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OUTCOME TRAJECTORIES AND MEDIATION IN PSYCHOTHERAPEUTIC TREATMENTS OF MAJOR DEPRESSION

Trajectories and mediators of change were investigated in a process-outcome study. Patients were allocated at random to psychoanalytic therapy (PA) or psychodynamic therapy (PD), and later to cognitive-behavioral therapy (CBT). Measurement points were at pre-treatment, during ongoing treatment, at post-treatment, and during a three-year follow-up. Outcome trajectories were assessed using the Beck Depression Inventory (BDI; Hautzinger et al. 1994), the Symptom Checklist 90 Revised Version (SCL-90-R; Franke 1995), and the Inventory of Interpersonal Problems (IIP; Horowitz, Strauss, and Kordy 2000). Therapeutic alliance and introject were tested as mediators, assessed using the Helping Alliance Questionnaire (HAQ; Bassler, Potratz, and Krauthauser 1995) and INTREX, introject surface (Tress 1993). Multilevel modeling was applied to estimate outcome trajectories and to test for mediation. Symptoms decreased in early and ongoing treatment in all treatment groups. After the end of treatment, depressive and general psychiatric symptoms continued to decrease in significantly greater degree in the PA group than in the PD and CBT cohorts. During early treatment, interpersonal problems decreased significantly more in those allocated to PD than in the PA and CBT groups. During ongoing treatment, improvement in

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interpersonal problems was significantly higher in the PA group than in the others and, compared to CBT, continued to increase significantly after termination. Mediational analyses revealed that neither introject affiliation nor therapeutic alliance mediated differential treatment effects.

Keywords: process-outcome, depression, mediator, introject, alliance

In recent decades, psychotherapy research has aimed at bridging the gap between process and outcome. To this end, a change process paradigm has been proposed (for a history of its development, see Knobloch-Fedders, Elkin, and Kiesler 2015). Going beyond the mere description of what happens in psychotherapy, the change process paradigm focuses on conceptually derived change events that can be meaningfully related to proximal and distal outcomes. Change process research is a systematic attempt at "identifying, describing, explaining, and predicting the effects of the processes that bring about therapeutic change over the entire course of therapy" (Greenberg 1986, p. 4) in the context of clinically meaningful units. Trajectories of change translate into this change process approach, capturing the individual therapeutic process by modeling multiple data points assessed during the course of treatment, as well as capturing discontinuities during the course of therapy as a starting point for a meaningful in-depth exploration of change patterns (see, e.g., Hayes et al. 2007). Another approach that translates into change process research is the investigation of mediators, "intervening variables that may account (statistically) for the relationship between the independent and dependent variable" (Kazdin 2007, p. 3). Mediators should be cogently selected constructs, based on a theory of change that cuts across all treatments investigated (Johansson and Høglend 2007; Kraemer et al. 2002). They have to be measured during treatment with a time-lagged model to strengthen causal inferences and must correlate with both treatment and outcome (Kraemer et al. 2002). Studies that include follow-up intervals long enough to take into account changes that continue to evolve after the end of treatment are still lacking (Kazdin 2007), as are studies providing a timeline to ensure that the mediator has changed before the outcome, which would strengthen the evidence for a causal relation (Kazdin 2007; Kraemer et al. 2002).

In previous publications, we have shown significant outcome differences between psychoanalytic therapy (PA), psychodynamic therapy

(PD), and cognitive-behavioral therapy (CBT) directly after treatment and at three-year follow-up (Huber et al. 2012, 2013).

From a process-outcome perspective, using data points during ongoing treatment, we now tried to elucidate the change processes that mediate differential treatment effects. Empirical process research indicates that therapeutic alliance is a potential mediator of outcome that cuts across all empirically supported treatments (see, e.g., Crits-Christoph, Connolly Gibbons, and Mukherjee 2013). However, it still is open to discussion whether improved therapeutic alliance is a consequence of symptom improvement or whether therapeutic alliance precedes symptom improvement (see Barber 2009; Zilcha-Mano et al. 2014). The concept of therapeutic alliance originates from the ego psychological school of psychoanalysis (see, e.g., Bibring 1937; Zetzel 1956), which emphasized "an actual alliance . . . formed essentially between the patient's reasonable ego and the analyst's analyzing ego" (Greenson 1965, p. 157) to achieve commonly agreed-upon goals. The introject, conceived as a subsystem within the system of self-representations (see, e.g., Jacobson 1964) is another putative mediator. According to interpersonal theory, it denotes an hypothesized personality structure comprising "a relatively stable conscious and unconscious repertoire of behavior directed by the self at the self" (Henry 1996, p. 1268). It includes self-appraisals, verbal and motor activities directed at the self, and self-images (Henry, Schacht, and Strupp 1990) and is central to the major interpersonal problems of an individual because it shapes maladaptive expectancies and behaviors between self and others. It cuts across PA, PD, and CBT because according to psychoanalytic/psychodynamic theory it can be viewed as a component of unconscious conflict (see, e.g., Strupp and Binder 1984); in CBT it can be viewed as a core belief underlying symptoms (Clark, Beck, and Alford 1999).

The aims of this study are (a) to model outcome trajectories of depressive symptoms, general psychiatric symptoms, and interpersonal problems for patients receiving PA, PD, or CBT, and (b) to test whether differences in these outcome trajectories between treatments are mediated by differences in change processes related to introjects and therapeutic alliance.

METHOD

Study Overview

The empirical basis of the study is the Munich Psychotherapy Study (MPS), a prospective, comparative process-outcome study that evaluates

the effectiveness of PA, PD, and CBT for a diagnostically homogeneous sample of depressed patients. For additional details about procedure, measures, and treatments, see Huber et al. (2012, 2013).

Participants

Patients seeking treatment for unipolar, single, or recurrent depression at the Outpatient Department for Psychosomatic Medicine and Psychotherapy, Technical University of Munich, were invited to participate in the study. Inclusion criteria required participants to have a primary diagnosis of a moderate or severe episode of major depressive disorder according to ICD-10 F 32.1/2 (World Health Organization 1993) or DSM-IV 296.22/23, a recurrent depressive disorder, current episode moderate or severe without psychotic symptoms (ICD-10 F 33.1/2 or DSM-IV 296.32/33), or a double depression (Keller, Hirschfeld, and Hanks 1997), and to be between twenty and fifty years of age. There should be no contraindication for any of the three treatments, no psychotherapeutic treatment for the two years previous, and no antidepressive medication during the four weeks before treatment. Exclusion criteria were bipolar affective disorder, depression due to somatic illnesses or diseases of the brain, and alcohol or substance dependence. All patients were requested to give written informed consent to be included in the study. The Ethics Committee of the Technical University of Munich approved the study protocol. Because financial resources were limited, randomized allocation started with psychoanalytic and psychodynamic therapy and included cognitive-behavioral therapy only later.

One hundred fifty patients with depressive symptoms were screened. Thirty-one were excluded because they did not meet the inclusion criteria or refused to participate in the study. One hundred nineteen patients were allocated to the three experimental groups. Seven patients did not contact the therapist and twelve did not begin treatment after five trial sessions (in Germany patients and therapists usually have five trial sessions before they sign a therapy contract); eight belonged to the PA group, four to the PD group, and seven to the CBT group. In accordance with a recommendation by Lambert and Ogles (2004), only patients who agreed to the therapy contract were included in the study. As a result, 35 PA patients,31 PD patients, and 34 CBT patients were followed up using an intent-to-treat approach.

Assessments and Measures

Patients received questionnaires including relevant measures every three months in CBT and every six months in PD and PA once the therapy

contract was established. This differential time schedule was applied because the treatments were expected to differ considerably in duration. Moreover, outcome variables were measured at the end of treatment, and at one-, two-, and three-year follow-up. Note that the three-year follow-up period seems of adequate duration (Roth and Fonagy 2005) to disentangle treatment effects from the natural course of depressive disorder. Outcome measures were the Beck Depression Inventory (BDI; Hautzinger et al. 1994), the Global Severity Index (GSI) of the Symptom Checklist 90 Revised (SCL-90-R; Franke 1995), and the Inventory of Interpersonal Problems (IIP; Horowitz, Strauss, and Kordy 2000). These were employed to grasp symptomatology and the interpersonal domain (for a more detailed description, see Huber et al. 2012).

Therapeutic alliance was assessed with the Helping Alliance Questionnaire (HAQ; Bassler, Potratz, and Krauthauser 1995), an 11-item self-rating instrument used to estimate the patient's and the therapist's experience of the therapeutic relationship. Because their perspectives differ significantly, we used both patient and therapist forms (Lambert 2013). The questionnaire comprises two types of experiences: perceived helpfulness of the therapist (5 items) and the patient's bonding with the therapist (6 items). Each item is rated on a 6-point Likert scale with a range from 1 ("Yes, I strongly feel that it is true") to 6 ("No, I strongly feel that it is not true"). In a review, internal consistency and interrater reliability were regarded as acceptable, and discriminant validity as demonstrable; convergent validity, however, was deemed less robust (Elvins and Green 2008). In order to avoid confusion between relationship and outcome (see, e.g., Hatcher and Barends 1996), we restricted our analyses to the bonding subscale. Note that the HAQ was not included in the follow-up assessments.

We also applied the introject surface of the INTREX, short form (Tress 1993), an 8-item self-rating instrument based on the Structural Analysis of Social Behavior (SASB; Benjamin 1974), to be filled out for times identified by the subject as best and worst. By best and worst times is meant specific times occurring a few days, weeks, or months ago but not as long ago as a year. We are aware that for psychoanalysts the concept of introjects would include object representations and self-other relations, but this study did not use measures that would provide that data. The items represent specific blends of the underlying dimensions of affiliation and autonomy, which can be conceived, in a broader frame of reference, as an interpersonal formulation (Benjamin 2005) within the two-polarities

model of relatedness and self-definition (Luyten and Blatt 2013). Introject[1] affiliation captures the degree of self-love versus self-hate. In interpersonal theory, it denotes "a hypothesized personality structure . . . , which comprises a relatively stable conscious and unconscious repertoire of ways of treating the self" (Henry, Schacht, and Strupp 1990, p. 769). By introjection the child comes to treat him- or herself as it has once been treated by significant others (Henry, Schacht, and Strupp 1990). Just for illustration, introject self-love is represented by the item "I tenderly, lovingly cherish myself," and self-hate is represented by the item "I punish myself by blaming myself and putting myself down." Participants rated each item on a scale from 0 (completely false) to 100 (completely true) in 10-point increments. We decided to focus on "worst" times because this procedure addresses social desirability response problems (Benjamin 2000) and is salient for depressive patients. From the various methods available for assessing the dimensions of SASB, we applied the vector index approach (Pincus et al. 1998) because of its satisfactory convergent validity and distribution characteristics. Benjamin, Rothweiler, and Critchfield (2006) reported satisfactory psychometric qualities for the INTREX, short form; it is sensitive to and captures changes in psychodynamic (Gumz, Bauer, and Brähler 2012) and cognitive-behavioral therapies (Bedics et al. 2012).

Treatments and Therapists

The twenty-one therapists in the study were all experienced in their field; no candidates were included. Mean duration of psychotherapeutic practice was 15 years (range: 6–29); mean age was 47 years (range: 38–56). Fourteen therapists delivered PA and PD and seven therapists CBT only. There was no significant difference in years of training, level of expertise, or amount of experience between therapists working in the three treatment modalities. All therapists graduated from their training institutes and were then licensed to apply PA and PD or CBT according to the German Psychotherapy Guidelines (Rüger, Dahm, and Kallinke 2005). PA and PD therapists graduated from the same institute in both modalities, CBT therapists from a CBT institute. To qualify for insurance reimbursement of treatment, each therapist had to apply to a PA/PD or CBT expert from the German Psychotherapy Guidelines to ensure that

[1]Note that the term *introject* is not used here in its psychoanalytic meaning (e.g., as defined by Schafer [1968]).

the therapy was performed according to established rules. During the course of treatment, the external expert controlled therapeutic technique again. To enhance external validity, treatments were not manualized.

PA[2] is defined as a "predominantly verbal, interpretative, insight-oriented approach which aims to modify or re-structure maladaptive relationship representations" that lie at the root of psychological disturbance (Fonagy and Kächele 2009, p. 1339). The usual duration of psychoanalytic therapy, according to the German Psychotherapy Guidelines, is 240 sessions; session frequency is between two and three sessions a week with the patient lying on the couch.

PD is based on the same principles of theory and technique, but is more limited, both in the depth of the therapeutic process and in its goals; its focus is on symptom-sustaining here-and-now conflicts. The usual duration of psychodynamic therapy, according to the guidelines, is 80 sessions. A single weekly session is carried out in a face-to-face setting.

CBT comprises therapeutic modalities developed on the basis of a psychology of learning and social psychology and requires analyses of the causal and maintaining factors of depression. It combines cognitive and behavioral techniques in varying degrees. Average duration, according to the guidelines, is between 45 and 60 sessions; session frequency is one session a week.

In this study, mean duration of PA was 39 months (range 3–91) or 234 sessions (range 17–370), of PD 34 months (range 3–108) or 88 sessions (range 12–313), and of CBT 26 months (range 2–78) or 45 sessions (range 7–100). Low values are due to the intent-to-treat approach.

Treatment fidelity was assessed with expert-rated measures. Independent raters assessed treatment fidelity using the Psychotherapy Process Q-set (PQS; Jones 2000). The 100 items of the PQS capture key treatment parameters, including patient behavior, therapist behavior, and patient-therapist interactions. To assess the degree to which treatments adhered to standard psychoanalytic or cognitive-behavioral practice we used the PQS prototypes for psychoanalytic therapy and CBT (Ablon and Jones 2005). Assessment was based on the analysis of three audiotaped middle sessions from 77 percent of all treatments in the intent-to-treat sample (Zimmermann et al. 2015). Data from blind raters confirmed that

[2]We follow a widespread tradition in psychoanalytic theory to call high-dose psychodynamic psychotherapy psychoanalytic therapy (PA) and low-dose psychodynamic psychotherapy psychodynamic therapy (PD).

therapists in the PA treatments adhered more closely to principles of psychoanalytic therapy than did therapists in the other two treatment conditions, and that therapists in the CBT treatments adhered more closely to the principles of that modality than did therapists in the other two treatment conditions (for details, see Zimmermann et al. 2015). In sum, treatment fidelity seemed adequate.

Statistical Analyses

To answer our two main questions we relied on a multilevel modeling framework (MLM; Raudenbush and Bryk 2002).[3] The first question, that regarding outcome trajectories, was approached by predicting outcome measures (BDI, GSI, IIP) from time variables in the long data-format (i.e., each row represented an assessment of a specific patient at a specific point in time). In particular, we operationalized time using three separate variables: (a) number of years from the beginning of treatment until the end of the first half year (range 0–0.5), (b) number of years from the first half year until termination (range 0 to individually varying maxima), and (c) number of years after termination until three-year follow-up (range 0–3). Jointly predicting outcome from these three within-person (level 1) variables represents a "three-stage" piecewise linear growth model: the intercept indicates the expected outcome value prior to treatment, and the three slopes indicate expected annual rates of change during (a) the first six months of treatment, (b) the remaining time of treatment, and (c) the three years after termination (Raudenbush and Bryk 2002).[4]

At the between-person level (level 2), we started with fitting unrestricted models that allowed for random variation and covariation in intercepts and slopes across patients. However, as these models were thought to provide a basis for the more complex mediation models to be discussed below, we tried early on to reduce the number of parameters by deleting random coefficients (i.e., variance and covariance parameters) with p-values > .3. Finally, we included two dummy variables representing the three treatment groups as level 2 predictors, using PA as the

[3]MLM provides several advantages over traditional methods, including its ability to treat time as a continuous predictor and to allow for missing data across time. Moreover, as compared to methods relying on structural equation modeling, MLM is more flexible in handling variably spaced measurement occasions and individually varying assessment periods (Singer and Willett 2003).

[4]This piecewise approach seemed more appropriate than including quadratic or cubic terms of time, in that it better captures the distinct phases of the assessment period, allows an intuitive interpretation of slope parameters, and provides a useful framework for testing mediation.

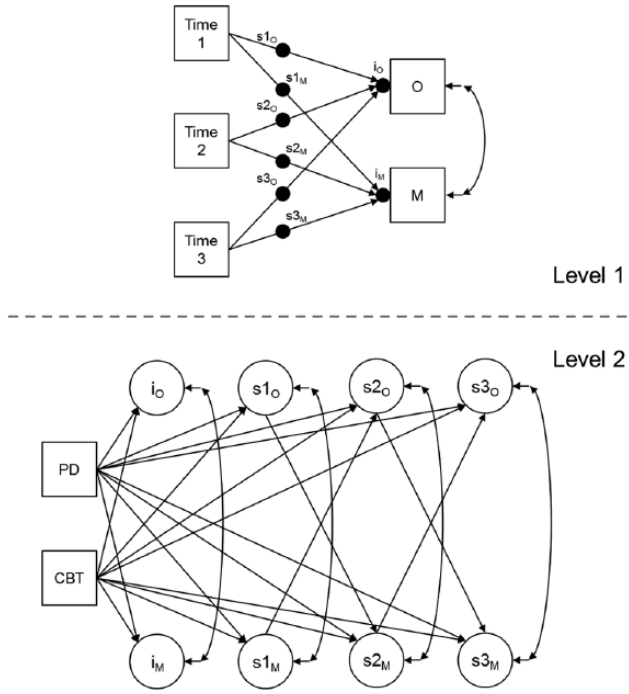
reference group (= 0). Note that we repeated all analyses using CBT as the reference group to additionally provide estimates of the differential effects of PD vs. CBT. Models were fit separately for each of the three outcome measures, based on Mplus 7.0 (Muthén and Muthén 2012) using maximum likelihood estimation with robust standard errors.

In sum, the analyses presented so far can be conceived of as a reanalysis and extension of the results published in Huber et al. (2012, 2013). Specifically, going beyond Huber et al., analyses (a) were based on a larger number of measurement points within therapy, (b) explicitly distinguished between early change and later change within therapy, and (c) reframed effect sizes along a common timeline. A consequence of the last point is that significant differences between treatment groups in time parameters will not be confounded by differences in treatment duration. This is because they refer to differences in the annual rate of change, and not to differences in the total effect of treatments.

The second question, that regarding mediation, was approached by extending the multilevel growth models of outcome. In particular, we estimated models with two parallel change processes, jointly capturing changes in outcome and in mediator, and explicitly testing for paths between sequential slopes of mediator and outcome at level 2 (Preacher 2015; Selig and Preacher 2009). In a preparatory step, we repeated the multilevel analyses presented above, this time using mediators (i.e., introject affiliation and patient- or therapist-rated alliance) as dependent variables. In the case of alliance, we used a standard "one-stage" linear growth model with a single slope representing the annual rate of change during treatment (except for the first six months). We did so because alliance was not assessed before treatment began or after termination.

In the main analyses we estimated joined parallel process models for each combination of outcome and mediator variable. Figure 1 presents the basic structure of this model. We focused on two sets of model parameters. First, the covariance parameters of the residual random variation in slopes that refer to the same time frame (e.g., $s1_O$ and $s1_M$) indicate whether changes in outcome and mediator run in parallel. That is, they provide insight into whether patients who improve during a specific phase in terms of outcome improve also in terms of the mediator, and vice versa. Second, the regression paths predicting slopes of outcome from slopes of the mediator that refer to a previous time frame (e.g., $s2_O$ from $s1_M$) indicate whether changes in outcome are preceded by changes in the

Figure 1. Three-stage parallel process growth model with sequential mediation



At Level 1, O represents the outcome measure, M the mediator, Time 1 the number of years from the beginning of treatment to the first half year, Time 2 the number of years from the first half year to termination, and Time 3 the number of years from termination to three-year follow-up. At Level 2, PD and CBT represent dummy-coded treatment variables with PA acting as the reference group, i_O and $s1_O$ to $s3_O$ random variation in intercept and slopes of outcome, and i_M and $s1_M$ to $s3_M$ random variation in intercept and slopes of the mediator. Note that estimated models may include covariances between random variables at Level 2 that are not depicted in this figure.

mediator. This would support the assumption that the mediator is causally involved in producing changes in the outcome.

Besides these informative parameters, our model also provides a basis for testing the mediation hypotheses. Technically, mediation analysis requires assessing the effect of the independent variable on the mediator (commonly denoted as “path a”), and the effect of the mediator on the outcome controlling for the independent variable (commonly denoted as

"path b"). After that, the significance of the mediated, or "indirect," effect can be tested by inspecting the product of the two path coefficients (MacKinnon 2008). In our model, the effects of the dummy-coded treatment variables PD and CBT on the first two slopes of the mediator represent path a, and the effects of the first two slopes of the mediator on the last two slopes of the outcome represent path b (see Preacher 2015; Selig and Preacher 2009). That is, we tested whether treatment effects on later changes in outcome were mediated by changes in the mediator during the first six months of treatment, and whether treatment effects on changes after treatment were mediated by changes in the mediator during the remaining time of treatment.

RESULTS

Trajectories of Outcome

Table 1 presents the parameter estimates of the final linear growth models for depressive symptoms (BDI), general psychiatric symptoms (GSI), and interpersonal problems (IIP). Depressive symptoms decreased dramatically during the first six months of treatment, and continued to improve during the remaining time of treatment, though to a much lesser degree than in the early treatment phase. As treatment effects on the first two slopes were not significant, this pattern of decrease can be expected for participants from all treatment groups.

The following example should clarify the exact amount of symptom changes: Depressive symptoms of the average patient in the PA group decreased from 25.4 BDI points at the beginning of treatment by roughly 1.9 BDI points per month during the first six months of treatment and by 0.17 BDI points per month during the remaining time of treatment. Taken together, these estimates suggest that after three years of treatment the average patient showed a total decrease of roughly 16 BDI points, which clearly is a clinically significant change (Ogles, Lambert, and Masters 1996) and roughly corresponds to a within-group effect size of $d = 2.1$ (i.e., when standardizing this estimate by the pre-treatment standard deviation of the BDI).

However, the third slope, representing the annual rate of change during the follow-up period, differed significantly between PA and PD, and between PA and CBT. Whereas patients who received PA showed a continuing decrease in depressive symptoms even after termination,

Table 1. Parameter estimates of linear growth models for three outcome measures

	BDI		GSI		IIP	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
Intercepts and Effects						
i (Intercept)	25.406***		1.266***		1.792***	
	(1.232)		(0.087)		(0.071)	
i ← PD (vs. PA)	−0.210	−.017	−0.095	−.043	0.064	.080
	(1.964)		(0.128)		(0.091)	
i ← CBT (vs. PA)	−1.192	−.100	−0.136	−.064	−0.122	−.160
	(1.868)		(0.131)		(0.109)	
[i ← CBT (vs. PD)]	−1.014	−.085	−0.040	−.019	−0.187#	−.246
	(2.073)		(0.135)		(0.100)	
s1 (Intercept)	−22.656***		−0.703***		−0.269*	
	(1.776)		(0.133)		(0.118)	
s1 ← PD (vs. PA)	−6.792#	−.352	−0.277	−.335	−0.587**	−.420
	(3.658)		(0.186)		(0.180)	
s1 ← CBT (vs. PA)	−2.893	−.157	−0.017	−.022	0.174	.130
	(3.703)		(0.227)		(0.208)	
[s1 ← CBT (vs. PD)]	3.983	.216	0.260	.329	0.761**	.570
	(4.603)		(0.224)		(0.221)	
s2 (Intercept)	−1.998***		−0.124***		−0.149***	
	(0.513)		(0.028)		(0.026)	
s2 ← PD (vs. PA)	0.690	.228	0.052	.263	0.090**	.526
	(0.542)		(0.037)		(0.031)	
s2 ← CBT (vs. PA)	1.077	.372	0.048	.254	0.072*	.440
	(0.690)		(0.036)		(0.033)	
[s2 ← CBT (vs. PD)]	0.380	.131	−0.004	−.021	−0.018	−.110
	(0.639)		(0.043)		(0.031)	
s3 (Intercept)	−1.198**		−0.083**		−0.068**	
	(0.349)		(0.024)		(0.020)	
s3 ← PD (vs. PA)	1.310*	.290	0.085*	.385	0.064#	.305
	(0.578)		(0.037)		(0.037)	
s3 ← CBT (vs. PA)	2.154**	.499	0.105**	.497	0.068*	.339
	(0.804)		(0.037)		(0.030)	
[s3 ← CBT (vs. PD)]	0.841	.195	0.021	.099	0.004	.020
	(0.869)		(0.041)		(0.038)	
Variances and Covariances						
Var(i)	31.591***		0.173***		0.133***	
	(6.805)		(0.025)		(0.028)	
Var(s1)	68.849**		0.126		0.339**	
	(22.793)		(0.085)		(0.117)	
Var(s2)	1.729		0.007		0.004	
	(1.440)		(0.005)		(0.003)	

(continued)

Table 1 (continued)

	BDI		GSI		IIP	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
Var(s3)	3.027*		0.007#		0.007#	
	(1.480)		(0.004)		(0.004)	
Var(e)	30.531***		0.105***		0.064***	
	(4.086)		(0.017)		(0.006)	
i ↔ s2	−6.647*	−.857	−0.023*	−.611		
	(2.908)		(0.009)			
s1 ↔ s3					−0.018	−.300
					(0.012)	

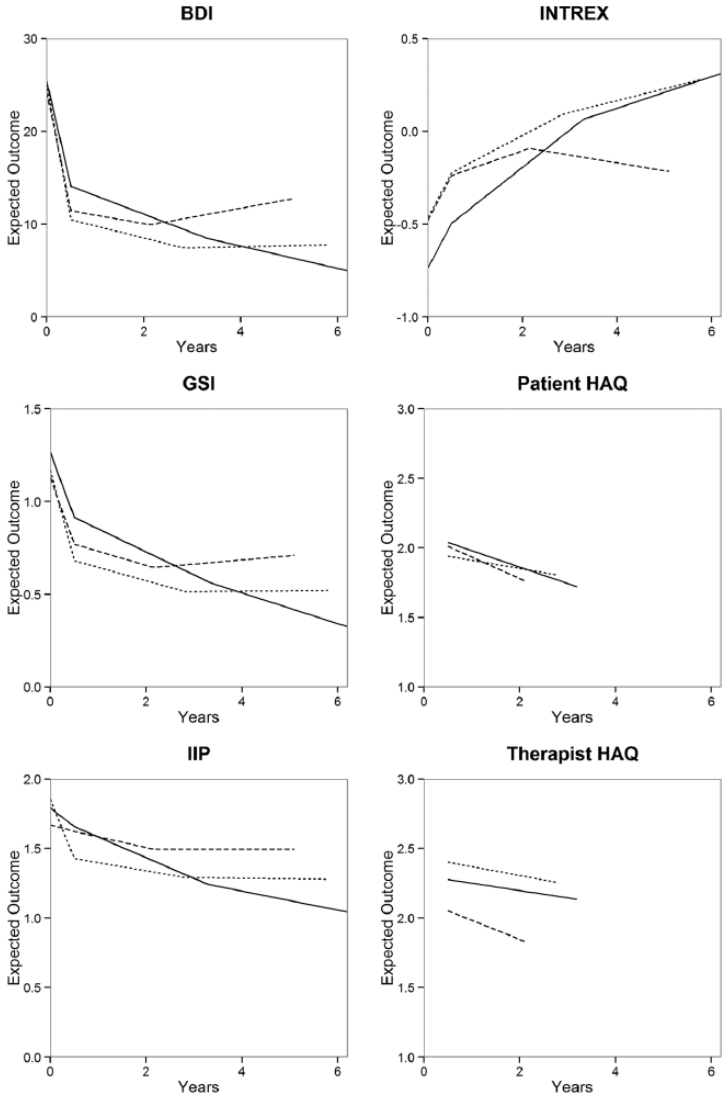
Note. $N = 100$. Number of measurement points per model was 886 for BDI, 895 for GSI, and 891 for IIP. BDI = Depressive symptoms assessed by Beck Depression Inventory. GSI = General psychiatric symptoms assessed by the Symptom Checklist 90 Revised. IIP = Interpersonal problems assessed by Inventory of Interpersonal Problems. PA = Psychoanalytic Therapy. PD = Psychodynamic Therapy. CBT = Cognitive-behavioral Therapy. i = Estimated value at pretreatment. s1 = Estimated annual rate of change during the first six months of treatment. s2 = Estimated annual rate of change during the remaining time of treatment. s3 = Estimated annual rate of change during the three-year follow-up period. Note that intercepts of random parameters refer to the estimated value of participants receiving PA. Effects of CBT vs. PD are denoted in brackets because they were estimated in separate models. *B* and *SE* refer to unstandardized effects or (co-)variances and robust standard errors. β/*r* refers to effects or correlations that were standardized using estimated variances in models without level-2 predictors. All models were fitted with maximum likelihood estimation.
#$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

participants who received PD or CBT evinced a less favorable pattern of change.

The expected difference between PA and CBT patients ran on average to more than 6 BDI points during the three years of follow-up (roughly translating into a between-group effect size of $d = 0.8$). This differential treatment effect is apparent in the top left panel in Figure 2, showing the estimated trajectories of depressive symptoms as a function of treatment group.

A very similar picture emerged when considering general psychiatric symptoms as the outcome. Irrespective of treatment group, symptoms rapidly decreased during the first six months of treatment, and continued to decrease, though more slowly, during the remaining months. However, after termination, we again found significant differences between PA and PD, and between PA and CBT, in the annual rate of change: patients in the PA group continued to show improvement, whereas those in the PD and CBT groups

Figure 2. Linear growth trajectories of outcome measures (left side) and mediators (right side) as a function of treatment group



Outcome measures include depressive symptoms (BDI), general psychiatric symptoms (GSI), and interpersonal problems (IIP); mediators include introject affiliation (INTREX), patient-rated alliance (HAQ), and therapist-rated alliance (HAQ). Psychoanalytic therapy is represented by solid lines, Psychodynamic therapy by dotted lines, and Cognitive-behavioral therapy by dashed lines.

did not (see Figure 2, middle left panel). A somewhat different pattern of results was found for the trajectories of interpersonal problems. They decreased considerably faster during the first six months of treatment in patients who received PD than in patients from the other two groups. However, in the later phase of therapy, the annual rate of improvement was highest in patients receiving PA. This advantage of PA was still noticeable after termination, at least in comparison to CBT (Figure 2, bottom left panel).

Trajectories of Mediators

Table 2 presents the parameter estimates of the final linear growth models for the putative mediators: introject affiliation (INTREX), patient-rated alliance (HAQ), and therapist-rated alliance (HAQ). For introject affiliation, we had to fix the random variation in the first slope to zero during the model building process, suggesting that patients did not differ in their individual rates of change during the first six months of treatment. The pattern of treatment effects was quite similar to the results regarding depressive symptoms and general psychiatric symptoms: participants in all treatment groups uniformly developed more affiliative introjects during the first and second treatment phases, but PA participants continued to improve after termination, whereas CBT participants did not (Figure 2, upper right panel; note that higher INTREX values represent more affiliative introjects). Patient-rated alliance improved over the course of treatment irrespective of treatment group (Figure 2, middle right panel; note that lower HAQ values represent better alliances). In contrast, although therapist-rated alliance did not change at the group level during treatment, therapists in the CBT group rated their alliance consistently more positive than did therapists in the PD group (Figure 2, bottom right panel).

Parallel and Sequential Associations between Changes in Outcome and Mediators

Tables 3, 4, and 5 present parameter estimates of the joined parallel process models for each combination of outcome and mediator variables. Regarding depressive symptoms, we found some indication for parallel changes in introject affiliation during the second phase of treatment (the coefficient for the covariance of $s2_O$ and $s2_M$ just missed statistical significance, $p = .05$). This suggests that, irrespective of treatment group, patients who improved in terms of depressive symptoms also improved in terms of introject affiliation, and vice versa. In contrast, sequential effects

Table 2. Parameter estimates of linear growth models for three putative mediators

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
Intercepts and Effects						
i (Intercept)	−0.736***		2.039***		2.277***	
	(0.111)		(0.094)		(0.095)	
i ← PD (vs. PA)	0.272#	.217	−0.098	−.069	0.125	.127
	(0.142)		(0.151)		(0.134)	
i ← CBT (vs. PA)	0.257	.214	−0.027	−.020	−0.224#	−.238
	(0.157)		(0.161)		(0.117)	
[i ← CBT (vs. PD)]	−0.015	−.012	0.070	.052	−0.349**	−.370
	(0.146)		(0.177)		(0.116)	
s1 (Intercept)	0.482***					
	(0.100)					
s1 ← PD (vs. PA)						
s1 ← CBT (vs. PA)						
[s1 ← CBT (vs. PD)]						
s2 (Intercept)	0.201***		−0.118*		−0.053	
	(0.039)		(0.056)		(0.048)	
s2 ← PD (vs. PA)	−0.067	−.178	0.059	.118	−0.011	−.034
	(0.062)		(0.072)		(0.067)	
s2 ← CBT (vs. PA)	−0.112#	−.311	−0.037	−.078	−0.085	−.271
	(0.067)		(0.112)		(0.090)	
[s2 ← CBT (vs. PD)]	−0.045	−.125	−0.096	−.201	−0.073	−.233
	(0.076)		(0.108)		(0.078)	
s3 (Intercept)	0.085*					
	(0.035)					
s3 ← PD (vs. PA)	−0.021	−.075				
	(0.053)					
s3 ← CBT (vs. PA)	−0.127*	−.475				
	(0.051)					
[s3 ← CBT (vs. PD)]	−0.105#	−.393				
	(0.055)					
Variances and Covariances						
Var(i)	0.311***		0.342***		0.174***	
	(0.055)		(0.061)		(0.048)	
Var(s1)	0					
Var(s2)	0.026***		0.055*		0.021	
	(0.007)		(0.027)		(0.017)	
Var(s3)	0.013*					
	(0.005)					
Var(e)	0.151***		0.113***		0.104***	
	(0.012)		(0.018)		(0.016)	

(continued)

Table 2 (continued)

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
i ↔ s2			−0.069# (0.036)	−.475		
i ↔ s3	−0.026* (0.012)	−.269				

Note. Sample size / number of measurement points per model was 100/867 for INTREX, 90/495 for Patient HAQ, and 88/425 for Therapist HAQ. INTREX = Introject affiliation assessed by INTREX questionnaire. Patient HAQ = Alliance assessed by patient version of the Helping Alliance Questionnaire. Therapist HAQ = Alliance assessed by therapist version of the Helping Alliance Questionnaire. PA = Psychoanalytic Therapy. PD = Psychodynamic Therapy. CBT = Cognitive-behavioral Therapy. i = Estimated value at pre-treatment (for INTREX) or at six month after beginning of treatment (for HAQ). s1 = Estimated annual rate of change during the first six months of treatment. s2 = Estimated annual rate of change during the remaining time of treatment. s3 = Estimated annual rate of change during the three-year follow-up period. Note that intercepts of random parameters refer to the estimated value of participants receiving PA. Effects of CBT vs. PD are denoted in brackets because they were estimated in separate models. *B* and *SE* refer to unstandardized effects or (co-)variances and robust standard errors. β/*r* refers to effects or correlations that were standardized using estimated variances in models without level-2 predictors. All models were fitted with maximum likelihood estimation.
#$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

of changes in mediators on later changes in depressive symptoms were not significant.[5]

For general psychiatric symptoms and interpersonal problems, a similar pattern of results emerged. Specifically, the association between changes in outcome and in introject affiliation during the second phase of treatment again just missed statistical significance ($p = .06$ and $p = .05$), suggesting that the development of affiliative introjects may be intertwined with general improvement across different outcome measures. Again, sequential effects of changes in mediators on later changes in

[5]Note that our model also included regression paths testing the reverse direction (i.e., whether changes in the mediator are preceded by changes in outcome). Indeed, we observed significant sequential effects of changes in depressive symptoms during the first six months of treatment on later changes in introject affiliation and patient-rated alliance (Figure 3, upper and middle panels). This means that patients who showed early responses in terms of depressive symptoms developed more affiliative introjects and better alliances later on. However, we refrain from interpreting these effects (and similar effects for general psychiatric symptoms and interpersonal problems) because we had no a priori hypotheses in this regard.

Table 3. Parameter estimates of parallel process growth models for depressive symptoms

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
Treatment Effects on Outcome						
$i_O \leftarrow$ PD (vs. PA)	−0.516 (1.976)	−.041	−0.212 (1.965)	−.017	−0.210 (1.964)	−.017
$i_O \leftarrow$ CBT (vs. PA)	−1.215 (1.916)	−.102	−1.284 (1.889)	−.107	−1.200 (1.871)	−.100
[$i_O \leftarrow$ CBT (vs. PD)]	−0.699 (2.127)	−.058	−1.071 (2.095)	−.090	−0.990 (2.075)	−.083
$s1_O \leftarrow$ PD (vs. PA)	−5.616 (3.648)	−.291	−6.750# (3.674)	−.350	−6.606# (3.643)	−.343
$s1_O \leftarrow$ CBT (vs. PA)	−3.063 (3.696)	−.166	−2.451 (3.800)	−.133	−2.733 (3.741)	−.148
[$s1_O \leftarrow$ CBT (vs. PD)]	2.552 (4.613)	.139	4.294 (4.697)	.233	3.873 (4.616)	.210
$s2_O \leftarrow$ PD (vs. PA)	0.306 (0.555)	.101	0.714 (0.545)	.236	0.555 (0.593)	.183
$s2_O \leftarrow$ CBT (vs. PA)	1.222# (0.679)	.422	0.972 (0.767)	.336	0.942 (0.705)	.326
[$s2_O \leftarrow$ CBT (vs. PD)]	0.917 (0.670)	.317	0.262 (0.699)	.091	0.387 (0.669)	.134
$s3_O \leftarrow$ PD (vs. PA)	1.627** (0.593)	.361	1.398* (0.611)	.310	1.344* (0.595)	.298
$s3_O \leftarrow$ CBT (vs. PA)	2.495** (0.850)	.579	2.007* (0.785)	.465	2.081* (0.968)	.483
[$s3_O \leftarrow$ CBT (vs. PD)]	0.869 (0.884)	.202	0.611 (0.930)	.142	0.738 (0.993)	.171
Treatment Effects on Mediator						
$i_M \leftarrow$ PD (vs. PA)	0.278# (0.143)	.221	−0.109 (0.151)	−.077	0.120 (0.133)	.122
$i_M \leftarrow$ CBT (vs. PA)	0.258# (0.156)	.215	−0.007 (0.164)	−.005	−0.225# (0.118)	−.239
[$i_M \leftarrow$ CBT (vs. PD)]	−0.020 (0.146)	−.017	0.102 (0.180)	.075	−0.345** (0.116)	−.366
$s2_M \leftarrow$ PD (vs. PA)	−0.116# (0.070)	−.308	0.144 (0.096)	.289	−0.004 (0.079)	−.012
$s2_M \leftarrow$ CBT (vs. PA)	−0.136* (0.067)	−.378	−0.023 (0.115)	−.048	−0.077 (0.091)	−.246
[$s2_M \leftarrow$ CBT (vs. PD)]	−0.019 (0.082)	−.053	−0.168 (0.117)	−.352	−0.073 (0.088)	−.233
$s3_M \leftarrow$ PD (vs. PA)	−0.014 (0.052)	−.050				

(continued)

Table 3 (continued)

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
$s3_M \leftarrow$ CBT (vs. PA)	−0.125* (0.058)	−.468				
[$s3_M \leftarrow$ CBT (vs. PD)]	−0.111# (0.060)	−.416				
Sequential Effects of Slopes						
$s2_M \leftarrow s1_O$	−0.010* (0.004)	−.512	0.016* (0.008)	.618	0.000 (0.009)	.000
$s3_M \leftarrow s2_O$	−0.001 (0.030)	−.011				
$s3_O \leftarrow s2_M$	4.099 (3.076)	.342	−2.508 (2.679)	−.277	−1.739 (6.417)	−.126
Covariances						
$i_O \leftrightarrow i_M$	−1.542* (0.678)	−.479	0.114 (0.386)	.031	0.258 (0.252)	.102
$s2_O \leftrightarrow s2_M$	−0.141# (0.072)	−.604	−0.020 (0.101)	−.065	0.096 (0.101)	.472
$s3_O \leftrightarrow s3_M$	−0.058 (0.056)	−.225				
$i_O \leftrightarrow s2_O$	−3.375 (2.281)	−.435	−6.274* (3.136)	−.809	−6.360* (2.724)	−.820
$i_M \leftrightarrow s2_M$			−0.069 (0.042)	−.475		
$i_M \leftrightarrow s3_M$	−0.030** (0.011)	−.417				

Note. $N = 100$. Number of measurement points per model was 902 for INTREX, 892 for Patient HAQ, and 891 for Therapist HAQ as mediator (M). In all three models, depressive symptoms (assessed by Beck Depression Inventory) were used as outcome (O). PA = Psychoanalytic Therapy. PD = Psychodynamic Therapy. CBT = Cognitive-behavioral Therapy. i = Estimated value at pre-treatment (for INTREX and O) or at six months after beginning of treatment (for HAQ). s1 = Estimated annual rate of change during the first six months of treatment. s2 = Estimated annual rate of change during the remaining time of treatment. s3 = Estimated annual rate of change during the three-year follow-up period. Effects of CBT vs. PD are denoted in brackets because they were estimated in separate models. *B* and *SE* refer to unstandardized effects or covariances and robust standard errors. β/*r* refers to effects or correlations that were standardized using estimated variances in models without level 2 predictors. Note that coefficients for intercepts and variances are omitted in this table. All models were fitted with maximum likelihood estimation.

# $p < .10$. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 4. Parameter estimates of parallel process growth models for general psychiatric symptoms

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
Treatment Effects on Outcome						
$i_O \leftarrow$ PD (vs. PA)	−0.113	−.122	−0.095	−.102	−0.095	−.102
	(0.128)		(0.128)		(0.128)	
$i_O \leftarrow$ CBT (vs. PA)	−0.139	−.156	−0.139	−.156	−0.136	−.153
	(0.132)		(0.131)		(0.131)	
[$i_O \leftarrow$ CBT (vs. PD)]	−0.026	−.029	−0.044	−.050	−0.041	−.046
	(0.137)		(0.136)		(0.135)	
$s1_O \leftarrow$ PD (vs. PA)	−0.195	−.236	−0.276	−.334	−0.261	−.316
	(0.173)		(0.187)		(0.185)	
$s1_O \leftarrow$ CBT (vs. PA)	−0.016	−.020	0.001	.001	−0.004	−.005
	(0.221)		(0.228)		(0.227)	
[$s1_O \leftarrow$ CBT (vs. PD)]	0.179	.227	0.277	.351	0.258	.327
	(0.208)		(0.226)		(0.224)	
$s2_O \leftarrow$ PD (vs. PA)	0.024	.121	0.053	.268	0.042	.213
	(0.041)		(0.038)		(0.042)	
$s2_O \leftarrow$ CBT (vs. PA)	0.053	.281	0.045	.238	0.042	.222
	(0.035)		(0.037)		(0.037)	
[$s2_O \leftarrow$ CBT (vs. PD)]	0.028	.148	−0.008	−.042	0.000	.000
	(0.042)		(0.043)		(0.045)	
$s3_O \leftarrow$ PD (vs. PA)	0.092*	.416	0.089*	.403	0.088*	.398
	(0.041)		(0.040)		(0.037)	
$s3_O \leftarrow$ CBT (vs. PA)	0.103*	.488	0.101**	.478	0.109**	.516
	(0.041)		(0.035)		(0.040)	
[$s3_O \leftarrow$ CBT (vs. PD)]	0.011	.052	0.012	.057	0.021	.099
	(0.041)		(0.046)		(0.045)	
Treatment Effects on Mediator						
$i_M \leftarrow$ PD (vs. PA)	0.280*	.223	−0.105	−.074	0.117	.119
	(0.141)		(0.152)		(0.135)	
$i_M \leftarrow$ CBT (vs. PA)	0.261#	.217	−0.011	−.008	−0.228#	−.242
	(0.154)		(0.162)		(0.117)	
[$i_M \leftarrow$ CBT (vs. PD)]	−0.020	−.017	0.095	.070	−0.344**	−.365
	(0.143)		(0.178)		(0.116)	
$s2_M \leftarrow$ PD (vs. PA)	−0.098	−.261	0.174	.349	−0.004	−.012
	(0.069)		(0.126)		(0.086)	
$s2_M \leftarrow$ CBT (vs. PA)	−0.119#	−.331	−0.051	−.107	−0.066	−.211
	(0.067)		(0.113)		(0.085)	
[$s2_M \leftarrow$ CBT (vs. PD)]	−0.021	−.058	−0.225#	−.472	−0.062	−.198
	(0.081)		(0.122)		(0.093)	

(continued)

Table 4 (continued)

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
$s3_M$ ← PD (vs. PA)	−0.014 (0.053)	−.050				
$s3_M$ ← CBT (vs. PA)	−0.130* (0.056)	−.487				
[$s3_M$ ← CBT (vs. PD)]	−0.116* (0.057)	−.434				
Sequential Effects of Slopes						
$s2_M$ ← $s1_O$	−0.169 (0.108)	−.371	0.488* (0.204)	.809	0.049 (0.228)	.124
$s3_M$ ← $s2_O$	0.124 (0.397)	.088				
$s3_O$ ← $s2_M$	0.000 (0.147)	.000	−0.091 (0.120)	−.206	−0.006 (0.199)	−.009
Covariances						
i_O ↔ i_M	−0.103*** (0.029)	−.431	0.005 (0.026)	.018	0.038* (0.018)	.202
$s2_O$ ↔ $s2_M$	−0.010# (0.006)	−.657	−0.001 (0.006)	−.050	0.005 (0.006)	.377
$s3_O$ ↔ $s3_M$	−0.002 (0.004)	−.158				
i_O ↔ $s2_O$	−0.018* (0.007)	−.478	−0.022* (0.009)	−.585	−0.025** (0.009)	−.664
i_M ↔ $s2_M$			−0.071* (0.034)	−.489		
i_M ↔ $s3_M$	−0.033** (0.011)	−.459				

Note. $N = 100$. Number of measurement points per model was 903 for INTREX, 900 for Patient HAQ, and 900 for Therapist HAQ as mediator (M). In all three models, general psychiatric symptoms (assessed by the Symptom Checklist 90 Revised) were used as outcome (O). PA = Psychoanalytic Therapy. PD = Psychodynamic Therapy. CBT = Cognitive-behavioral Therapy. i = Estimated value at pre-treatment (for INTREX and O) or at six months after beginning of treatment (for HAQ). s1 = Estimated annual rate of change during the first six months of treatment. s2 = Estimated annual rate of change during the remaining time of treatment. s3 = Estimated annual rate of change during the three-year follow-up period. Effects of CBT vs. PD are denoted in brackets because they were estimated in separate models. *B* and *SE* refer to unstandardized effects or covariances and robust standard errors. β/*r* refers to effects or correlations that were standardized using estimated variances in models without level 2 predictors. Note that coefficients for intercepts and variances are omitted in this table. All models were fitted with maximum likelihood estimation.
#$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

Table 5. Parameter estimates of parallel process growth models for interpersonal problems

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
Treatment Effects on Outcome						
i_O ← PD (vs. PA)	0.050 (0.091)	.063	0.064 (0.091)	.080	0.064 (0.091)	.080
i_O ← CBT (vs. PA)	−0.126 (0.109)	−.165	−0.128 (0.109)	−.168	−0.123 (0.109)	−.162
[i_O ← CBT (vs. PD)]	−0.176# (0.101)	−.231	−0.192# (0.101)	−.252	−0.187# (0.100)	−.246
$s1_O$ ← PD (vs. PA)	−0.523** (0.176)	−.374	−0.587** (0.180)	−.420	−0.584** (0.181)	−.418
$s1_O$ ← CBT (vs. PA)	0.189 (0.198)	.142	0.191 (0.212)	.143	0.175 (0.208)	.131
[$s1_O$ ← CBT (vs. PD)]	0.712** (0.209)	.533	0.778** (0.224)	.583	0.759** (0.221)	.568
$s2_O$ ← PD (vs. PA)	0.068* (0.034)	.397	0.090** (0.031)	.526	0.087** (0.033)	.508
$s2_O$ ← CBT (vs. PA)	0.071* (0.035)	.434	0.069* (0.034)	.422	0.070* (0.034)	.428
[$s2_O$ ← CBT (vs. PD)]	0.003 (0.037)	.018	−0.020 (0.033)	−.122	−0.017 (0.032)	−.104
$s3_O$ ← PD (vs. PA)	0.073# (0.037)	.348	0.060 (0.037)	.286	0.065# (0.037)	.310
$s3_O$ ← CBT (vs. PA)	0.075* (0.032)	.374	0.071* (0.033)	.354	0.067# (0.038)	.334
[$s3_O$ ← CBT (vs. PD)]	0.002 (0.038)	.010	0.011 (0.041)	.055	0.002 (0.046)	.010
Treatment Effects on Mediator						
i_M ← PD (vs. PA)	0.287* (0.142)	.229	−0.093 (0.149)	−.065	0.124 (0.133)	.126
i_M ← CBT (vs. PA)	0.260# (0.154)	.217	−0.034 (0.158)	−.025	−0.229# (0.117)	−.243
[i_M ← CBT (vs. PD)]	−0.027 (0.144)	−.022	0.059 (0.174)	.043	−0.352** (0.114)	−.374
$s2_M$ ← PD (vs. PA)	−0.133* (0.067)	−.354	0.167* (0.080)	.335	0.045 (0.062)	.137
$s2_M$ ← CBT (vs. PA)	−0.077 (0.069)	−.214	−0.087 (0.090)	−.182	−0.101 (0.095)	−.322
[$s2_M$ ← CBT (vs. PD)]	0.056 (0.089)	.156	−0.254** (0.096)	−.533	−0.146 (0.096)	−.466
$s3_M$ ← PD (vs. PA)	−0.012 (0.083)	−.043				

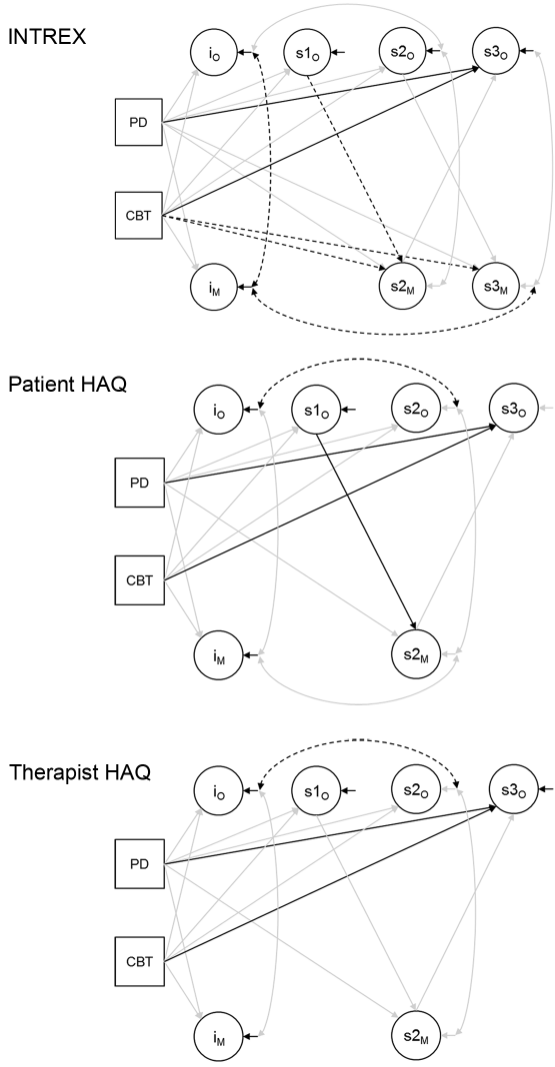
(continued)

Table 5 (continued)

	INTREX		Patient HAQ		Therapist HAQ	
	B (*SE*)	β/*r*	*B* (*SE*)	β/*r*	*B* (*SE*)	β/*r*
$s3_M \leftarrow$ CBT (vs. PA)	−0.113 (0.090)	−.423				
[$s3_M \leftarrow$ CBT (vs. PD)]	−0.101# (0.059)	−.378				
Sequential Effects of Slopes						
$s2_M \leftarrow s1_O$	−0.119* (0.049)	−.442	0.182** (0.064)	.510	0.091 (0.077)	.388
$s3_M \leftarrow s2_O$	−0.202 (0.920)	−.124				
$s3_O \leftarrow s2_M$	0.075 (0.233)	.135	0.052 (0.123)	.124	−0.025 (0.215)	−.039
Covariances						
$i_O \leftrightarrow i_M$	−0.125*** (0.033)	−.610	0.039 (0.025)	.168	0.005 (0.018)	.031
$s2_O \leftrightarrow s2_M$	−0.009# (0.005)	−.682	0.001 (0.003)	.057	0.003 (0.005)	.261
$s3_O \leftrightarrow s3_M$	−0.004 (0.003)	−.333				
$s1_O \leftrightarrow s3_O$	−0.013 (0.015)	−.217	−0.022 (0.017)	−.367	−0.018 (0.013)	−.300
$i_M \leftrightarrow s2_M$			−0.063* (0.029)	−.434		
$i_M \leftrightarrow s3_M$	−0.023* (0.011)	−.320				

Note. $N = 100$. Number of measurement points per model was 899 for INTREX, 894 for Patient HAQ, and 895 for Therapist HAQ as mediator (M). In all three models, interpersonal problems (assessed by Inventory of Interpersonal Problems) were used as outcome (O). PA = Psychoanalytic Therapy. PD = Psychodynamic Therapy. CBT = Cognitive-behavioral Therapy. i = Estimated value at pre-treatment (for INTREX and O) or at six months after beginning of treatment (for HAQ). s1 = Estimated annual rate of change during the first six months of treatment. s2 = Estimated annual rate of change during the remaining time of treatment. s3 = Estimated annual rate of change during the three-year follow-up period. Effects of CBT vs. PD are denoted in brackets because they were estimated in separate models. *B* and *SE* refer to unstandardized effects or covariances and robust standard errors. β/*r* refers to effects or correlations that were standardized using estimated variances in models without level 2 predictors. Note that coefficients for intercepts and variances are omitted in this table. All models were fitted with maximum likelihood estimation.
#$p < .10$. *$p < .05$. **$p < .01$. ***$p < .001$.

Figure 3



Results of three parallel process growth models with depressive symptoms (BDI) as outcome and (a) introject affiliation (INTREX), (b) patient-rated alliance (HAQ), and (c) therapist-rated alliance (HAQ) as a mediator, respectively. PD and CBT represent dummy-coded treatment variables with PA acting as the reference group, i_O, $s1_O$, $s2_O$ and $s3_O$ random variation in intercept and slopes of outcome, and i_M, $s2_M$ and $s3_M$ random variation in intercept and slopes of the mediator. Solid arrows indicate significantly positive coefficients ($p < .05$), dashed arrows indicate significantly negative coefficients, and gray arrows indicate nonsignificant coefficients.

outcomes were not significant. This means that our results do not support the hypothesis that introject affiliation and alliance are causal mediators of treatment effects, because path b in mediation analyses (i.e., the effects of $s2_M$ on $s3_O$) was generally not significant (the same was true for the products of paths a and b).

DISCUSSION

Outcome Trajectories

This study investigated outcome trajectories during the early and later treatment phases, and during the follow-up phase, across PA, PD, and CBT. It extends the findings of a three-year follow-up study reported by Huber et al. (2012) and a process-outcome pilot study by Klug et al. (2012).

Trajectories of symptom outcome (BDI and GSI) decreased dramatically in the early phase and less so in the later phase and did not differ significantly between treatment groups. This absence of a significant difference indicates that in these treatment phases total dose (= number of sessions) may presumably not be a salient factor for alleviating symptom load, because symptomatic gains may be enhanced by the therapeutic alliance (Castonguay et al. 2006) and other common factors that work across treatments, and that explain nearly 50 percent of outcome variance in adult depression (Cuijpers et al. 2012). Moreover, therapists of various orientations tend to concentrate on well-being and symptoms initially (Howard et al. 1993, 1996), though some psychoanalysts do not view symptom relief or even improved well-being a central initial goal. Further, our data showed that most patients are responsive in the early treatment phase, as reflected in the pronounced decline of depressive and general psychiatric symptoms. The same pattern emerged in the ongoing treatment phase, during which all three treatments continued to improve depressive symptoms and general psychiatric symptoms, though at a much slower rate, thus confirming the "negatively accelerated curve" of a dose-response model originally developed by Howard et al. (1986) and later confirmed by Anderson and Lambert (2001).

After treatment, however, depressive and general psychiatric symptoms continued to decrease in PA, whereas they did not in PD and CBT. This finding may reflect a change in the underpinning intrapsychic structures enhanced by the more intense treatment process of PA, but we cannot with the present study design disentangle the therapeutic factors

that may lead to stable symptomatic relief. In another study based on this sample, we found empirical evidence that psychoanalytic technique (especially discussing the patient's dreams or fantasies, and memories and reconstructions from infancy and childhood) mediates the long-term stability of symptomatic improvement (Zimmermann et al. 2015). Psychoanalytic theory suggests that when, after termination of a successful psychoanalysis, the therapist is no longer present in reality, patients make use of the ability to analyze their problems as a product of identification with the analyst's analyzing function, a construct first outlined by Freud (1937), elaborated by Horney (1942) and Hoffer (1950), and empirically supported by Falkenström et al. (2007) and Sandell et al. (2000). But there are other helpful internalizations as well—self-calming, self-soothing, self-supporting ones, for instance, or internalized positive experiences ("positive repetitions") with the analyst (Pfeffer 1980).

In contrast to symptomatic outcome measures (BDI and GSI), trajectories of interpersonal problems (IIP) decreased significantly faster during the early treatment phase in PD than in PA or CBT. This may be due to the fact that PD techniques have an early and pronounced focus on interpersonal problems. In the later phase of therapy and after termination, this process comes to a standstill in PD and CBT, while a substantial reduction of interpersonal problems was still noticeable in PA, marking a a significant difference between PA and CBT. One explanation for this finding could be that CBT does not offer a total dose large enough to allow substantial gains in interpersonal problems, while in PA after six months in treatment, possibly due to higher session frequency (Reese, Toland, and Hopkins 2011), specific technique factors can take effect and patients can enter into a salient phase leading to sustained benefits. However, this remains rather speculative; more fine-grained process-outcome research will be necessary to further elucidate the mechanisms of change in interpersonal problems.

Our results do not fit with the "good-enough level" model that assumes "that patients who receive low doses of treatment are those who change rapidly, whereas patients who receive high doses of treatment are those who change slowly" (Baldwin et al. 2009, p. 204). If this were true, one would expect that slopes of trajectories would be steeper in the shorter or less intense treatments (PD and CBT). Comparisons with results from previous studies on outcome trajectories of long-term psychotherapies are complicated by differently defined measurement schedules, different

statistical analyses, and differently operationalized treatments (Knekt et al. 2008; Leichsenring et al. 2005; Berghout et al. 2012; Blomberg, Lazar, and Sandell 2001; Puschner et al. 2007; Brockmann, Schlüter, and Eckert 2006). However, our results are generally in line with their pattern of results, in that outcome trajectories (symptoms and interpersonal problems) from all three treatment groups decline significantly during ongoing treatment, that trajectories of symptoms did not differ significantly between PA and PD during ongoing treatment, and that trajectories of interpersonal problems tended to differ significantly between PA and CBT during ongoing treatment and the follow-up phase.

Mediation Analyses

The results of the mediation analyses provided no support for the hypothesis that introject affiliation is a causal link between any of the treatments and long-term outcome.[6]

Our findings could not confirm the mediating function of the introject, thus contradicting several process-outcome studies (see, e.g., Henry, Schacht, and Strupp 1990; Quintana and Meara 1990) and a more recently published study applying a four-months' time-lagged model in dialectical behavior therapy (DBT) of borderline patients (Bedics et al. 2012).

Besides the possibility that introject affiliation is not a mediator of change, our divergent results may reflect certain methodological limitations of our study design. In particular, the small sample size and modest number of measurement points limit the power of the study to detect relevant effects, thereby inflating Type II error (i.e., failure to reject a false null hypothesis when it should have been). For example, we were not able to test mediating effects of early changes in introject affiliation because individual differences in the first slope (i.e., Var[s1]) did not differ significantly from zero. This was probably due to the fact that we had access to only one or two measurement points per person during the first six months of treatment, making the estimation of change processes unreliable. Further, self-rated measurement of the introject cannot, by definition, capture the unconscious features of the introject, features that according to psychoanalytic theory are salient in the development and persistence of symptoms and maladaptive behaviors (see, e.g., Schafer 1968).

[6]Included in the discussion of mediators were only studies with treatment as the independent variable, as Johansson and Høglend (2007) proposed, and formal mediational analysis was performed, thus going beyond, for example, a predictor study (Kazdin 2007).

Our data, as well as the data of a pilot study (Klug et al. 2012), offer no support for the hypothesis that therapeutic alliance mediates differential effectiveness in PA, PD, and CBT. Although therapeutic alliance is a critical therapeutic element, especially in the treatment of depressive disorders (Castonguay et al. 2006), its function as a putative mediator is discussed controversially in the literature. Kazdin (2007) has questioned the empirical evidence for the alliance as a mediator, and Wilson et al.'s results (1999, 2002) show no evidence that therapeutic alliance is a mediator, nor do those of Puschner et al. (2007). Barber, Khalsa, and Sharpless (2010), summarizing the literature, state that studies of therapeutic alliance are interesting and evocative, but that more research is clearly required. The findings of Falkenström, Granstöm, and Holmqvist (2007) and of Tasca and Lampard (2012) suggest that therapeutic alliance is part of a circular causal chain in which it predicts subsequent symptom change and symptom change predicts subsequent alliance change. A more recent study, by Zilcha-Mano et al. (2014), lends some support to the hypothesis that therapeutic alliance is a curative factor, although the specific underlying mechanisms could not be elucidated since influences of unmeasured third variables could not be ruled out. To sum up, empirical evidence has accumulated that therapeutic alliance can be conceived as a vehicle for transmitting the active elements of therapy (= mediators) (Hartley and Strupp 1983), in the sense that it creates a "working space" (Horvath et al. 2011) in which to understand the patient's symptoms and problems from a new perspective, but it may not be conceived as occurring in a causal chain, as would a mediator, which must change in order for change to occur in the target variables (Johansson and Høglend 2007; Kraemer et al. 2001, 2002).

Limitations

Our study has several limitations, summarized in Roose (2014); in the first place the small sample sizes, incomplete simultaneous randomized allocation, and lack of a Structured Clinical Interview for DSM-IV (SCID-I and SCID-II) assessment of primary and comorbid diagnoses. Another limitation is the lack of a low-intensity treatment group like "treatment as usual" (TAU) to control for the natural course of the disorder; that is, depressive episodes in some patients are self-limited and possibly remit within six to eight months (Berger and van Calker 2004; Wittchen 1988). On the other hand, patients were followed up three years

after termination and would have relapsed by then had the treatments not changed the natural course of the disorder.

Regarding internal validity, the differing dose of the treatments can be considered confounding, but duration of treatment was controlled. We argue that each treatment has a different underlying working model that needs a specified time frame with a stipulated number of sessions and specific interventions in order to initiate a specific process. Moreover, we deliberately wanted to investigate treatment packages with their prototypical doses to inform practitioners about their everyday practice, thus helping to bridge the gap between research and practice. Another limitation and threat to internal validity (though a strength in terms of external validity) was the lack of treatment manuals and the absence of a manual-guided adherence measure. However, treatment fidelity was assessed by an observer-rated measure for PA, PD, and CBT (Zimmermann et al. 2015).

Among the strengths of the study is the use of instruments with good psychometric properties applied at multiple time points for the assessment of introject, therapeutic relationship, and outcome, but it is clearly a limitation that long-term follow-ups were from the patient's perspective only, whereas a multimodal perspective throughout is strongly recommended (Hill and Lambert 2004). The HAQ yields only a global assessment of the therapeutic relationship, emphasizing collaboration and helpfulness with an in-the-moment and companionable quality, thus neglecting other important aspects of alliance (Hatcher et al. 1995). Another critical issue is the ecological validity of our change measures. For example, Kazdin (2006) has contended that patients who have changed in clinically significant ways in terms of a standard outcome measure "may not have improved in any way that affected their lives or the lives of those with whom they are in contact" (p. 46). Unless outcome data are linked to specific referents in everyday life, measures are of unknown ecological validity and have "arbitrary metrics" (Blanton and Jaccard 2006). Eagle and Wolitzky (2011) have addressed this very issue in their critical discussion of outcome research of psychoanalytic and psychodynamic psychotherapy. Finally, the assessment schedule of this study (e.g., variably spaced measurement occasions during therapy, different durations of therapy, etc.) posed some challenges for the statistical models. We tried to handle this by defining three phases of change a priori and estimating piecewise linear growth models. However, the exact definition of these phases (e.g., separating the

first six months of treatment from the remaining time in treatment) is arbitrary, and the assumption of a linear change process within each phase might not be valid. Future studies should address these issues by employing an even larger number of measurements per person.

In sum, our findings provide provisional support that patterns of change differ between PA, PD, and CBT, especially after termination. Further, our data confirm the hypothesis that patients do not just "take their time" in attaining their therapeutic goals, but that patients profit by extended treatment duration as a driving force to reach more significant therapeutic goals. According to psychoanalytic theory, further improvement after termination can be explained by the patient's identification with the analyst's analysing function or by other helpful internalizations, such as self-calming, self-soothing, self-supporting capacities or internalized positive experiences ("positive repetitions") with the analyst (Pfeffer 1980). However, neither introject affiliation nor therapeutic alliance was shown to be a differential mediator of treatment and long-term outcome. We attribute this finding to methodological shortcomings of our study design, especially to the application of self-report measures that cannot probe therapeutic processes to grasp the unconscious mechanisms of change. Thus, more fine-grained, concept-based, and expert-rated measures to grasp microprocesses at the session level are needed to approach the more subtle change processes in long-term psychotherapy.

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