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Disentangling within- and between-patient effects of defensive functioning on psychotherapy outcome using mixed models

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Disclosure of interest

All authors declare that they have no conflict of interest.

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Abstract

There is a great need to identify predictors of treatment response, and the analysis of defense mechanisms is a promising approach. Defensive functioning may influence psychotherapy outcome in two ways: First, when it is generally higher or lower for some patients relative to others and second, as it shifts in individual patients over time. The present study examined both within- and between patient effects of defenses using hierarchical linear modeling. Forty-seven patients diagnosed with depression, anxiety, or adjustment disorders received 25±3 sessions of integrative cognitive-behavioral therapy in a university outpatient clinic. The Defense Mechanism Rating Scale (DMRS) was used to assess defenses in the 1st, 8th, 16th, and 24th session and relate them to symptom severity of depression and anxiety. A higher number of adaptive defense mechanisms was associated with less severe depressive symptoms during treatment while a higher number of immature defenses was related to more severe depressive and anxiety symptoms. An increase in adaptive and a decrease in immature defenses over the course of treatment predicted symptom reduction of depression whereas a decrease in neurotic and immature defenses was associated with reductions in anxiety symptoms. Our results empirically support defensive functioning as a mechanism of change in psychotherapy.

Introduction

There is a great need to understand the mechanisms of change at work in psychotherapy (Crits-Christoph, Gibbons, & Mukherjee, 2013) and the analysis of defense mechanisms is a promising approach. Defenses are defined as "automatic psychological processes which protect the individual from anxiety and unnecessary awareness of internal and external dangers and stressors" (APA, 2000). A three-level hierarchical classification of defenses was introduced: adaptive, neurotic, and maladaptive (Vaillant, 1971). Adaptive defenses typically maximize awareness of internal states and result in both, positive outcome and the most effective psychological protection, whereas maladaptive defenses act to restrict or alter awareness of internal states and conflicts, thus limiting positive outcome (Kneepkens & Oakley, 1996). Overall defensive functioning (ODF) represents a summary score and reflects the average level of adaptation.

Defensive functioning may influence psychotherapy outcome in at least two ways: First, when generally higher or lower for some patients relative to others (between-patient differences) and second, it may also promote improvement as it shifts in individual patients over time (within-patient changes). So far, research has focused on between-patient effects of defensive functioning on treatment outcome using defenses as measured at intake to predict outcome at the symptomatic level: lower (more maladaptive) scores in defensive functioning were significantly related to the presence and severity of depressive (e.g., DeFife & Hilsenroth, 2005; Hoglend & Perry, 1998) and anxiety symptoms (Heldt, Manfro, & Kipper, 2003). However, such analyses do not necessarily mean that improving defensive functioning with a given patient will improve therapy outcome (within-patient effects; Falkenström, Finkel, Sandell, Rubel, & Holmqvist, 2017).

So far, only a limited number of studies addressed defenses using repeated measures to observe changes in defense style and its predictive value for therapy outcome (e.g. Hill et al., 2015; Johansen, Krebs, Svartberg, Stiles, & Holen, 2011; Kramer, deRoten, Perry &

Despland, 2012; Kramer, Despland, Michel, Drapeau, & deRoten, 2010; Perry & Bond, 2012). Overall defensive functioning (ODF) has been shown to improve from pre to post therapy in patients with depression and anxiety disorders (e.g., Drapeau, deRoten, Perry, & Despland, 2003; Kramer et al., 2012). In studies with primarily depressed patients, adaptive defenses increased, and maladaptive defenses decreased while neurotic defenses did not change meaningfully (Akkermann, Lewin, & Carr, 1999; Bond & Perry, 2004; Kneepkens & Oakley, 1996; Perry & Bond, 2009). In studies with mostly anxious patients, both neurotic (Kipper et al., 2005) and maladaptive defenses (Heldt et al., 2007; Hersoug, Bøgwald, & Høglend, 2005) decreased over the course of treatment with no significant change in the adaptive defense category. Change in ODF has repeatedly been presented as a significant predictor of therapy outcome at the symptomatic level (e.g., Bond & Perry, 2004; Heldt et al., 2007; Kramer, de Roten, Perry, & Despland, 2013).

Taken together, findings suggest that both within- and between patient effects of defensive functioning predict psychotherapy outcome. However, to date they were never analyzed within the same model, thereby controlling for the effect of the respective other. Further, since previous studies assessed defenses only twice, usually at baseline and treatment termination, longitudinal analyses were methodologically not feasible (Singer & Willet, 2003). Thus, these studies limited their analyses to the estimation of the fixed effects defenses exhibited on therapy outcome, assuming that effects were homogenous among patients while not accounting for the nested structure of the data (i.e., repeated measures nested within patients; Gómez Penedo, Muiños, Hirsch, & Roussos, 2019). Also, it could not be determined if defense change preceded symptom change or the other way around.

Building on and extending previous research, the present study aims to analyze the effects of defenses on outcome using longitudinal models (i.e., addressing the nestedness of the data) and to disaggregate within- and between-patient effects of this predictor. To our

knowledge, this is the first study to apply such methods to defenses, making it a useful addition to previous research in the area. The following four hypotheses were tested:

1. ODF, adaptive defenses, and maladaptive defenses during treatment will display significant between-patient effects on depressive symptoms. More specifically, a higher frequency of adaptive defenses and a lower frequency of maladaptive defenses will be associated with lower severity of depressive symptoms.

2. ODF, neurotic defenses, and maladaptive defenses during treatment will exhibit significant between-patient effects on anxiety symptoms. A lower frequency of neurotic and maladaptive defenses will be associated with a lower severity of anxiety symptoms.

3. Change in ODF and in the frequency of adaptive and maladaptive defenses will show significant within-patient effects on change of depressive symptoms. An increase in adaptive defenses and a decrease in maladaptive defenses will be associated with a reduction in depressive symptoms.

4. Change in ODF and in the frequency of neurotic and maladaptive defenses will result in significant within-patient effects on change of anxiety symptoms. A decrease in neurotic and maladaptive defenses will be associated with a reduction in anxiety symptoms.

Methods

Participants

The sample consisted of 47 consecutively recruited patients from a randomized controlled trial who all received 25±3 sessions of integrative cognitive-behavioral therapy in a university outpatient clinic. Of 47 patients, 29 (62%) were female with a mean age of 32.09 years (*SD* = 10.5). Twenty-two patients (44%) met diagnostic criteria for a principal diagnosis of unipolar depression (ICD; F32), 17 patients (34%) for an anxiety disorder (ICD; F40, F41) and eight (16%) for adjustment disorder (ICD; F43.2). Previous research failed to identify variables that independently differentiated adjustment disorder from depressive episodes (Casey et al., 2006), which is why patients diagnosed with adjustment disorder were included

in our study. Research diagnoses were established by trained staff with the Structured Clinical Interview for DSM-IV-R (First, Spitzer, Gibbon, & Williams, 2004). Exclusion criteria included substance dependence within the last six months, current risk of suicide, immediate risk of self-harm or harm to others, and the presence of a likely organic cause for the mental disorder. Subjects simultaneously receiving other psychological treatments were also excluded. Treatment was provided by 18 (69.23%) female and eight (30.76%) male psychotherapists with a mean age of 34.49 years (*SD* = 8.63). The trial was approved by the Cantonal Ethics Committee (KEK BE 168/15), and all patients gave written informed consent for their therapy sessions to be video-recorded and the data being used in the context of the trial.

Study design

The design included the assessment of defensive functioning (ODF, adaptive, neurotic, and immature defenses) as well as the severity of depression and anxiety symptoms as both, within and between subject factors (see Babl et al., 2016 for the study design of the RCT).

Material

Defense Mechanism Rating Scale. The *Defense Mechanism Rating Scale* (DMRS; 5th edition; Perry, 1990), is an observer-rated manual for the identification of 30 individual defense mechanisms in session transcripts of psychotherapy. The manual comprises a definition of each defense mechanism, a description of the intra-psychic function and a list of similar mechanisms and indications of how to distinguish them. The 30 defense mechanisms are arranged hierarchically, divided into seven levels. The higher the level on which a defense mechanism is located, the greater the score assigned to it. With adaptive defense mechanisms positive and negative aspects of reality are seen together with minimal distortion, minimal avoidance of stressors and maximal gratification of wishes. Adaptive defenses receive seven points, since they belong to level seven. Neurotic defenses, as located on levels five and six,

lead to mild distortion of reality, some avoidance of stressors, some gratification and some symptom formation. Maladaptive defenses comprise levels one to four. Here, positive and negative aspects of reality are kept separate with a clear distortion, stressors are simplified to either internal or external and small stressors receive big meaning. All defense mechanisms are evaluated with a score corresponding to their level. Based on the scoring, the overall defensive functioning and defense category scores can be calculated.

Six Master-level psychology students underwent six months of intensive rater training in the DMRS. Over the course of the rater training, nine session transcripts with 21 to 30 pages each (M = 26) were coded, and eleven consensus meetings of two to six hours each were held, amounting to around 34 hours. Raters then transcribed and rated a total of 192 therapy sessions in a secured and designated rating room at the University, between August 2017 and August 2018. For each patient, the 1st, 8th, 16th and 24th sessions were transcribed and rated for defense mechanisms to reflect the course of the treatment. In case of technical malfunctioning of the video, such as audio failure, a neighboring session number was being transcribed and rated instead. Twenty-two sessions were substituted. Session length slightly varied (Mean = 62.1 min, SD = 7.69 min). Reliability coefficients were established on 20% (n = 36 sessions) of the ratings. The intraclass correlation coefficients ranged from ICC(2,1) = .46 to .86 (Mean = .72). This indicated acceptable to good agreement (Shrout, 1998).

Beck Depression Inventory. The revised version of the *Beck Depression Inventory* (BDI-II; Hautzinger, Keller, & Kühner, 2006) is a self-assessment tool consisting of 21 items to determine depressive symptoms during the past two weeks. The BDI-II is not only an indicator of the severity of depressive symptoms in accordance with DSM-IV but also one of the most widely used self-report measures for depression in clinical practice and research (Kühner, Bürger, Keller, & Hautzinger, 2007). It has shown robust psychometric properties (Hautzinger et al., 2006).

Beck Anxiety Inventory. The *Beck Anxiety Inventory* (BAI; Margraf, Beck, & Ehlers, 2007) is a self-report questionnaire to detect the severity of anxiety symptoms. The BAI consists of 21 descriptive statements with regard to somatic and cognitive symptoms of anxiety during the last seven days. The BAI can be cited as a reliable and valid questionnaire (Margraf et al., 2007).

Procedure

In this study, patients were treated with an integrative form of CBT. The duration of the treatment was predefined by the RCT and based on the usual length of cognitive-behavioral therapies in the given outpatient setting, i.e., 25 ± 3 sessions of 50 minutes each. All therapy sessions were video recorded for quality assurance, research and supervision purposes. Four sessions per therapy (1, 8, 16 and 24) were transcribed and subsequently rated for defense mechanisms. Symptom severity as measured with the BDI-II and BAI was assessed at the same four measurement points.

Analytic strategy

All data analyses were done using the international scientific software HLM 7 (Raudenbush, Brok, & Congdon, 2011). For data analysis, we used hierarchical linear models (HLM; Raudenbush & Bryk, 2002; see Appenix A for equations of the HLM and detailed description). These models address the dependency of the data presented in longitudinal studies, due to the repeated measures (here, assessment of all measurements in therapy sessions 1, 8, 16, and 24) nested within patients. Besides providing a more robust estimation of change than classical parametric statistics, HLM has other strengths like handling missing data mimicking an intent-to-treat approach by including all participants into the analysis who completed outcome measures at least once.

We first ran two-level fully unconditional models with BAI and BDI as outcome variables. Based on these two unconditional models we calculated intraclass correlation coefficients to establish the variance explained by patient effects (level 2). In the BAI

unconditional model this was ICC=0.41, in the BDI unconditional model ICC=0.52, meaning that 41% of the variance of the BAI and 52% of the variance of the BDI is explained by patient effects, indicating that HLM is necessary. Then, two conditional models (one for each outcome variable) were calculated including ODF as a predictor and disaggregating its within- and between-patient effects (Falkenström et al., 2017). In these models, ODF was included as a level 1 predictor patient-mean centered, capturing the within-patient effect of ODF on the outcome variables. At the same time, the patient's average level of ODF during treatment (grand-mean centered) was included as a level 2 predictor of the intercept (estimated value of the outcome variable on a session where the patients have an average ODF), capturing the between-patient effect. The level of ODF during treatment was also included (grand-mean centered) as a level 2 predictor of the ODF within-patient effect, to test if there was variability based on the patients' ODF-level during therapy.

In a second set of analysis, we ran two-level models including the frequency of (i) adaptive, (ii) neurotic, and (iii) immature defenses as predictors of the outcome variables. The frequencies of the three defense categories were included in the same model person-mean centered. This model allows disaggregating the within-patient effects of each category, controlling for the within-patient effects of the other ones. Furthermore, the frequency of the three variables (grand-mean centered) was included as level 2 predictors of the intercept, disentangling the between-patient effect of each type of defense when controlling for the effect of the other ones. Finally, the frequency of the three types of defenses was included (again, grand-mean centered) as predictors of the within-patient effects of each of the three defense categories.

For the significant effects we calculated effect sizes by computing *pseudo* R^2. To compute this indicator of the size of the effects we subtracted the variance explained by the conditional models (i.e., the one including the targeted predictor) from the variance explained by the unconditional model (i.e., the one not including the predictor) and divided it by the

variance explained by the unconditional model. Thus, *pseudo* R^2 is to be interpreted as the percentage of outcome variance explained by the predictor.

Figures of the BDI and BAI session effects (Appendix B) were produced using R (R Core Team, 2017) and the packages lme4 (Bates, Meachler, Bolker, & Walker, 2015) and ggeffects (Lüdecke, 2018).

Results

ODF as a predictor of treatment outcome

The results of the HLM analyses with ODF as predictor of treatment outcome showed that there was a significant between-patient effect of ODF on symptom severity of depression as measured with the BDI, fixed effect(γ_{01}) = -7.51, *SE* = 1.62, CI_{95} [-10.69, -4.33], $t(44) = -4.63$, $p < .001$, *pseudo* $R^2 = .38$. Patients with one ODF score unit above the mean across treatment have 7.51 units *less* in the BDI during treatment. Furthermore, the within-patient effect of ODF on BDI approached significance, fixed effect(γ_{10}) = -2.43, *SE* = 1.23, CI_{95} [-4.84, -0.02], $t(44) = -1.98$, $p = .054$. Again, a one-unit variance in patient's ODF from the individual mean was associated with a 2.43 points lower score in the BDI at that session. With regard to random effects on level 2, patients significantly varied in their BDI scores when having their average level of ODF during treatment ($VAR_{u0} = 34.31$, $\chi^2(42)=173$, $p < .001$) and depending on ODF fluctuations during treatment ($VAR_{u1} = 13.59$, $\chi^2(42)=62$, $p = .03$). When analyzing the effects of ODF on symptom severity of anxiety as assessed with the BAI, we also found a significant between-patient effect, fixed effect(γ_{01}) = -5.50, *SE* = 1.53, CI_{95} [-8.50, -2.50], $t(44) = -3.59$, $p < .001$, *pseudo* $R^2 = .33$. Patients with an ODF-level one-unit above the mean during treatment tend to have 5.50 units *less* in the BAI during treatment. However, the within-patient effect of ODF on BAI was not significant, fixed effect(γ_{10}) = -1.66, *SE* = 1.10, CI_{95} [-3.82, 0.50], $t(44) = -1.51$, $p = .14$. Random effects on level 2 showed that patients varied significantly in their BAI scores when having their average level of ODF during treatment ($VAR_{u0} = 26.04$, $\chi^2(42)=112$, $p < .001$) but not significantly depending on

ODF fluctuations during treatment (VAR_{u1} = 0.09, $\chi^2(42)=55$, p = *.09*). For overall effects of sessions on symptomatology (BDI, BAI) see Appendix B.

Insert Table 1 here

Adaptive, neurotic and immature defenses as a predictor of therapy outcome

The calculated models estimated the effects of the different defense categories and presented a significant between-patient effect of adaptive defenses on symptom severity of depression, fixed effect(γ_{01}) = -0.49, *SE* = 1.16, CI_{95} [-2.76, 1.78], $t(42)$ = -3.04, p = .004. *pseudo* R^2= .21. A one-unit *greater* frequency of adaptive defenses across treatment, was associated with 0.49 points *less* on the BDI during therapy. However, there was no significant between-patient effect of adaptive defenses on severity of anxiety, fixed effect(γ_{01})= -0.02, *SE* = 0.14, CI_{95} [-0.29, 0.25], $t(42)$ = -0.12, p = .90. Furthermore, when controlling for the effect of the other defense categories, a significant within-patient effect of adaptive defenses on depression severity was shown, fixed effect(γ_{10}) = -0.35, *SE* = 0.16, CI_{95} [-0.66, -0.04], $t(42)$ = -2.27, p = .03, *pseudo* R^2= .19, but not on anxiety severity, fixed effect(γ_{10}) = -0.21, *SE* = 0.18, CI_{95} [-0.56, 0.14], $t(42)$ = -1.19, p = .24. A one-unit *increase* in the frequency of adaptive defenses was associated with 0.35 units *reduction* of depression severity.

The frequency of neurotic defenses over the course of treatment was neither significantly associated with depression severity, fixed effect(γ_{02}) = -0.10, *SE* = 0.26, CI_{95} [-0.61, 0.41], $t(42)$ = -0.40, p = .70, nor anxiety severity, fixed effect(γ_{02}) = 0.24, *SE* = 0.23, CI_{95} [-0.21, 0.69], $t(42)$ = 1.03, p = .31, during treatment. Also, there was no significant within-patient effect of neurotic defenses on depression severity, fixed effect(γ_{20}) = 0.24, *SE* = 0.14, CI_{95} [-0.03, 0.51], $t(42)$ = 1.68, p = .10. However, the within-patient effect of neurotic defense on anxiety severity was significant, fixed effect(γ_{20}) = 0.37, *SE* = 0.15, CI_{95} [0.08, 0.66], $t(42)$ = 2.522, p = .02, *pseudo* R^2= .08. A one-unit *increase* in neurotic defense frequency was associated with a 0.37 *increase* in anxiety severity.

The immature defenses exhibited significant between-patient effects on both depression severity, fixed effect(γ_{03}) = 0.40, *SE* = 0.17, CI_{95} [0.07, 0.73], *t*(42) = 2.42, *p* = .02, *pseudo* R^2= .14, and anxiety severity, fixed effect(γ_{03}) = 0.52, *SE* = 0.15, CI_{95} [0.23, 0.81], *t*(42) = 3.53, *p* = .001, *pseudo* R^2= .26. A one-unit *greater* frequency of immature defenses across treatment, was related to 0.40 units *greater* depressive severity and a 0.52 units *greater* anxiety severity during therapy. Additionally, there was a significant within-patient effect of the immature defenses on depressive severity, fixed effect(γ_{30}) = 0.34, *SE* = 0.17, CI_{95} [0.01, 0.67], *t*(42) = 2.01, *p* = . 05, *pseudo* R^2= .24, and anxiety severity, fixed effect(γ_{30}) = 0.38, *SE* = 0.15, CI_{95} [0.09, 0.67], *t*(42) = 2.50, *p* = . 02, *pseudo* R^2= .23. A one-unit *increase* of the frequency of immature defenses was associated with a 0.34 units *increase* of depression severity and a 0.38 units *increase* of anxiety severity.

Here, random effects on level 2 represent the variability around the estimated value of BDI (VAR_{u0} = 42.50, χ^2(26)=187, *p* < .001) and BAI (VAR_{u0} = 32.26, χ^2(26)=149, *p* < .001) when patients have their average adaptive, neurotic, and immature defenses. Both were significant. Further, patients significantly varied in their BAI scores depending on the fluctuations in adaptive (VAR_{u1} = 0.39, χ^2(26)=66, *p* < .001), neurotic (VAR_{u2} = 0.02, χ^2(26)=49, *p* < .01), and maladaptive (VAR_{u3} = 0.10, χ^2(26)=54, *p* < .001) defenses. Not so in their BDI values (VAR_{u1} = 0.13, χ^2(26)=37, *p = .07;* VAR_{u2} = 0.02, χ^2(26)=27, *p = .4;* VAR_{u3} = 0.20, χ^2(26)=28, *p = .34*). Further, see Appendix C for random effects graphed for randomly chosen individual patients.

Insert Table 2 here

Discussion

The present study aimed to analyze the effects of defensive functioning on psychotherapy outcome during acute treatment using longitudinal models and to disaggregate the within- and between-patient effects of this predictor.

In line with our first hypothesis and with previous research (e.g., DeFife & Hilsenroth, 2005), ODF exhibited a large between-patient effect on symptoms of depression, explaining 38% of variance in the estimated value for an average patient. As expected, a higher number of adaptive defense mechanisms was associated with less severe depressive symptoms during treatment while a higher number of immature defenses was related to more severe depressive symptoms. The second hypothesis was largely, but not completely upheld. ODF and maladaptive defenses displayed a significant between-patient effect on anxiety symptoms, with ODF explaining 33% of outcome variance and maladaptive defenses explaining 26%, while neurotic defenses did not. This pattern was also found by Euler et al. (2018) and Perry (2001) who reported a significant change on the level of maladaptive defenses first. One possible explanation as put forward by Vaillant (1993) is that defenses change up the hierarchy in a stepwise fashion with maladaptive defenses moving up to neurotic and eventually adaptive defenses. On the contrary, other studies did not confirm such stepwise changes (Albucher, Abelson, & Nesse, 1998; Akkermann et al., 1999; Babl et al., 2019). Our third hypothesis was upheld: the within-patient effect of ODF, adaptive and maladaptive defenses on symptom severity of depression reached the significance level, indicating that an increase in adaptive defenses and a decrease in maladaptive defenses over the course of psychotherapy is accompanied by an improvement in depressive symptoms. Together with the evidence presented by former trials (e.g. Heldt et al., 2007; Kramer et al., 2012), it can be concluded with some confidence that not only differences in defensive functioning between patients but also differences within patients over time are potent predictors of therapy outcome at the symptomatic level. The fourth hypothesis addressed within-patient effects of defenses on anxiety symptoms and was partially upheld. The within-patient effect of ODF on the severity of anxiety symptoms was non-significant. This may be due to the different nature of anxiety and depressive disorders. Previously, depression has been referred to as a state, while anxiety has been discussed as a trait (Bond & Perry, 2004; Perry, 2001). In general,

traits are more enduring, while states change with respect to internal and external stimuli (Bond & Perry, 2004). Our findings support this notion. However, when looking more specifically at defense categories, the within-patient effect of neurotic and immature defenses on anxiety severity was significant, explaining 8% of variance in the BAI score in the case of the former, and 23% in the case of the latter.

In line with previous research, our study confirmed defensive functioning not only as a significant between- but also within-patient predictor of therapy outcome. One might, therefore wonder how such improvements of defensive functioning in individual patients over time can be fostered. Preliminary evidence suggests that the process of addressing defenses is related to improvements in ODF (Olsen, Perry, Janzen, Petraglia, & Presniak, 2011), neurotic (Winston, Samstag, Winston, & Muran, 1994), maladaptive defenses (Hersoug et al., 2005), and overall therapy outcome (Perry, Petraglia, Olson, Presniak, & Metzger, 2012). Psychoanalytic and dynamic therapy specifically addresses defenses in-session aiming to develop more adaptive defensive functioning. However, even therapeutic approaches not aiming at changing defensive functioning, as the integrative interventions applied in this study, seem to exhibit a favorable effect on defenses (Babl et al., 2019). This indicates that despite their psychodynamic roots, defenses can be applied as trans-theoretical constructs, suggesting that knowledge about a patient's predominant defense mechanisms could be helpful to therapists of all orientations. By this, knowledge about individual defenses may contribute to a better understanding of the patients' psychological functioning and thus increase therapists' responsiveness. Working with defenses may generally play an important role in treatment, which warrants further study. Future studies that examine the processes leading to improved defensive functioning are likely to inform good clinical practice.

The results of our study need to be interpreted in light of several potential limitations. First, while the sample size was limited, modeling data from multiple assessment times resulted in fairly precise and significant measurements of change. Second, the subjects in our

study were diagnosed with depression, anxiety- and adjustment disorders, treated in an outpatient setting. Therefore, the generalizability of our results to more severely impaired patients or patients in an inpatient setting is uncertain. Furthermore, our sample was too small to calculate subgroup analyses based on diagnoses, thus disregarding possible differential effects. Hereby, we refer to previous studies that have investigated defensive functioning for the distinct diagnostic groups of depressive (Bond & Perry, 2004; Perry & Bond, 2009), anxiety- (Kipper et al., 2005; Heldt et al., 2007), and adjustment disorders (Doruk, Sütcigil, Erdem, Isintas, & Özgen, 2009). Third, the direction of causal influences between change in defenses and change in symptoms could not be determined. Last, our study did not address the relationship between defenses and other important factors in psychotherapy, such as the therapeutic alliance, interpersonal problems, personality pathology and recovery.

The novel contributions of our study are twofold. First, we measured the effects of defensive functioning and changes thereof on psychotherapy outcome using longitudinal analyses. Compared to previous research in the field with two measuring points per patient, we had four assessment times of both defenses as well as symptoms of anxiety and depression. Thus, we were able to apply mixed models and to estimate the random effects of defenses on treatment outcome (Raudenbush & Bryk, 2002). In doing so, we were able to deal with the dependency of the observations (i.e., correlations of the residuals), accounting for the variability of defense effects among patients and estimating the effects for each individual patient. Second, to our knowledge, this is the first study that calculates both within- and between patient effects of defensive functioning on symptom severity of depression and anxiety in the same model, thus controlling for the effect of the respective other (i.e., hybrid random effects models; Falkenström et al., 2017). These procedures allow for a robust and unbiased estimation of the parameters and could build a basis for future studies aiming to measure defensive functioning longitudinally.

In conclusion, two main findings emerge from our study. The first is that overall defensive functioning (ODF) exhibited a significant between-patient effect on both depressive and anxiety symptoms. The second main finding concerns a differential prediction of changes in anxiety and depressive symptoms by defenses, i.e., an increase in adaptive and a decrease in immature defenses over the course of treatment (within-patient effects) predicted symptom reduction of depression whereas a decrease in neurotic and immature defenses was associated with reductions in anxiety symptoms.

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Table 1.
Models with ODF as predictor

	Severity level		Within-patient ODF effect	
Fixed Model Effects	γ	*SE*	γ	*SE*
BDI models				
Unconditional model				
Intercept	13.97***	1.22		
Main effect model				
Intercept	13.78***	1.01	-2.43*	1.23
Btw ODF	-7.51***	1.62	-0.59	1.69
Model comparison	$\Delta \chi^2(5) = 24.66, p < .001$			
BAI models				
Unconditional model				
Intercept	12.04***	1.09		
Main effect model				
Intercept	11.84***	0.95	-1.66	1.10
Btw ODF	-5.50***	1.53	0.003	1.38
Model comparison	$\Delta \chi^2(5) = 13.74, p = .02$			

Note.

Table 2.
Models with defense categories as predictors

	Severity level		Within-patient Adaptative Def. effect		Within-patient Neurotic Def. effect		Within-patient Immature Def. effect	
Fixed Model Effects	γ	*SE*	γ	*SE*	γ	*SE*	γ	*SE*
BDI model								
Unconditional Model								
Intercept	13.97***	1.22						
Main Effects Model								
Intercept	13.83***	1.07	-0.35*	0.16	0.24	0.14	0.34*	0.17
Adaptative Def.	-0.49**	0.16	0.03	0.02	0.01	0.02	-0.02	0.02
Neurotic Def.	-0.10	0.25	0.08	0.04	0.01	0.03	0.04	0.05
Immature Def.	0.40*	0.17	-0.02	0.02	0.01	0.02	-0.03	0.03
Model comparison			$\Delta \chi^2(24) = 35.30$, $p = .06$					
BAI model								
Unconditional Model								
Intercept	12.04***	1.09						
Interactional Model								
Intercept	12.01***	0.96	-0.21	0.18	0.37*	0.15	0.38*	0.15
Adaptative Def.	-0.02	0.14	0.03	0.03	0.04*	0.02	-0.02	0.02
Neurotic Def.	0.24	0.23	0.08	0.04	0.02	0.03	-0.01	0.01
Immature Def.	0.52***	0.15	-0.02	0.02	0.02	0.02	0.001	0.03
Model comparison			$\Delta \chi^2(24) = 45.02$, $p = .006$					

Note. *** $p < .001$, ** $p < .01$, * $p \leq .05$