4.75	5.04	4.79
Time=32	4.60	5.13	4.94
Negative causal attributions			
Time=0	4.47	4.75	4.51
Time=8	4.37	4.78	4.51
Time=16	4.26	4.81	4.51
Time=32	4.06	4.87	4.51

Note: SET=supportive-expressive psychotherapy; MED=clinical management combined with pharmacotherapy; PBO=clinical management with placebo pill.

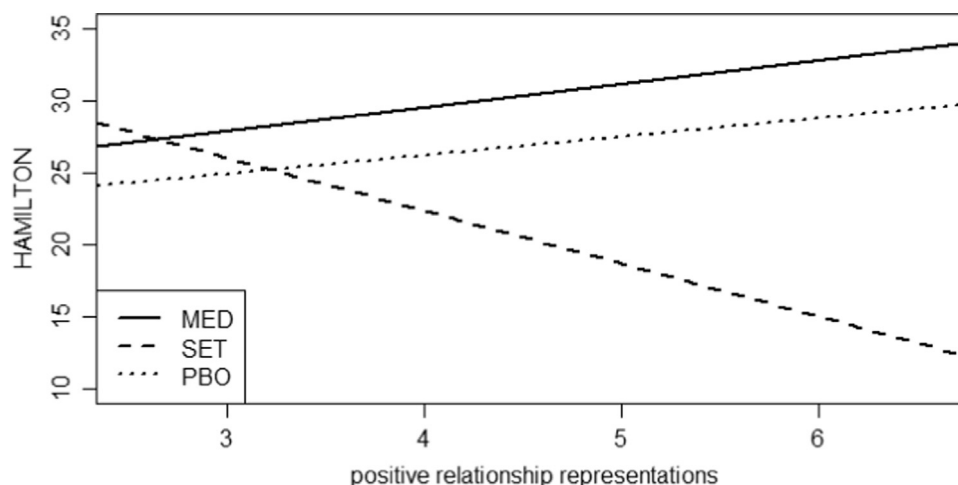


Fig. 1. The interaction between positive relationship representations and treatment conditions in predicting outcome (Hamilton scores). SET=supportive-expressive psychotherapy; MED=clinical management combined with pharmacotherapy; PBO=clinical management with placebo pill.

3.2. Which mechanisms changed during treatment?

Analyses showed that both causal attributions and relationship representations improved during treatment. Patients adopted more positive causal attributions over time [$B=.013$, $t_{(191)}=3.31$, $p=.001$]. Negative causal attribution did not change over time [$B=-.005$, $t_{(191)}=-1.65$, $p=.10$], and was dropped from further analyses. Patients adopted more positive interpersonal representations over time [$B=.007$, $t_{(198)}=3.55$, $p=.0005$], and less negative interpersonal representations [$B=-.004$, $t_{(198)}=-2.26$, $p=.02$]. No significant interactions emerged between change in mechanisms over time and type of treatment (all p 's > .33).

3.3. Do the potential mechanisms predict subsequent changes in symptoms?

Only mechanisms that showed changes over time were tested as potential mechanisms of change, resulting in an examination of two relational representation scores and one score of causal attribution. Patients who endorsed more symptoms at one point in time, were also more likely to endorse higher levels of symptoms at subsequent times. More important, a main effect was found for negative representations: reduction in negative representation predicted subsequent reduction in symptoms [$B=1.97$, $t_{(70)}=2.15$, $p=.03$]. No significant main effect was found for positive relationship representations [$p=.74$]. However, as shown in Fig. 1, a significant interaction was found between positive relationship representations and treatment condition [$F_{(2,56)}=3.19$, $p=.04$]. Greater improvement in positive relationship representations predicted greater symptom reduction in SET than in PBO [$B=-4.97$, $t_{(56)}=2.18$, $p=.02$], but not in MED vs. PBO [$B=0.33$, $t_{(56)}=1.71$, $p=.84$].² No significant interaction or main effect were found for positive causal attributions [$F_{(2,55)}=0.53$, $p=.59$ and $B=-.94$, $t_{(71)}=-1.52$, $p=.13$, respectively].

The main outcome study on this data found significant differences in outcomes for minority and gender in predicting differences between treatments. SET was more efficacious for minority men than MED and PBO. PBO was more efficacious for white men

than MED and SET. For white women, MED and SET were more efficacious than placebo. No differences between treatments were found for minority women (Barber et al., 2012). Therefore, we redid all the analyses controlling for minority status, gender, and the interaction between them. The findings of these analyses remained similar to those reported above (Table 3 in the Supplemental Material).

4. Discussion

The present study evaluated two theory-driven mechanisms of change related to mental representations: changes in causal attributions and changes in relationship representations, in SET and in clinical management with medication or placebo (CM+MED/PBO). Changes in relationship representations are at the heart of one of the treatments we examined (SET), but changes in causal attributions are not key to any of the examined treatments (rather to cognitive treatment). Therefore, we were able to examine whether a mechanism of change (in this case, changes in causal attributions) can act as such even in treatments that were not deliberately designed to focus on such mechanism (Barber et al., 2005). We were also able to examine whether a mechanism of change (in this case, changes in relationship representations), which is the focus of one of the treatments we examined (SET) and not of the others, has an effect only on that treatment.

Our findings revealed that both mechanisms changed significantly across treatments. Patients adopted more positive causal attributions, and showed more positive and less negative representations of their interpersonal relationships across treatment conditions. This finding appears to support the existence of common processes that occur in distinct treatments for depression. However, evidence that these changes occur does not necessarily mean that they are responsible for the reduction of depressive symptoms observed. Changes in causal attributions and in relationship representations may also be a “byproduct” of symptomatic change. Therefore, it was essential to further investigate whether changes in such mechanisms were predictors or products of symptomatic change.

When examining whether changes in causal attributions and in relationship representations predicted symptomatic change, we found that only relationship representations predicted subsequent levels of symptoms when controlling for prior symptomatic levels. A decrease in the patients' negative views on their interpersonal relationships predicted fewer subsequent symptoms of depression

² A power analysis demonstrated that the sample size was adequate for detecting the effect size of obtained interactions with treatment condition. We conducted a Monte Carlo simulation analysis (10,000 repetitions) in Mplus statistical modeling software (Version 6.12; Muthén and Muthén, 2011), using parameters estimates obtained by our model, to determine a power of significant interaction effect between the potential mechanisms and treatment condition in predicting outcome (Muthén, 2002). The estimated power effect of the interaction was 0.836.

across all treatment conditions. Furthermore, an increase in positive relationship representation predicted symptom improvement only in patients receiving supportive-expressive psychotherapy, but not in those receiving medication/placebo together with clinical management. Changes in causal attribution did not predict symptom change in any treatment condition.

The finding that a reduction in negative relational representation was a common mechanism of change across all treatment conditions may appear surprising at first glance. In particular, one may expect such findings for SET, but not for CM. To better understand this finding, a brief description of the CM manual used in this RCT is warranted. CM sessions followed the manual developed for the Treatment for Depression Collaborative Research Program (Fawcett et al., 1987). Although formal re-educative and reconstructive psychotherapeutic techniques are prohibited in CM, supportive interventions (helping patients express their experiences and emotions, pointing out or acknowledging gains, reinforcing accomplishments, and offering empathy, warmth, and hope) are allowed. The manualized CM can be regarded as a relatively potent form of supportive therapy. Repeated RCTs for depression have shown its efficacy in bringing symptomatic change at least to the level of other “active treatments” (Elkin et al., 1985; Frank et al., 2008; McIntosh et al., 2005), and some studies show that no differences could be detected between this supportive CM, interpersonal psychotherapy, or cognitive-behavioral therapy across different mode-specific outcomes (Imber et al., 1990). Therefore, building on previous findings, the supportive nature of CM may have helped patients be less depressed by decreasing their negative expectations about interpersonal relationships, similarly to SET. As it has been well articulated in the past, a benevolent interpersonal experience with another person may oppose previous adverse interpersonal experiences and have a potent effect on the process of therapeutic change (Bowly, 2005; Kohut, 1984; Mallinckrodt, 2010). This finding agrees with the common factor approach to psychotherapy (Castonguay, 2011; Laska et al., 2014; Stricker and Gold, 2002), which directs attention to the preponderance of commonalities among treatments (Weinberger, 1995).

We also found support for a treatment-specific mechanism of change. For patients who received SET but not CM, an increase in positive relationship representation predicted further reductions in depressive symptoms. Perhaps a supportive relationship with a provider (CM or SET) went far enough to reduce negative expectations about relationships, but SET offered additional ingredients, not available in CM, to increase positive representations of interpersonal relationships in order to effect symptom change. For example, deliberate work on representations of self and others and on patient wishes regarding the relationship with significant others may have contributed to building representations of others as loving and of the self as a valuable and lovable person. The process of building positive representations in interpersonal relationships may therefore require deeper, more deliberate work on interpersonal representations than is necessary to reduce levels of negative representations (Book, 1998).

Improvement in causal attributions was not found to be related to symptomatic change. Given that causal attribution is theoretically a mechanism of change in cognitive therapy, current findings suggest that it is not a common mechanism of change across all treatments of depression. It may be argued that changes in negative causal attributions require deliberate focused work, such as in cognitive treatment. This finding is consistent with previous studies suggesting that changes in causal attributions are specific mechanisms of change for cognitive treatment and not for antidepressant medications (DeRubeis et al., 1990). A growing literature also questions whether targeting negative cognitions is necessary for creating a treatment response in depression

(e.g., Dimidjian et al., 2006). Our result that all patients improved in positive causal attribution may be a byproduct of reduction in depressive symptoms, at least in the non-CBT treatments examined in the present study. Furthermore, it might be suggested that there are multiple and perhaps overlapping pathways to symptom change in depression that can be preferentially engaged by different treatments.

The medication condition engaged another potential mechanism that we could not measure: serotonergic changes in the brain (Stahl, 1998; Vaswani et al., 2003). Animal and human studies have documented differences after administration of antidepressant compounds, which may be related to change in depressive symptoms. Furthermore, psychodynamic techniques may alter brain functioning (Buchheim et al., 2012; Karlsson, 2011; Loughhead et al., 2010; Roffman et al., 2014), and symptom change may occur through changes in neurotransmitter processes. Some theorists have proposed that serotonin changes resulting from antidepressant medication may directly alter cognitive biases and relationships as well through alterations in emotion processing in the brain (Warren et al., 2015). The role of neurochemical and brain pathways remains an important subject in the study of depression treatment mechanisms.

It is important to highlight that the preset findings were obtained using a relatively conservative methodological approach. To determine whether cognitive and relational mechanisms have a causal effect on outcome, to date researchers have employed limited methods for examining time precedence, including (a) using time overlap between predictor (mechanism) and outcome (symptomatic change) variables (e.g., Coleman et al., 2010), or (b) limiting the symptomatic change examined to follow-up only, when unable to do so in the active part of treatment (e.g., Gibbons et al., 2009a). The methodology used in the present study enabled us to address some of the limitations of previous research by performing a more rigorous test of the temporal relation between mechanisms of change and outcome, moving one step forward in addressing causality. Furthermore, we measured the mechanisms and depressive symptoms using self-report and clinician ratings, respectively, avoiding inflated association due to shared method variance. An additional strength of the present study is the high representation of ethnic minorities and patients with diagnostic comorbidity, which is generally lacking in comparable depression trials.

The present study focused only on two possible mechanisms of change across supportive (with medication or placebo) and supportive-expressive treatments for depression. Future studies should incorporate other specific mechanisms, such as those relevant to case management, placebo, and SSRI effects. For example, expectancy can serve in examining mechanisms specific to the placebo condition (Rutherford and Roose, 2013). In addition to considering other treatments and mechanisms of change, it is important to acknowledge that the two mechanisms under investigation are significantly associated and may influence each other or other mechanisms of change, as is the case for many mechanisms of change in treatment (Gibbons et al., 2009a), or be influenced by a third variable (e.g., spontaneous recovery over time). Furthermore, the sample size used in this study may not provide sufficient statistical power to detect small effect sizes of interaction with type of treatment. Future studies may compare the mechanisms of change across more distinct treatments, such as SET and CBT. Although the findings of the present RCT of no differences between treatment conditions for depression are consistent with previous meta-analyses and with psychopharmacological studies attributing the rise in failed trials to increases in placebo effect (Rutherford and Roose, 2013), it may be argued that the active treatments (SSRI and SET) were not effective enough in reducing symptoms, which may have affected the present

findings. Therefore, further studies are needed to examine the existence of common and unique mechanisms in RCTs in which psychotherapy and medication showed superiority over placebo. Moreover, similarly to other studies that include follow-up data, it is difficult to know whether patients indeed did not seek other treatment during the time under study. Finally, although in this paper we adopt the general convention of treating symptomatic change as the gold standard for therapeutic change, we acknowledge that changes in attributional and relational representations can be an important outcome in themselves (Zilcha-Mano et al., 2014b).

Many attempts have been made to discover and characterize the mechanisms of change in psychotherapies for depression, but little evidence-based account of the temporal precedence between therapeutic mechanisms and symptom improvement has emerged (Lorenzo-Luaces et al., 2014). The present study identifies the decrease in negative relational representations as a common mechanism of change in the examined treatments for depression (SET and CM+MED/PBO), and the increase in positive relational representations as a specific mechanism of change in SET. Identifying the common and specific mechanisms of change in today's therapeutic interventions is essential for shaping tomorrow's most effective, personalized treatments (DeRubeis et al., 2014).

Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jad.2015.12.073>.

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