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Does alliance predict symptoms throughout treatment, or is it the other way around?

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Abstract

Objective—Scholars increasingly recognize that therapeutic alliance and symptomatic change are associated with one another. A common assumption is that alliance predicts symptomatic change. However, the issue is far from settled. One challenge in determining the causality is the establishment of temporal precedence showing that alliance, as opposed to previous symptomatic change, drives subsequent symptomatic reduction.

Method—To make further advances in untangling this chicken and egg question, we employed autoregressive cross-lagged modeling over 4 time points in a sample of 149 depressive patients receiving supportive-expressive psychotherapy or clinical management combined with pharmacotherapy or clinical management combined placebo.

Results—Using this methodology, we found that both alliance and symptoms across treatment made significant and unique contributions in predicting subsequent symptomatic levels throughout treatment. Additionally, alliance, but not symptoms, predicted subsequent alliance levels. No differences were found between treatments.

Conclusions—Our findings imply that alliance temporally precedes symptomatic levels throughout treatment.

Keywords

Alliance; depression; psychotherapy outcome; psychodynamic psychotherapy

The relation of the therapeutic alliance with outcome is one of the most researched topics in the field of psychotherapy, with three decades of empirical research consistently linking these two variables. Horvath, Del Re, Flückiger and Symonds (2011) showed that the correlation between alliance and psychotherapy outcome across 14,000 treatments was small to moderate ($r = .27$) but reliable, with no significant differences among treatment orientations. Based on these and similar findings, many scholars have posited that alliance is

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an active ingredient in therapy, meaning it is therapeutic in and of itself, and accounts for at least part of the variance in treatment outcome (e.g., Flückiger, Del Re, Wampold, Symonds, & Horvath, 2012).

However, in recent years, there have been several challenges to the idea that greater alliance causes good outcomes. Namely, some researchers have proposed that good alliances may be the result of changes in symptoms, rather than the other way around (e.g., Barber, 2009; DeRubeis, Brotman, & Gibbons, 2005). Support of this hypothesis is found in studies of the alliance-outcome correlation that accounted for symptomatic change prior to alliance measurement. More specifically, some studies showed that early symptomatic change predicted alliance and that only early symptomatic change (and not alliance) could predict subsequent changes in symptoms (e.g., Barber et al., 1999). Other studies found that alliance still makes a unique contribution to the prediction of outcome, even after controlling for early symptomatic change (e.g., Barber, Connolly, Crits-Christoph, Gladis, & Siqueland, 2000), and that early symptomatic change does not necessarily drive subsequent changes in alliance (e.g., Klein et al., 2003)(for a review, see Crits-Christoph, Gibbons, & Mukherjee, 2013). Taken together, these studies, which control for symptomatic change early in treatment, have called into question the direction of causality between alliance and symptomatic change.

Most if not all of the studies reviewed by Crits-Christoph et al. (2013) focused on the ability of the alliance at only a single time point to predict symptomatic change. Recently, researchers have started to implement analytic methods that enable us to assess the alliance-outcome relation at more than one time point and therefore facilitate the examination of reverse causality between alliance and symptoms. Such analytic methods include autoregressive cross-lagged modeling (ARCL), which enables the exploration of temporal precedence between variables examined longitudinally (e.g., Kenny & Harackiewicz, 1979). While this method has been successfully applied to many research areas both outside of psychology and within it (including the field of psychotherapy research, e.g., Brossart, Willson, Patton, Kivlighan, & Multon, 1998; Donegan & Dugas, 2012; Liverant, Suvak, Pineles, & Resick, 2012; Smits, Rosenfield, McDonald, & Telch, 2006; Zilcha-Mano, Dinger, McCarthy, Barrett, & Barber, in press), it has seldom been used to investigate the temporal precedence between alliance and symptoms. Using the ARCL and similar methods, preliminary studies have found that in a very brief treatment (Falkenström, Granström, & Holmqvist, 2013) and in a relatively small sample size of patients treated by inexperienced therapists (Crits-Christoph, Gibbons, Hamilton, Ring-Kurtz, & Gallop, 2011), alliance may still predict symptoms while controlling for the previous symptomatic levels in individual psychotherapy (for the use of similar methods in group psychotherapy and family therapy, see Tasca & Lampard, 2012 and Marker, Comer, Abramova, & Kendall, 2013, respectively).

Building on these findings, we used the ARCL in the current study to examine whether (a) previous alliance levels could predict subsequent symptomatic levels throughout treatment while controlling for prior symptomatic levels (see Figure 1a); and (b) whether previous symptomatic levels could predict subsequent alliance levels throughout treatment, while controlling for prior assessments of alliance (see Figure 1b). In this study the temporal relationship between alliance and symptoms was examined across three treatments—

supportive-expressive psychotherapy (SET), supportive clinical management combined with pharmacotherapy (CM+MED), and supportive clinical management combined with placebo (CM+PBO)—in a population of patients with major depressive disorder (MDD). Treatment type was examined as a potential moderator of the association between alliance and symptoms.

Method

Participants

Patients diagnosed with depression were randomly assigned to one of three treatment conditions: SET, CM+MED, or CM+PBO (for more details see Barber, Barret, Gallop, Rynn, & Rickels, 2012). Of the 156 patients randomized in the original study, 149 filled out at least one alliance questionnaire and were included in this study. The mean age was 37.8 ($SD = 12.1$) and 92 participants (60.1%) were female. About half (49%) were Caucasian, 43.8% were African Americans, and the rest were Latino (5.2%) or Asian (2%). At intake, all patients met the DSM-IV criteria for Major Depressive Disorder, and 85% had at least one comorbid disorder. Axis-1 comorbidities included anxiety disorders (45%) and current substance abuse or past dependence disorder (35%). In addition, 46.3% had a comorbid Axis-2 personality disorder.

Treatments

All treatments were administered for 16 weeks. In the SET condition ($n = 49$), patients received a time-limited dynamic therapy adapted for depression that focused on understanding the patient's problematic relationship patterns and helping them work through core relational difficulties within the context of supportive techniques aimed at establishing a positive relationship (Luborsky, 1984; 1995). Patients in this condition received 20 sessions of individual psychotherapy, twice weekly for the first month and then weekly for the remaining 3 months. In the other two conditions, patients received clinical management (CM) combined with either Sertraline (CM+MED) or a placebo pill (CM+PBO). Patients in both of these conditions met weekly with their psychopharmacotherapists for the first six weeks and could switch to every other week for the remaining study period if patients' condition warranted it. In CM (Fawcett et al., 1987) formal psychotherapeutic techniques were prohibited, but supportive interventions (such as helping patients express their emotions and experiences, acknowledging gains, reinforcing accomplishments, and offering empathy and warmth) were allowed. The study was approved by the university IRB.

Measures

Therapeutic alliance—The quality of the therapeutic alliance was assessed with the 12-item patient-rated version of the Working Alliance Inventory (WAI; Tracey & Kokotovic, 1989).¹ Items were rated on a 7-point Likert scale ranging from 1 (never) to 7 (always). In the current study the internal reliability range for the four time points was .92-.95.

¹We also used the California Psychotherapy Alliance Scale (CALPAS; Gaston & Marmar, 1994) to measure the alliance. However, since the findings from both questionnaires were very similar, we report on only on the WAI, the most widely used measure.

Depressive symptoms—The severity of depressive symptoms was assessed with the 17-item clinician-administered semi-structured interview version of the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967). Total scores ranged from 0 to 52, with higher scores indicating a greater severity of depression. Inter-judge reliability for the current study as assessed by intraclass correlation (ICC [2,1]; Shrout & Fleiss, 1979) was .92.

Procedure

For the aims of this study, we chose to assess the therapeutic alliance and symptomatic level at four scheduled time points and used the closest available assessment points for each patient: Week 2 (beginning of treatment, $M = 2.13$, $SD = 0.34$), Week 4 (early treatment, $M = 4.26$, $SD = 0.52$), Week 8 (mid-treatment, $M = 8.15$, $SD = 0.46$) and Week 16 (end of treatment, $M = 16.08$, $SD = 0.57$).

Statistical Analyses

Due to the hierarchical structure of the data (measures nested within individuals), we chose to use general mixed modeling using PROC MIXED in SAS for multilevel modeling (Littell, Milliken, Stroup, Wolfinger, & Schabenberger, 2006). This approach permits flexibility in the assumptions made about the covariance structure of the repeated assessments. To examine the bidirectional association between therapeutic alliance (as measured by the WAI score) and symptoms (as measured by the Hamilton score, HRSD) over time, we employed autoregressive cross-lagged modeling (ARCL; e.g., Collins & Sayer, 2001). The ARCL allowed us to examine whether the previous level of WAI ($WAI_{[T-1]}$) predicted the subsequent HRSD level ($HRSD_{[T]}$) throughout treatment while controlling for the previous level of HRSD ($HRSD_{[T-1]}$), as illustrated in Figure 1a. The ARCL also enabled us to test whether $HRSD_{(T-1)}$ predicted subsequent WAI level ($WAI_{[T]}$) throughout treatment, controlling for the previous level of WAI ($WAI_{[T-1]}$), as illustrated in Figure 1b. Due to previous findings showing that the decrease in HRSD in this dataset resulted in a linear trend over the log of time (Barber et al., 2012), the current analyses used exponential time intervals ($T = 2, 4, 8, 16$), so that the changes in HRSD were constant between these time points.

In order to examine the bidirectional association between WAI and HRSD over time, we introduced $WAI_{(T-1)}$ and $HRSD_{(T-1)}$ in Level 1 of the models (within-subject) as well as the dependent variable at the subsequent time (either $WAI_{[T]}$ or $HRSD_{[T]}$, depending on the model; see Collins & Sayer, 2001 for more information). Type of treatment was added as a three-level categorical variable of condition (SET, CM+MED, CM+PBO) to Level 2. Since the association between HRSD and WAI may differ at the different conditions or at the different time points, the interaction of $WAI_{(T-1)}$ (or $HRSD_{[T-1]}$) and condition as well as $WAI_{(T-1)}$ (or $HRSD_{[T-1]}$) and time were added to the model predicting $HRSD_{(T+1)}$ (or $WAI_{[T+1]}$) while regarding time as a categorical variable (Model 1). The same model was repeated, this time without the two interactions (Model 2). If the inclusion of these interactions will improve the model by a -2 log-likelihood χ^2 test, those interactions will be further investigated, if not, Model 2 will be used to examine the bidirectional association between HRSD and WAI.

Results

We compared the fits of model 1 (with the two interactions above) and model 2 (without the interactions) using the log-Likelihood test. Results show that the models including the two interactions of the predictors with condition and with time did not contribute significantly to the model fit and therefore the models without the interactions were used in the subsequent analyses. Specifically, for $HRSD_{(T)}$ as the predicted variable, the change in $-2*\log$ -likelihood of the two models was $\chi^2(4) = 2.6, p = .63$. Similarly, for the $WAI_{(T)}$ as the predicted variable, the change $-2*\log$ -likelihood of the two models was $\chi^2(4) = 3, p = .58$. Examining each interaction separately using the Wald test also showed that all interactions were non-significant for both predicted variables. Therefore, Model 2 (without interactions with treatment condition and time) was used while predicting $HRSD(T)$ and while predicting $WAI_{(T)}$.

We then tested which covariance structure showed the best fit (i.e., the lowest AIC) in models predicting $WAI_{(T)}$ or $HRSD_{(T)}$. In predicting $WAI_{(T)}$, an unstructured covariance matrix exhibited the lowest AIC. In predicting $HRSD_{(T)}$, an autoregressive covariance matrix provided best fit.

Did alliance predict subsequent symptomatic level?

Participants who showed higher $HRSD$ levels at one time point also showed higher $HRSD$ levels at subsequent times, $\beta = .82, SE = .04, t(111) = 19.61, p < .0001$. More importantly, higher WAI at a given time point predicted a lower $HRSD$ level at the subsequent time point, $\beta = -.05, SE = .01, t(111) = -2.43, p = .01$, while controlling for previous levels of $HRSD$ (measured at the same time point as the WAI predictor, see Figure 1a).

Did symptoms predict subsequent alliance level?

Participants who showed higher WAI levels at a given time point also showed higher WAI levels at the subsequent time point, $\beta = .81, SE = .03, t(102) = 21.68, p < .0001$. Of importance, $HRSD$ levels at one time point were not associated with WAI levels at the subsequent time point, $\beta = .01, SE = .07, t(102) = 0.18, p = .86$, while controlling for previous levels of WAI (measured at the same time point as the $HRSD$ predictor, see Figure 1b).

Discussion

Identifying the determinants of therapeutic change and their sequence constitutes one of the core aims of psychotherapy research (e.g., Barber, 2009). One promising path in the process of tackling this issue lies in investigating a possible causal role of the therapeutic alliance as a common factor in bringing about therapeutic change across different treatment modalities (DeRubeis et al. 2005). Although important knowledge has been gathered on the alliance-symptoms association throughout the last three decades, it is an open question whether alliance actually predicts symptomatic change or whether it is merely the result of previous symptomatic change. In the current study ARCL method was used to help clarify the temporal precedence between alliance and symptomatic levels.

The findings of the current study demonstrate the theoretically expected associations between alliance and outcome while controlling for the option of reversed causality. Specifically, our main finding using ARCL modeling indicates that stronger alliance predicts lower levels of depressive symptoms while accounting for temporal precedence between alliance and symptoms throughout treatment. This finding was consistent across all three treatment conditions. The ARCL allowed us to move beyond the common, but somewhat narrow focus on early alliance (e.g., Session five) to the therapeutic alliance as it develops throughout treatment. The ARCL methodology also allowed us to address some of the limitations of previous research by performing a more systematic test of the temporal relation between alliance and symptoms. So far, in addressing the issue of whether alliance has a causal impact on outcome, researchers have employed a variety of methods to examine time precedence. These attempts have often resulted in inconsistent findings, with the correlation between the alliance and subsequent symptoms ranging broadly, from .07 to .42 (Crits-Christoph et al., 2013). Although previous studies on the associations between alliance and symptoms have varied with respect to their designs, measures, and analytic strategies, most of them have not measured hypothesized causes (alliance) and effects (symptoms) in the appropriate temporal order throughout treatment, while controlling for autoregressive effects (i.e., the previous values of both the predictor and the dependent variables).

Examining the temporal precedence between alliance and symptoms enables us to more carefully address one of the oldest questions in psychotherapy: does the therapeutic relation really have an effect on outcomes? The current study lends some support to the theoretical view of the alliance as a curative factor (Norcross, 2002; Rogers, 1951) which precedes therapeutic outcome in both dynamic therapy and supportive clinical management, although the specific underlying mechanisms cannot be elucidated by the current study.

The finding above regarding the potential role of the therapeutic alliance in predicting subsequent symptomatic levels across both supportive CM and SET might appear surprising if one expects the psychodynamic work on interpersonal relationships to play a causal role in the ability of alliance to bring about symptomatic change. However, our finding is in line with studies demonstrating the role of the alliance as a common factor across different treatment orientations, including supportive treatment (Horvath et al., 2011). It will be interesting to see in future research whether in long-term dynamic treatment the interpretative components may have an additive role to the ability of alliance to predict symptomatic change, beyond the effects of the supportive components. Clearly, examination of the temporal precedence between alliance and symptoms throughout treatment in other treatment approaches, such as cognitive behavioral therapies and interpersonal psychotherapy, is needed.

Our second main finding was that symptomatic levels did not predict subsequent alliance levels while controlling for the previous alliance levels. This finding was also consistent across all three treatment conditions. Previous studies yielded mixed results while testing the ability of symptomatic levels to predict alliance levels (e.g., Barber et al. 2000; DeRubeis & Feeley, 1990; Klein et al., 2003). It is important to stress that those studies did not examine the temporal precedence between alliance and symptoms throughout treatment while

controlling for autoregressive effects (previous values of both the predictor and the dependent variables). We could find only two studies that did examine directly the ability of symptoms to predict alliance throughout individual treatment while controlling for previous alliance levels. Specifically, while controlling for the previous alliance levels, Crits-Christoph et al. (2011) found that symptoms can predict alliance only in specific time points in treatment and not in others (in sessions 10–16 but not in sessions 3–9). Falkenström et al. (2013), on the other hand, did find that symptoms can predict alliance after controlling for previous alliance levels in a very brief treatment (with a median of four sessions). Therefore, it seems that the ability of symptoms to predict alliance warrants further close attention in the future and also may require more frequent assessments of alliance and symptoms during the course of treatment.

The results of the present study represent only initial steps in the systematic process of inferring causality between alliance and improvement in depressive symptoms, as the exact manner in which alliance and symptoms interact to produce benefits to patients cannot be fully determined based on any one study. It is important to stress that while the analyses conducted in this study were aimed at ruling out the possibility of reversed causality, our results do not necessarily imply a causal role, since unmeasured third variables may still influence both alliance and symptoms. Future studies measuring potential theoretically-relevant variables to explain the mechanisms behind this association, at exactly the same time points as alliance and symptoms are examined, will be helpful in the systematic process of inferring causality between alliance and symptoms. Another limitation of the current study is the use of only four time points. Although the use of three or four time points is both common in the field of clinical psychology and statistically adequate (Kenny & Harackiewicz, 1979), the use of additional times would have been preferable and should be implemented in future studies. Furthermore, since the current study focused only on a moderately sized sample of depressed patients receiving dynamic treatment and supportive clinical management, additional research should make use of larger sample sizes, and examine the temporal precedence between alliance and symptoms with other clinical populations, with different therapeutic orientations, and with alternative analyses methods, such as Structural Equation Modeling.

In conclusion, the current study demonstrates that while examining temporal precedence between alliance and symptoms amongst a population of depressive patients receiving SET, CM+MED and CM+PBO, alliance is the predictor (and not the product) of subsequent symptomatic levels. In the current study the alliance and depressive symptoms were measured independently using different methods in order to avoid inflated association due to shared method variance. The specific methodology used in the current study, ARCL, enabled us to address time sequences between alliance and symptoms while controlling for reverse causality. We hope that this methodology, which has been widely used in many fields of psychology and even inside the field of psychotherapy research, will continue to be employed in future studies on the alliance and outcome associations.

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Figure 1.

The examination of the temporal precedence between alliance and symptomatic levels throughout treatment (a) Predicting depressive symptoms at the current time point ($HRSD_T$) from therapeutic alliance (WAI_{T-1}) and depressive symptoms ($HRSD_{T-1}$) at the previous time point, and (b) predicting alliance at the current time point (WAI_T) from alliance (WAI_{T-1}) and depressive symptoms ($HRSD_{T-1}$) in the previous time point $HRSD$ = Hamilton; WAI = therapeutic alliance; β = the beta's in the model are unstandardized and therefore are not comparable, $*** p < .001$, $* p < .05$.