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How personality functioning shapes symptom development during and after treatment: A random intercept cross lagged panel analysis

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ABSTRACT

Objectives: This study investigates the long-term influence of fluctuations around the individual's means of personality functioning (PF) on mental and physical symptoms in a large sample of patients receiving psychotherapy.

Methods: We reanalyzed observational data of 2094 participants (68% female; age M = 39.89 years; SD = 14.20 years) which took part in the Multicenter Effectiveness Study of Inpatient Psychosomatic Psychotherapeutic Treatment (short: MEPP) at three time points. A Random Intercept-Cross Lagged Panel Model (RI-CLPM) was used to examine within-person longitudinal effects between PF (OPD-SQS) and depressive symptoms (PHQ-9), anxiety (GAD-7) and somatic symptom load (PHQ-15). The model was controlled for age, sex, treatment condition and treatment length.

Results: Strong between-person intercorrelations were observed between all investigated constructs. At the longitudinal within-person level PF at hospital discharge predicted somatic symptom load ($p < .01$), depressive symptoms, anxiety and itself (all $p < .05$) at follow up, however, PF showed no effect during treatment ($p > .05$). Furthermore, depressive symptoms at baseline influenced general symptom burden and PF development during treatment (all $p < .05$) and showed strong effects on anxiety and somatic symptoms at follow up ($p < .001$).

Conclusions: The results suggest a sustained influence of intraindividual PF deviations on general symptom development after psychotherapy, but also emphasize the effect of depressive symptoms in psychotherapy outcomes.

1. Introduction

The categorical classification of personality disorders of the DSM-IV [4] and ICD-10 [81] has undergone a considerable paradigm shift towards a dimensional approach. This shift has manifested in the inclusion of the Alternative Model for Personality Disorders (AMPD) in the DSM-5 [5] and the integration of a dimensional severity approach of personality pathology in the ICD-11 [68]. These developments can be viewed as an incorporation of the psychodynamic concept of personality structure into the mainstream conceptualization of mental health, operationalized through the construct of personality functioning [9].

PF represents the observable formations of the latent personality structure [71]. In turn, personality structure may be conceptualized as the foundational framework of personality, encompassing the disposition for inter- and intrapersonal regulatory capacities [39]. Rooted in psychodynamic theory, the development of this concept can be traced back to Freud's early models [32,33], but has since undergone extensive development and reformulation over the past century (e.g. [1,11,31,37,45,74]). More recently, the task force Operationalized Psychodynamic Diagnosis (OPD) strived to develop a psychodynamically grounded and at the same time cross-theoretical and empirically informed approach to assess personality structure and functioning [70,71]. The structural axis of the OPD describes personality functioning as basic abilities of perception, regulation, emotional contact and attachment to inner and outer objects [22]. Originally designed as a semi-structured, expert-rated interview, the OPD framework has since been complemented by a self-report questionnaire available in both long (OPD-SQ; [21]) and short (OPD-SQS; [22]) versions. The short version was constructed based on exploratory factor analysis [23] and showed strong convergence with the DSM-5 expert rating of PF [82] and other self-report measures of PF like the Inventory of Personality Organization and Borderline Personality Inventory [50,79].

Previous studies indicated that PF as measured with the OPD-SQS exhibit significant associations with a wide range of psychopathological symptoms [23]. Investigations using the OPD-SQS specifically indicate links between PF and adverse childhood experiences and general psychopathology [51], suicide and self-injury [25], type I trauma and PTSD [28], depression and somatization [14,80], as well as anxiety [23,30].

PF has been increasingly assessed in psychotherapy studies as well. Studies demonstrated, for example, that psychotherapy is able to significantly improve PF [18,19,52,56,61,62]. Moreover, deficits in PF were suggested as predictor of psychotherapy dropout [41,47] and poorer symptomatic outcomes during treatment in multiple studies [7,8,38,40,52].

Specifically Bierling et al. [7] and Heinzmann et al. [38] used OPD-SQS data from the naturalistic "Multicentre Effectiveness Study of Inpatient Psychosomatic Psychotherapeutic Treatment" (short: MEPP) study [16,17], aimed to estimate the role of PF in symptom change by utilizing the latent change score modelling approach. Bierling et al. [7] observed a recursive relationship between PF and symptom change, meaning that more change in PF predicted larger changes in anxiety and depression symptoms, and vice versa, while Heinzmann et al. [38] observed that baseline PF as well as change in PF predicted improvement in eating disorder symptomatology. From a theoretical perspective, it can be expected that the level of achieved PF at the end of therapy is also relevant for the symptom development in the time after treatment, as the supportive effect of the therapeutic relationship gradually diminishes and the patient increasingly has to rely on their own internal regulatory capacity.

Traditional cross-lagged panel models (CLPMs) have a long history of use in the study of longitudinal interactions between psychological constructs [20,43]. In short, CLPMs examine how variables influence each other over time while accounting for their previous values. However, these models have been substantially criticized in the past decades, e.g., for not distinguishing between stable differences between individuals and dynamic changes within individuals over time [36,64]. In the light of recent developments in longitudinal modelling, the application of the so called Random Intercept Cross-Lagged Panel Model (RI-CLPM; [36]) may offer a complementary perspective on the role of PF in symptom development in response to psychotherapy [29]. As an extension of traditional CLPMs, RI-CLPM not only controls for autoregressive effects (e.g. the effect of baseline PF on PF at discharge), but also – unlike traditional cross-lagged or latent change score models – explicitly separates within-person dynamics from stable between-person differences. Between-person effects reflect stable average differences across individuals, while within-person effects capture time-specific deviations from an individual's mean level of functioning or symptoms. As such, the within-person temporal effects (i.e., autoregressive and cross-lagged estimates) provide insight into fluctuations from each individual's average scores over time [36].

1.1. Research aims and hypothesis

Generally, we assume significant correlations between PF and the investigated health indicators (anxiety, depressive and somatic symptom burden) at all three time points. However, the overarching goal of this study is to increase the understanding of intraindividual processes of change in response to treatment. For this aim, it is necessary to disentangle trait-like between-person effects from dynamic within-person

processes. Hence, we will investigate the examined symptom variables in relation to PF, all measured at three time points (T0 = Baseline, T1 = at discharge, T2 = at 1-year follow up).

Based on prior findings, we hypothesize that deviations from an individual's average PF mean will predict fluctuations in symptom severity from T0 to T1 (admission to discharge) and T1 to T2 (discharge to 1 year after discharge). In this regard, the employed RI-CLPM approach will also make potential within-person reciprocal effects observable.

2. Material and methods

2.1. Sample and procedure

This study is a reanalysis of the *Multicentre Effectiveness Study of Inpatient and Day Hospital Treatment in Departments of Psychosomatic Medicine and Psychotherapy in Germany* (short: MEPP), which includes data from 19 German university hospitals. All measures were administered at three time points (T0, T1 and T2). The detailed study description is given in Doering et al. [16].

2.2. Psychotherapeutic treatment

As described by Bierling et al. [7] and Doering et al. [17], all investigated patients received either day hospital or inpatient treatment, with an either cognitive-behavioral or psychodynamic approach, at one of the hospitals participating in the study. Patients remained in day hospital treatment for a mean duration of 46.5 (SD = 20.2) days, while inpatient treatment had an average duration of 53.8 (SD = 23.0) days.

2.3. Psychometric assessment

2.3.1. Sociodemographic information

After providing informed consent, participants completed a sociodemographic questionnaire collecting all personal data relevant to the study.

2.3.2. Personality functioning

The 12 item version of the Operationalized Psychodynamic Diagnostics Structure Questionnaire (OPD-SQS; [22]) is a self-report instrument designed to assess impairments in personality functioning, as conceptualized in the Operationalized Psychodynamic Diagnosis [70]. The items are rated on a 5-point Likert scale from 0 (“strongly disagree”) to 4 (“strongly agree”) and assess impairments in the subdomains “self-perception”, “shaping contact”, and “relationship model”. Only the total score was used in the current study, which reflects overall deficits in personality functioning, with higher scores indicating more severe impairments. The total score showed good to excellent internal consistency in previous studies with Cronbach's $\alpha = 0.87–0.89$ [22] and McDonald's $\omega = 0.92$ [23]. The test retest reliability was assessed as acceptable with $r_{tt} = 0.63–0.67$; [63].

2.4. Psychopathological symptoms

2.4.1. Depressive symptoms

Depressive symptoms were measured using the depression module of the Patient Health Questionnaire (PHQ—D;), which assesses depressive symptoms in the last 2 weeks based on DSM-IV criteria with 9 items (PHQ-9; [53]) on a 4-point-likert scale ranging from “0 = not at all” to “3 = nearly every day”. Previous investigations suggested good reliability with Cronbach's $\alpha = 0.85–0.89$; retest reliability = 0.84 [55].

2.4.2. Anxiety

To operationalize anxiety the Generalized Anxiety Disorder (GAD-7; [77]) module of the PHQ-D was used. The GAD-7 uses 7 items which are self-reported on the same Likert format as the PHQ-9. Previously, excellent internal consistency and retest reliability with Cronbach's $\alpha = 0.92$ and $r_{tt} = 0.83$, respectively, were observed [77].

2.4.3. Somatic symptoms

The PHQ-D somatic symptoms module (PHQ-15; [54]) was utilized to measure somatic symptom burden over the past four weeks. Each of the 15 items is self-rated on a 3-point scale: 0 (“not bothered at all”), 1 (“bothered a little”), and 2 (“bothered a lot”). The PHQ-15 showed good internal consistency in previous studies ($\alpha = 0.82$; [48]) and a moderate test-retest reliability ($\kappa = 0.60$; [78]).

2.5. Statistical analysis and analysis strategy

SPSS 29.0 was used for data management, descriptive statistics, repeated measure ANOVAs, intra class coefficients (ICC), internal consistencies (using McDonald's ω) and bivariate Pearson correlations.

To examine longitudinal associations a Random-Intercept Cross-Lagged Panel Model (RI-CLPM) was estimated using the R package lavaan [75] in RStudio (Version 4.3.3). The syntax was adapted from the code provided by Mulder and Hamaker [67] and is provided in the supplementary file S1. Initially proposed by Hamaker et al. [36] the RI-CLPM is able to separate the analysis of within- and between-person effects, as it accounts for stable, trait-like differences between individuals, by modelling them as random intercepts. Hence, autoregressive and cross-lagged paths in the RI-CLPM assess variability of participants' mean values [29]. In the model, random intercepts are conceptualized as latent constructs, with individual observations acting as their respective indicators. To establish model identification, the factor loadings were constrained to 1. Missing data were handled using full information maximum likelihood (FIML). Maximum likelihood with robust standard errors (MLR) was employed as estimator. The model included all autoregressive and cross-lagged paths, as well as random intercepts for each variable to account for stable between-person differences. In line with Byrne [12], the following fit indices were considered as markers for an excellent model fit: (a) the comparative fit index (CFI) > 0.95, (b) Tucker-Lewis index (TLI) relative fit index >0.95, (c) the square root error of approximation (RMSEA) < 0.05, and the upper bound of its 90% confidence interval < 0.1 as well as (d) Standardized Root Mean Square Residual (SRMR) < 0.08. The model was controlled for age, sex, treatment condition (inpatient vs. day clinic treatment) and treatment duration.

3. Results

3.1. Drop out

While 152 (7.3%) patients dropped out between T0 and T1 (see the flowchart in [17] for further details), at follow-up, 1269 (60.6%) of the original patients at admission were still participating in the study. Between T0 and T1, most participants dropped out of treatment due to discharge against medical advice (47%) or early dropout in the first week of treatment (35%), followed by violations of treatment contracts (11%) and transfers to other departments (7%). No further information was available regarding attrition between T1 and T2.

3.2. Sample characteristics, descriptive results and trends

Reported in Table 1, the majority of participants were female (68%), had a high school diploma (29.9%), were in full time employment (32.2%) and were married or in a partnership (48.3%) and of German nationality (89.6%). Further diagnostic details are provided in [16] online Supplementary Table S1 (see www.karger.com/doi/10.1159/000527881). The most common diagnoses were depression (ICD-10: F32, F33, F34.1, F43.2) in 82.6% of patients. Most patients (85%) had multiple axis I diagnoses, and 65.1% had at least one somatic disorder. The detailed sample description can be found in Doering et al. [16].

Table 2 depicts the mean values and SDs of the investigated

Table 1
Sample descriptives.

		Mean	SD
Age		39.89	14.20
	Category	**Frequency**	**%**
Sex	Female	1424	68.0
	Male	660	31.5
	Missing	10	0.5
Educational status	No degree	36	1.7
	Compulsory education	160	7.6
	Apprenticeship	488	23.3
	High school diploma	621	29.7
	University	362	17.3
	Still in school	47	2.2
	Other	183	8.8
	Missing data	197	9.4
Occupation	Still in education/training	278	13.3
	Homemaker	65	3.1
	Unemployed	411	19.6
	Part-time employed	267	12.8
	Full-time employed	675	32.2
	Retired	217	10.4
	Missing	181	8.6
Marital Status	Single without partner	623	29.8
	Single with partner	431	20.6
	Married / Registered partnership	581	27.7
	Divorced	252	12.0
	Widowed	28	1.3
	Missing	179	8.5
Diagnosis	Depressive disorder (ICD-10: F32, F33, F34.1, F43.2)	1729	82.6
	Anxiety disorder (F40, F41)	1079	51.5
	PTSD / acute stress reaction (F43.0, F43.1, F43.8, F43.9)	478	22.8
	Eating disorder (F50)	430	20.5
	Somatoform disorder (F44, F45)	896	42.8
	Personality disorder (F60)	858	41.0

variables. Calculated using baseline SDs, effect sizes (Cohen's d) from admission to discharge (T0–T1) revealed medium-to-large improvements for depression (d = 0.76) and anxiety (d = 0.74), and small improvements for somatic symptoms (d = 0.38) and PF (d = 0.31). These reductions were largely sustained at 1-year follow-up (T0–T2) for depression (d = 0.61), anxiety (d = 0.63), somatic symptoms (d = 0.32), and personality functioning (d = 0.30).

3.3. Internal consistency, intra class coefficients and bivariate correlations

As displayed in Table 2, all variables showed internal consistencies ≥0.79 and generally significant positive correlations amongst each other (all $p < .001$). The strongest cross-sectional zero-order correlations were found between anxiety and depressive symptoms at T1 ($r = 0.80$), and at T2 ($r = 0.79$). For the investigated variables the ICC indicated moderate to high retest reliability (Depressive symptoms: ICC =0.80; Somatic symptoms: ICC = 0.86; Anxiety: ICC = 0.76; and PF: ICC =0.88).

3.4. Random intercept cross lagged panel model

The Yuan-Bentler corrected RI-CLPM exhibited excellent model fit (RMSEA = 0.044 (90% CI: 0.032–0.046), CFI = 0.993, TLI = 0.978, SRMR = 0.026), indicating that the specified between- and within-person structure adequately accounts for the observed data.

3.4.1. Random intercepts correlations

Random intercepts (RI) of the investigated variables showed significant correlations ranging from $r = 0.80$ ($p < .001$) between RI depressive symptoms and RI anxiety to $r = 0.53$ ($p < .001$) between PF and somatic symptoms. The detailed RI correlations are presented in Table 3.

3.4.2. Within correlations

The within term correlations followed a similar pattern (see Table 3). With the exception of the within factor of PF and somatic symptoms at T0 ($p < .05$), all investigated within factors were positively correlated with each other at all 3 measuring points, ranging from anxiety x PF: $r = 0.30$ ($p < .01$) at T0 to anxiety x depressive symptoms: $r = 0.80$ ($p < .001$) at T1 (see Table 4).

3.4.3. Within longitudinal effects

Fig. 1 visualizes the significant within autoregressive and cross lagged paths of the RI-CLPM model. The detailed results can be found in Table 5. Thereby, a significant effect indicates that levels above the participant's mean at one time point predicted an increase in the level of the same variable in later time points.

Table 3
Intercorrelations between random intercepts (between-factor).

	1	2	3	4
1. RI Depressive Symptoms	–			
2. RI Anxiety	0.80	–		
3. RI Somatic Symptoms	0.58	0.64	–	
4. RI PF	0.78	0.74	0.53	–

Note. All $p \leq .001$.

Table 2
Bivariate zero-order correlations, means and standard deviations of investigated variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Depressive Symptoms T0	–											
2. Somatic Symptoms T0	0.51	–										
3. Anxiety T0	0.70	0.51	–									
4. PF T0	0.60	0.37	0.57	–								
5. Depressive Symptoms T1	0.60	0.42	0.43	0.45	–							
6. Somatic Symptoms T1	0.41	0.72	0.39	0.34	0.66	–						
7. Anxiety T1	0.48	0.42	0.53	0.47	0.80	0.62	–					
8. PF T1	0.52	0.38	0.46	0.72	0.67	0.53	0.68	–				
9. Depressive Symptoms T2	0.49	0.34	0.35	0.36	0.61	0.46	0.50	0.50	–			
10. Somatic Symptoms T2	0.34	0.60	0.31	0.26	0.47	0.69	0.44	0.41	0.67	–		
11. Anxiety T2	0.43	0.40	0.48	0.39	0.55	0.46	0.59	0.49	0.79	0.65	–	
12. PF T2	0.49	0.35	0.42	0.67	0.54	0.42	0.54	0.73	0.69	0.54	0.69	–
Mean	1.64	0.87	1.66	2.10	1.14	0.73	1.13	1.84	1.24	0.75	1.21	1.85
Standard Deviation	0.66	0.37	0.72	0.83	0.65	0.37	0.70	0.89	0.72	0.40	0.77	0.92
McDonald's Omega	0.85	0.79	0.85	0.87	0.88	0.84	0.89	0.91	0.89	0.84	0.90	0.91

Note. All correlations are significant with p < .001; values in the "Total Scale Mean," "SD," and "McDonald's Omega" rows refer to the respective variables in columns 1 to 12.

Table 4
Intercorrelations between within factors.

	1	2	3	4	5	6	7	8	9	10	11
1. wDepressive Symptoms T0											
2. wSomatic Symptoms T0	0.44										
3. wAnxiety T0	0.59	0.33									
4. wPF T0	0.32	0.05	0.30								
5. wDepressive Symptoms T1											
6. wSomatic Symptoms T1					0.74						
7. wAnxiety T1					0.80	0.58					
8. wPF T1					0.52	0.51	0.57				
9. wDepressive Symptoms T2											
10. wSomatic Symptoms T2									0.73		
11. wAnxiety T2									0.77	0.62	
12. wPF T2									0.62	0.59	0.69

Note. All p ≤ .001, except PF x Somatic Symptoms (p > .05).

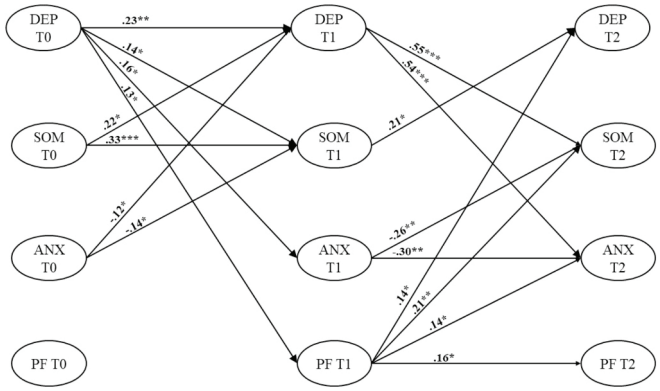

Fig. 1. RI-CLPM within effects between personality functioning (PF) and depressive symptoms (DEP), anxiety (ANX) and Somatic symptoms (SOM), controlled for age, sex effects, treatment condition (inpatient vs. day clinic) and treatment days.
* p < .05; ** p < .01; ***p < .001.

3.5. T0 ➔ T1

From baseline to discharge, somatic symptoms (β = 0.33; $p < .001$) and depressive symptoms (β = 0.23; $p = .001$) had significant autoregressive effects.

In terms of cross-lagged effects between T0 and T1, higher than subjective average somatic symptoms predicted increases in depressive symptoms (β = 0.33; p < .001), while increased anxiety predicted a decrease in depressive symptoms (β = −0.12; $p < .05$) and somatic symptoms (β = −0.14; p < .05). Depressive symptoms at T0 further predicted increased somatic symptoms and PF defiicts (β = 0.14 and β = 0.13, respectively; both $p < .05$) and anxiety (β = 0.16; p < .05).

3.6. T1 ➔ T2

Between discharge and follow up anxiety showed a negative autoregression effect (β = −0.27; $p < .01$), while PF had a positive autoregression (β = 0.16; $p = .05$).

Furthermore, PF had significant effects on depressive symptoms (β = 0.14; p < .05), somatic symptoms (β = 0.21; p < .01), and anxiety (β = 0.14; p = .05). Depressive symptoms at T1 predicted somatic symptoms (β = 0.55) and anxiety (β = 0.54; both $p < .001$). In terms, somatic symptoms predicted depressive symptoms (β = 0.21; p < .05). Finally, anxiety showed an inhibiting effect on somatic symptoms (β = −0.26; p < .01).

4. Discussion

This study investigated the effect of personality functioning (PF) in the intraindividual development of psychopathological symptoms within a naturalistic psychotherapy setting. In general, a pattern emerged suggesting a transdiagnostic significance of PF for predicting symptom development following psychotherapy. Contrary to our expectations, we did not find significant within-person effects of PF from baseline (T0) to discharge (T1) for depressive symptoms, somatic symptom burden as well as for anxiety and PF itself. However, consistent but modest effects of PF emerged from discharge (T1) to follow-up (T2). At the same time, baseline depression predicted PF at discharge, but not at follow up. Overall, specifically depressive symptoms showed strong influence at both measuring points, while – paradoxically - anxiety showed an inhibiting influence on psychopathology.

To our knowledge, this is the first study to disentangle within-person from between-person effects of PF on symptom change using the RI-CLPM method. Previous analyses of the role of PF in general [8,40,52] and in the MEPP dataset in particular [7,38] showed that poorer PF at admission negatively interferes with treatment outcomes. The present findings extend that work by indicating that in individuals, improvements in PF relative to their own average by the end of treatment, may foster a more favorable symptom courses once treatment at the hospital has ended.

This observation is in agreement with theoretical literature on the role of PF, which emphasizes its structural, i.e. time invariant, role regarding self and relational regulation skills [39]. However, from a within-person perspective, PF seems to be less relevant for symptom change during the time of the psychotherapeutic treatment. Here the holding and containing functions inherent to the psychotherapeutic relationship and milieu might compensate for a lack of internalized regulation capacities [69].

This observation suggests that patients whose personality functioning remains below their individually achieved level at the end of treatment may require more intensive, structurally oriented mental health care following discharge from inpatient or day hospital treatment. For these patients, outpatient aftercare structures such as the Psychosomatic Institute Outpatient Clinics (“PsIA”), or comparable ambulatory post-treatment programs, might offer promising options [76]. In clinical practice, this may imply that PF could be explicitly considered in discharge planning, for example by integrating it into psychoeducation and discussing the potential need for continued psychotherapy beyond the inpatient setting. Along these lines, clinicians might use information on PF to guide decisions regarding the duration, intensity, and focus of follow-up treatment, particularly with regard to previous findings indicating the effectiveness of interventions that specifically target structural capacities such as affect regulation and interpersonal functioning [18,47,56,62]. While recent work of Dönnhoff et al. [19] has already identified several general factors influencing the development of PF during treatment (including the detrimental effect of

Table 5
Within-person effects for investigated time waves.

Dependent	Predictor	SE	p	Standardized Estimate
Autoregression				
Depressive Symptoms T1	Depressive Symptoms T0	0.071	0.001	0.230
Somatic Symptoms T1	Somatic Symptoms T0	0.092	<0.001	0.328
Anxiety T1	Anxiety T0	0.058	0.169	−0.084
PF T1	PF T0	0.123	0.717	0.035
Depressive Symptoms T2	Depressive Symptoms T1	0.150	0.143	0.177
Somatic Symptoms T2	Somatic Symptoms T1	0.134	0.751	−0.037
Anxiety T2	Anxiety T1	0.128	0.004	−0.304
PF T2	PF T1	0.078	0.019	0.160
Cross-Lagged				
Depressive Symptoms T1	Somatic Symptoms T0	0.173	0.010	0.218
Depressive Symptoms T1	Anxiety T0	0.053	0.030	−0.123
Depressive Symptoms T1	PF T0	0.117	0.208	−0.125
Somatic Symptoms T1	Depressive Symptoms T0	0.032	0.020	0.139
Somatic Symptoms T1	Anxiety T0	0.025	0.023	−0.116
Somatic Symptoms T1	PF T0	0.055	0.540	−0.054
Anxiety T1	Depressive Symptoms T0	0.067	0.018	0.155
Anxiety T1	Somatic Symptoms T0	0.176	0.247	0.099
Anxiety T1	PF T0	0.119	0.549	−0.06
PF T1	Depressive Symptoms T0	0.071	0.043	0.129
PF T1	Somatic Symptoms T0	0.180	0.193	0.105
PF T1	Anxiety T0	0.059	0.277	−0.062
Depressive Symptoms T2	Somatic Symptoms T1	0.204	0.017	0.205
Depressive Symptoms T2	Anxiety T1	0.107	0.348	−0.081
Depressive Symptoms T2	PF T1	0.063	0.011	0.141
Somatic Symptoms T2	Depressive Symptoms T1	0.081	<0.001	0.545
Somatic Symptoms T2	Anxiety T1	0.058	0.007	−0.259
Somatic Symptoms T2	PF T1	0.039	0.003	0.205
Anxiety T2	Depressive Symptoms T1	0.159	<0.001	0.540
Anxiety T2	Somatic Symptoms T1	0.228	0.711	−0.036
Anxiety T2	PF T1	0.077	0.033	0.144
PF T2	Depressive Symptoms T1	0.149	0.788	−0.032
PF T2	Somatic Symptoms T1	0.224	0.093	0.158
PF T2	Anxiety T1	0.114	0.277	0.100

Note. PF = Personality Functioning.

restrictions in social participation due to physical and emotional difficulties, as well as interpersonal problems marked by emotional detachment or excessive self-sacrifice), the present findings support further investigation of specific treatment factors that promote improvements in PF. However, it should be noted that the present study does not allow for the identification of specific cut-off values or risk thresholds. Conclusions regarding individual prognosis should therefore be drawn with restraint.

Of note, while the presented results are – at first glance – in contradiction to Bierling et al. [7], they mirror methodological differences between Latent Change Score (LCS) models and RI-CLPM. The LCS model assessed how a person's starting PF level related to their subsequent symptom improvement (and vice versa) without isolating stable traits [72]. Consequently, the LCS findings may partly reflect between-person differences (patients with worse initial PF generally improve less compared to others), whereas RI-CLPM pinpointed within-person change processes. In practical terms, two patients who start therapy with similarly low PF can have very different prognoses one year after therapy, depending on how much their PF has improved by the time they finish treatment. If one patient's PF remains impaired at discharge, that patient is more likely to experience a resurgence or continuation of symptoms by follow-up. In contrast, if another patient with an initially poor PF attains a higher level of PF by the end of treatment, their symptom improvement is more likely to be sustained over the long term.

While it was observed that lower depression at baseline facilitated PF improvement till discharge, after treatment no other investigated variable influenced PF development. From previous research it is known that there is a strong overlap between general and personality pathology [23]. This was reiterated in the general correlation patterns regarding PF and psychopathology in the investigated data. In prinicple, it seems plausible that a reduction of psychopathological symptom burden might foster the stabilization of PF, as the decrease of mental health burden might have an exonerating impact on intra- and interpersonal functioning. To further investigate the question of potential reciprocal effects, future studies could investigate models, which only observe the links between PF and one psychopathological symptom cluster at a time, in order to exclude potential overlap effects regarding the closely associated psychopathological syndromes. With this reservation, however, the results suggest that PF has more influence on symptom development after psychotherapy than vice versa. Consequently, routinely monitoring PF may help identify patients in need of more intensive follow-up and support, ultimately enhancing long-term treatment outcomes.

We further observed longitudinal effects of the differential symptom clusters amongst each other. In this context, depression was suggested as particularly influential, with links to all investigated measures at discharge as well as a strong influence on somatization and anxiety at follow up. Hitherto, research on within effects between different psychopathological disorders has been very sparse. Previous studies using the RI-CLPM approach suggested bidirectional effects between somatic symptoms and depression [3]. Similarly, Komischke-Konnerup et al. [49] observed intertwined relationships between PTSD, depression and prolonged grief. Along these lines, cross sectional comorbidity networks indicated a strong hub function of depressive symptoms and affects in relation to other disorders [2,10,13,34,66,73].

Another noteworthy finding emerged for anxiety. In the investigated model, increased anxiety exhibited significant decreasing influences on somatic (T1 & T2) and on depressive symptoms (T1) as well as on itself (T2). This seemingly counterintuitive result likely reflects a statistical suppression effect, driven primarily by the strong positive cross-lagged influence of depression on anxiety. Depression and anxiety are highly interrelated [46,58], and their within-person fluctuations are strongly intertwined in the present sample. Hence, the result could suggest that anxiety fluctuations occurring *independently* of concurrent depression spikes might be more transient or perhaps trigger compensatory mechanisms leading to a subsequent decrease below the individual's average anxiety level.

4.1. Limitations and future perspectives

While this study has several strengths including a large multicenter generated sample in naturalistic settings, the conclusions are also limited by a number of shortcomings.

A major limitation of this study concerns missing data. Although the proportion of missingness is within the expected range for a large-scale naturalistic psychotherapy study, attrition by follow-up accounts for nearly 40% of the initial sample. This raises the possibility of systematic bias in parameter estimation [60]. To address this issue, full information

maximum likelihood (FIML) was employed, a method considered robust and relatively unbiased for handling incomplete data [24,59]. However, FIML relies on the Missing At Random (MAR) assumption that cannot be empirically tested from the observed data alone. In clinical settings, attrition is often Missing Not At Random (MNAR); for example, patients who experience clinical deterioration after discharge may be systematically less likely to complete follow-up assessments. Because FIML cannot correct for distortion driven by unmeasured factors, in particular the strength of the long-term effects of the investigated variables may be estimated in a biased way.

A further methodological consideration relates to the complexity of the estimated RI-CLPM. The model simultaneously estimates a large number of parameters in the context of substantial missing data. Some of the observed within-person cross-lagged and autoregressive effects, such as the long-term influence of PF on anxiety, were modest in size and borderline significant. Hence, those pathways will require independent replication before firm clinical conclusions should be drawn.

Another factor limiting the generalizability of our findings is the symptomatic heterogeneity of the investigated, predominantly female and highly educated patient sample, as well as the broad range of therapeutic interventions. Treatment comprised an integrative approach including psychodynamic, cognitive-behavioral, and systemic elements, as well as components of trauma therapy. This encompassed individual and group psychotherapy, creative and body-oriented therapies, mindfulness-based interventions, psychoeducation, social work support, and medical as well as psychopharmacological treatment delivered within a multimodal, interdisciplinary setting.

Given this complexity, it was not possible to control for or disentangle the effects of specific therapeutic components within the MEPP data beyond basic treatment characteristics (inpatient vs. day clinic treatment and treatment duration). Accordingly, the findings are most readily generalizable to similarly complex psychosomatic clinical populations, but may be less applicable to more demographically diverse or socioeconomically disadvantaged groups, or to healthcare systems outside the German context.

Importantly, our findings concern PF as measured in the psychodynamic context of the OPD. Although this construct overlaps strongly with related dimensional models such as DSM-5 AMPD Criterion A and ICD-11 severity on an empirical level [42,44,82], as well as on a conceptual level – insofar as all three assess impairments in self and interpersonal functioning – these frameworks are not identical with regard to their underlying theoretical models. Direct generalization should therefore be made with caution (see [39] for an in-depth discussion of this matter).

Further limitations are the use of self-report measures, as specifically for complex constructs like PF, it might be beneficial to use semi-structured interviews like the OPD-3 clinical interview [57]. However, previous studies showed that the OPD-SQ correlates substantially with the OPD interview rating of personality dysfunction [15].

Further, given the heterogeneity and complexity of post-discharge care, it was not possible to adequately control for its intensity and modality, such as outpatient psychotherapy, medication changes, life events, or participation in structured programs, which poses a significant restriction. Hence, differential utilization of additional treatments may have influenced the observed variability in symptom trajectories during follow-up. Consequently, the within-person effects of PF from T1 to T2 on long-term symptom development may be partially attributable to, or confounded by, these unmeasured post-treatment interventions. Although improved PF at discharge may theoretically enable patients to make better use of subsequent aftercare, future studies should explicitly assess post-discharge treatment in order to disentangle the direct effects of PF from the benefits of additional clinical support.

More generally, this study touches onto the subject of the debates concerning the use of traditional CLPMs and RI-CLPM. While a large part of the methodological literature make a convincing case for the virtues of RI-CLPMs in longitudinal research [26,27,29,35], some aspects (e.g. separation of potentially relevant between-effects, limited potential to control for confounders) of this methodology have been criticized by others in favor of the traditional CLPM [6,65,72]. Hence, for future research it might be interesting to comparatively analyze the MEPP data in a traditional CLPM manner.

Finally, while this study aimed to control for the effects of different treatment durations by including them as a control variable, the variance between measuring points, bias might have been introduced in the estimation of the model. Further research, using e.g. continuous time models, will be necessary to cross-validate the reported relationships.

Future lines of research might include investigating within-person effects between PF and symptom burden in participants not undergoing psychotherapy, in order to further disentangle spontaneous changes from therapy-related improvements. Finally, techniques such as Ecological Momentary Assessment could be employed to achieve higher temporal resolution in capturing the dynamic relationship between PF and symptom burden.

5. Conclusions

In sum, this study denotes that within-person fluctuations in personality functioning (PF) are a robust transdiagnostic predictor of long-term change following psychotherapy. Specifically, when a patient's PF at the end of treatment drops below their personal average, the risk of relapse in somatic and psychopathological symptoms appears to increases. In contrast, improvements in PF beyond baseline levels may be associated with greater health related stability. This pattern remains even after controlling for stable trait differences, as well as autoregressive and cross-lagged effects of anxiety, depression and somatization symptoms. Hence, the findings imply that enduring symptom change is facilitated by structural change and, therefore, support clinical emphasis on improving PF during treatment.

Potential identification of study participants

Not applicable.

CRediT authorship contribution statement

Jürgen Fuchshuber: Visualization, Methodology, Formal analysis, Writing – review & editing, Writing – original draft. **Aram Kehyayan:** Investigation, Writing – review & editing. **Magdalena Pape:** Investigation, Writing – review & editing. **Tobias Hofmann:** Investigation, Writing – review & editing. **Matthias Rose:** Investigation, Writing – review & editing. **Katrin Imbierowicz:** Investigation, Writing – review & editing. **Franziska Geiser:** Investigation, Writing – review & editing. **Ilona Croy:** Supervision, Methodology, Investigation, Conceptualization, Writing – review & editing, Writing – original draft. **Kerstin Weidner:** Conceptualization, Writing – review & editing, Writing – original draft. **Jörg Rademacher:** Investigation, Writing – review & editing. **Silke Michalek:** Investigation, Writing – review & editing. **Eva Morawa:** Investigation, Writing – review & editing. **Yesim Erim:** Investigation, Writing – review & editing. **Martin Teufel:** Investigation, Writing – review & editing. **Alexander Bäuerle:** Investigation, Writing – review & editing. **Stanislav Heinzmann:** Investigation, Writing – review & editing. **Claas Lahmann:** Investigation, Writing – review & editing. **Eva Milena Johanne Peters:** Investigation, Writing – review & editing. **Johannes Kruse:** Investigation, Writing – review & editing. **Dirk von Boetticher:** Investigation, Writing – review & editing. **Christoph Herrmann-Lingen:** Investigation, Writing – review & editing. **Mariel Nöhre:** Investigation, Writing – review & editing. **Martina de Zwaan:** Investigation, Writing – review & editing. **Ulrike Dinger:** Investigation, Writing – review & editing. **Hans-Christoph Friederich:** Investigation, Writing – review & editing. **Alexander Niecke:** Investigation, Writing – review & editing. **Christian Albus:** Investigation, Writing – review & editing. **Rüdiger Zwerenz:** Investigation, Writing –

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Consent for publication

All authors consented for the publication of the manuscript.

Ethics statement

The study was initially approved by the Ethics Committee of the medical faculty of the Ruhr-University Bochum on October 17, 2018 (ID: 18–6388, this approval was subsequently confirmed by the Ethics Committees of the participating universities) and was registered at the German Clinical Trials Register (www.drks.de; ID: DRKS00016412). All patients gave written informed consent for their participation in the study.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work ChatGPT 5 was used for grammar and spell checking. After using this tool the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Declaration of competing interest

Christoph Herrmann-Lingen reports royalties from Hogrefe Publishing. Christoph Herrmann-Lingen reports a speaker's honorarium from Novartis AG. All authors, except for Stephan Doering, are working at one of the German university departments of psychosomatic medicine and psychotherapy. The other authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The European General Data Protection Regulation (GDPR) does not allow to share personal data of patients publicly (https://gdpr.eu). The Ethics Commissions of all of the study centers have approved the study under the condition that even the transfer of data from the German sites to the Austrian PI (Stephan Doering) can only take place according to specific security regulations. Further inquiries can be directed to the corresponding author.

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