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Schema modes as mechanisms of change in treating borderline personality disorder: A model replication study

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ABSTRACT

Objective: Schema modes have been theorized and tested as mechanisms of change in the treatment of personality disorders. We investigated whether schema modes also function as mechanisms of change in borderline personality disorder (BPD) treatment.

Method: Data from 494 patients (N = 68 male, N = 426 female) with borderline PD who participated in an international randomized clinical trial on effectiveness of two group schema therapy formats vs treatment as usual were analyzed. Granger Causality was tested partially replicating the model predicting PD severity as derived in Yakın et al. (2020) with generalized linear mixed models, testing within-person relationships over time.

Results: The effect of the Healthy Adult, the Vulnerable Child and Detached Self-Soother as an Avoidant Coping mode on PD severity, and the reciprocal relationship between the Healthy Adult and the Vulnerable Child were replicated. Unlike previous findings, the Avoidant Coping mode is not predicted by the Healthy Adult. Moreover, the relationship between Impulsive Child and PD severity was unidirectional. The relationships between Healthy Adult, Self-Aggrandizer, and functioning over time were also replicated, but unlike earlier results, Self-Aggrandizer did not influence later scores of the Healthy Adult.

Conclusions: Central relationships in the model were replicated. The centrality of the Healthy Adult and the Vulnerable Child for the treatment of PDs was also applicable to BPD. It appears that these two modes should be primary treatment targets compared to the other modes, unless the avoidant modes block access to the more vulnerable parts of the personality.

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1. Introduction

Schema therapy (ST) is an evidence-based treatment for personality disorders (Arntz et al., 2022; Assmann et al., 2024; Bamelis et al., 2014; Dickhaut & Arntz, 2014; Farrell et al., 2009; Giesen-Bloo et al., 2006). Despite the expanded body of knowledge regarding the effectiveness of ST, inconsistencies within the literature hinder the development of a unified model of the mechanisms underlying therapeutic change in ST (Masley et al., 2012; Taylor et al., 2017). Theories previously postulated regarding the mechanisms of change in ST encompass early maladaptive schema modification (Nordahl et al., 2005; Renner et al., 2013; Taylor et al., 2017), insight and clarification (Tschacher et al., 2012), and the therapeutic alliance (Renner et al., 2018). However, the evidence supporting early maladaptive schema modification as a mechanism of change in ST is of low quality (Taylor et al., 2017).

Another theoretical suggestion about the mechanism of change in ST is limited reparenting which is a fundamental aspect of schema therapy (Fassbinder et al., 2016; Lockwood & Perris, 2012; Spinhoven et al., 2007). Schema therapists, within the boundaries of the therapeutic relationship, seek to meet the emotional needs that were inadequately met during the patient's childhood (Young et al., 2003). ST involves guiding the patient toward self-reparenting, a concept critical for addressing unmet emotional needs from childhood. To understand how self-re-parenting works, it is essential to examine how the various schema modes operate.

According to schema theory, when the primary caregivers repeatedly fail to create an environment that meets the child's basic needs, they develop early maladaptive schemas (Young, 1990). In adulthood, these early maladaptive schemas are triggered by situations involving real or perceived abandonment, rejection, or other forms of emotional adversities, leading the individual to flip to the Vulnerable Child (VC) or other maladaptive modes. In patients with borderline personality disorder (BPD) these modes are typically of brief duration, emerging in response to specific triggers and can quickly change to other emotional states. When the VC is activated, the person experiences intense aversive emotions such as fear, anxiety, and helplessness (Young et al., 2003). In this state, activating the Healthy Adult mode (HA) helps to facilitate recovery. The HA mode enables individuals to remain grounded in the present, cultivate self-compassion, and make informed, realistic decisions via battling against critical and demanding thoughts. During ST, the therapist exemplifies the HA role, guiding the patient toward independently addressing their own needs (Young et al., 2003). The significance of self-reparenting in terms of the temporal relationship between the VC and the HA in ST has been highlighted both theoretically (Fassbinder et al., 2016; Lockwood & Perris, 2012; Lockwood & Samson, 2020) and through empirical evidence (Aalbers et al., 2021; Koppers et al., 2021; Yakin et al., 2020).

It has been postulated that sudden changes in behavior or emotion regulation may be attributed to sudden shifts in schema modes, and that schema modes underlie pathological personality traits (Young et al., 2003). For example, abandonment panic is assumed to follow from activation of the VC mode. Empirical research has shown that modes can change momentarily in response to stress-inducing situations (Lobbestael & Arntz, 2010; Lobbestael et al., 2009) and during therapy sessions (Peled et al., 2017; van den Broek et al., 2011). There is also evidence that ST is associated with an improvement in the severity of maladaptive schema modes both in group and individual treatment settings (Koppers et al., 2021; Yakin et al., 2020). Building on prior evidence regarding the temporal variability of schema modes, we aimed to investigate the extent to which changes in the frequency of activation of schema modes account for changes in borderline personality disorder severity. In doing so, we (re)tested a core assumption of ST which states that change in psychopathological personality features is achieved through changes in schema modes. In an earlier study (Yakin et al., 2020) specific schema modes are identified as mechanisms of change in treating a sample with mixed personality disorders (including avoidant,

dependent, obsessive-compulsive, paranoid, histrionic, and narcissistic personality disorders). In Fig. 1, we provided a description of the key schema concepts related to the mechanisms of change. The model depicting schema modes as mechanisms of change from Yakin et al. is presented in the left panel of Fig. 2. In the light of previous findings, replication remains a critical step in minimizing the likelihood of attributing findings to chance, thereby reducing the risk of drawing erroneous conclusions. Besides, BPD is a more severe personality disorder than the six personality disorders examined in the previous study and usually requires a more extensive treatment. BPD is associated with elevated comorbidity rates with other psychiatric conditions (Shen et al., 2017), increased suicidal ideation, more frequent suicide attempts (Paris, 2019), higher frequency of hospitalization, and higher relapse rate (Leichsenring et al., 2024; Zanarini et al., 2018). Therefore, checking the generalizability of previous findings in patients with BPD is important. In this study we tested whether the previous empirical findings from Yakin et al. can also be found in a sample of patients diagnosed with BPD.

2. Method

2.1. Participants

The participants were recruited as a part of an international, multi-cultural randomized controlled trial (RCT) conducted between June 29, 2010, and May 18, 2016, by Arntz et al. (2022). Data were collected from 495 patients. The average age was 33.6 years ($SD = 9.4$), and the majority of the participants was female (426 patients, 86.2 %). The RCT had three arms: optimal treatment as usual (TAU), predominantly group schema therapy (PGST), and combined individual and group schema therapy (IGST). Participants were randomized in a 1:1 ratio within cohorts of 16–18 individuals per site to either PGST or TAU, or to IGST or TAU. In total, 246 (49.7 %) patients received treatment as usual (TAU), 125 (25.2 %) received PGST, and 124 (25.0 %) received IGST. The study was conducted across 15 sites: two in Australia, three in Germany, one in Greece, seven in the Netherlands, and two in the UK. There were six face-to-face assessment sessions conducted with participants, occurring at baseline and at 6, 12, 18, 24, and 36 months. A comprehensive description of the inclusion and exclusion criteria and methodological considerations including ethical approval, data collection procedures, study protocols, and intervention strategies can be found in Arntz et al. (2022) and Wetzelaer et al. (2014).

2.2. Materials

Schema modes. A modified version made up of subscales of the Schema Mode Inventory, short version (SMI; Lobbestael et al. (2010) and the SMI-2 (Bamelis et al., 2011)) was utilized. The questionnaire consists of 143 items rated on a six-point scale ranging from 'never or almost never' to 'all of the time,' with higher scores on a specific subscale indicating more frequent manifestations of the associated mode. The SMI and the SMI-2 have demonstrated adequate psychometric properties (Bamelis et al., 2011; Lobbestael et al., 2010).

Personality Psychopathology. The severity of BPD symptoms was assessed using the Borderline Personality Disorder Severity Index (BPDSI) (Arntz et al., 2003), a semi-structured interview consisting of 70 items. These items are rated on an 11-point scale ranging from 0 (never) to 10 (daily), except for the identity disturbance subscale which is evaluated using a 5-point Likert scale from 0 (absent) to 4 (dominant, clear and well-defined not knowing who he/she is). The BPDSI measures BPD manifestations over the past three months, therefore, we assessed BPD symptoms within this timeframe. The BPDSI has demonstrated adequate psychometric properties (Arntz et al., 2003; di Giacomo et al., 2018; Giesen-Bloo et al., 2010; Kröger et al., 2013; Leppänen et al., 2013).

Functioning. The Global Assessment of Functioning (GAF) from the

Schema Therapy is an evidence-based integrative psychotherapy developed by Jeffrey Young (1999) to treat chronic emotional and interpersonal difficulties, especially personality disorders. It focuses on identifying and changing deeply rooted schemas and maladaptive coping styles. An additional therapeutic goal of schema therapy is to promote strengthening the individual's capacity to recognize and meet their core emotional needs, while reducing reliance on maladaptive coping modes.

Core Concepts:

- *Early Maladaptive Schemas:* Negative schemas formed when a child's emotional needs remain unmet. They are understandable given the circumstances when they were formed, but later in life they create problems with adaptation, hence they are called "maladaptive". Schemas are trait-like cognitive-emotional structures that influence how individuals perceive themselves and others.
- *Maladaptive Coping Styles:* Automatic responses triggered by schema activation, typically reflecting underlying fight, flight, or freeze mechanisms. Traditionally defined as surrender, overcompensation, and avoidance, coping styles have been refined into: resignation (believing the schema is true), inversion (believing the opposite of the schema is true), and avoidance (disengaging from schema-related thoughts and feelings).
- *Schema Modes:* Schema modes are dynamic, state-like patterns of thoughts, emotions, and behaviors based on specific schemas triggered by specific situations. They reflect temporary combinations of schemas and coping styles and can shift rapidly.

Definition of the schema modes used in the models:

- *Healthy Adult:* When this schema mode is active, people take on adult roles such as responsible working, parenting, and making decisions. They responsibly meet their own emotional needs and consider the needs of others.
- *Vulnerable Child:* When this schema mode is active, people feel intense emotional pain and fear of abandonment or neglect. They might feel sad, vulnerable, hopeless, or needy. They can appear fragile and desperately need someone to take care of them.
- *Impulsive Child:* When this schema mode is active, people act selfishly or uncontrollably on their desires and impulses. When the underlying needs are not immediately met, they might feel intense anger, frustration, and impatience.
- *Avoidant Protector:* When this mode is active, people try to protect themselves by avoiding situations that could trigger painful or vulnerable emotions.
- *Self-aggrandizer:* When this mode is active, people act self-centeredly to get their way, disregarding others' feelings due to a sense of superiority, entitlement, or specialness. They suppress feelings of insecurity.
- *Detached Self-Soother:* When this mode is active, people engage in compulsive or excessive activities, such as substance abuse, working, sleeping, using the internet, chatting, exercising, or sexual activity, to soothe their painful emotions.

Fig. 1. Fundamental Concepts of Schema Therapy and Core Schema Modes for the Mechanism of Change in Personality Disorders. Note. These definitions are based on the conceptualizations proposed by Armtz et al., 2021; Mamali (in press); Young et al., 2003.

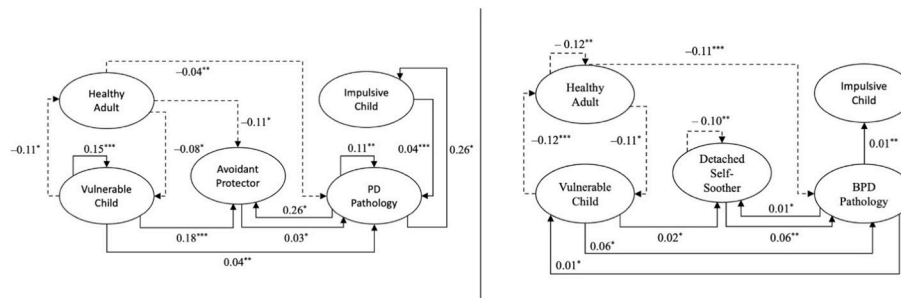


Fig. 2. Comparison of the time-lagged relationships between schema modes and borderline personality disorder symptoms over time: Yakın et al., 2020 (left panel) vs. current findings (right panel). Note. Arrows represents significant time-lagged associations. Parameter values are displayed next to respective arrows with the significance level (*p < .05; **p < .01; ***p < .001). Significant positive associations are displayed by continuous arrows, whereas negative associations are displayed by dashed arrows.

DSM-III and the Social and Occupational Functioning Assessment Scale (SOFAS) were used to evaluate participants' overall level of functioning. Participants were rated by an independent blinded assessor on a scale from 0 to 100 after taking a semi-structured interview considering their global functioning and their social and occupational/academic performance separately (Goldman et al., 2006).

2.3. Statistical analysis

We used Granger causality tests to investigate whether changes in the modes precede or follow changes in BPD-severity, and respectively functioning, using (generalized) linear mixed models (Granger, 1969; Rameckers et al., 2024). Granger causality tests whether one time series predicts another, while controlling for general time effects and prior levels of the outcome variable. Unlike experimental causality, it reflects a time-based form of inference. This is generally seen as a relatively weaker causality test than experimental tests, but the relatively best option when direct manipulation of the hypothesized cause is not possible. Similarly, understanding mechanisms of change in therapy requires establishing the temporal order and direction of influence between variables (Kazdin, 2009). Time-reversed Granger causality has been proposed as a useful method to test bidirectional relationships and reduce false causal inferences (Chvosteková et al., 2021). Accordingly, measurements from baseline to 24 months are used to predict

subsequent measurements of the same variables from 6 to 36 months. Our study tested whether changes in schema modes precede changes in BPD severity, consistent with the theoretical hypothesis that changes in modes drive psychopathological change. This does not imply that changes in BPD symptoms always lead to changes in schema modes. For example, situational factors may temporarily reduce BPD symptoms without altering underlying schema modes, meaning the reverse temporal effect would not hold. Therefore, we have also tested reverse temporal relationships for a complete test of the hypotheses.

First, the model predicting PD-severity as derived in Yakın et al. (2020) was tested. In that earlier study, which was based on data from a clinical trial investigating the treatment of personality disorders other than BPD, we developed a model based on a limited number of schema modes. This model identified schema modes as mechanisms of change in PD severity independently of the specific treatment approach. The primary aim of the current study was to replicate this model in individuals diagnosed with BPD. Accordingly, we restricted our analyses to the schema modes included in the original model. However, the ADP-IV and the Avoidant Protector mode tested in Yakın et al. were not included in the study by Armtz et al. (2022). In the current model, instead of the ADP-IV as an index of PD-severity, the BPDSI-IV total score was used as an index of BPD Severity. Additionally, in the place of the Avoidant protector mode, two alternative protector modes belonging to the avoidant coping modes (Armtz et al., 2021) and which might serve a

similar function as the Avoidant Protector mode from Yakin et al. were included in the multivariate model: the Detached Protector mode and the Detached Self-Soother mode. As the Detached Protector showed no significant effects, while the Detached Self-Soother did, we excluded the former to simplify the model and improve clarity without loss of information.

For the BPDSI-IV as a dependent variable, a similar model was employed as in the effectiveness tests of the RCT (Arntz et al., 2022): given the skewed distribution of the BPDSI-IV scores, a gamma distribution with loglink was chosen, the random part an intercept for participant (which takes out the interindividual variance in the BPDSI-IV, hence equivalent to person-centering of the dependent variable) and a random effect of time for cohort by site to control for site and cohort dependent effects of time (similar as in the RCT). In the fixed part, the following covariates were entered as control variables: the previous BPDSI-IV assessment (control for autocorrelation), time (as in the RCT a linear effect), treatment (the two group-ST types and the specialized Treatment as Usual (TAU)), and the interaction between treatment and time to control for the differences between the 3 treatments in development over time. The hypothesized predictors were person-centered to ensure that within-person changes were accounted for in the analysis and not confounded by between-person variance. All predictors were entered simultaneously into the equation to control for the variance explained by each other. For degrees of freedom in the fixed part tests, the Satterthwaite's approximation was used. Effect sizes for the fixed part were calculated as $r = \sqrt{(t^2/(t^2 + df))}$ (Rosenthal & Rosnow, 1991). The r-squared value represents the proportion of variance explained in the fixed part of the model.

Next, two models predicting GAF & SOFAS in Yakin et al. (2020) were tested. Similar to the previous study, the person-centered GAF, respectively SOFAS scores were regressed on the person-centered BPDSI-IV total score and the residuals were used for further analysis. Thus, GAF and SOFAS scores were corrected for effects of BPD-severity. Next, the models with person-centered HA and Self-Aggrandizer mode scores as predictors were built, first without the Self-Aggrandizer x treatment interaction, later with this interaction (as in Yakin et al. the effect of the Self-Aggrandizer mode was only found with ST). Time, treatment, time x treatment, and the previous person-centered GAF, respectively SOFAS scores, were added as covariates. As dependent variables were normally distributed, a linear mixed effect model was used, with time for cohort by site to control for site and cohort dependent effects of time (similar as in the RCT). Note that as the dependent variables were already person-centered, no random intercept for person had to be, and could be, added. The predictors were entered simultaneously. For degrees of freedom in the fixed part tests, the Satterthwaite's approximation was used. Effect sizes for the fixed part were calculated as $r = \sqrt{(t^2/(t^2 + df))}$ (Rosenthal & Rosnow, 1991).

For the reverse causality tests of each model, linear mixed effects models were used as dependent variables since they showed an approximately normal distribution. The random part contained an intercept for participant (which takes out the interindividual variance in the dependent variable, hence equivalent to person-centering of the dependent variable), and a random effect of time for cohort by site to control for site and cohort dependent effects of time. Each schema mode was investigated as dependent variable, predicted by the other modes of the model and the BPDSI-IV of the previous assessment, all person-centered, while controlling for time, treatment, and treatment by time. All the analyses were conducted with IBM SPSS statistics (version 29) for Windows.

3. Results

3.1. Replication model

Table 1 presents the results of the replication model. To enhance interpretation, the time effects are given per treatment condition. Note that the time x treatment interaction was significant, $F(2, 744) = 11.087, p < .001$, with superior effects in IGST compared to the other two conditions, as in the main study (Arntz et al., 2022). The results indicate independent significant effects of the HA, the VC and Detached Self-Soother modes, with small effect sizes, but a NS effect of the Impulsive Child mode. Between- and within-person correlation matrices are reported in the supplementary data.

Reverse Granger causality tests are summarized in Table 2. The HA mode was significantly predicted by the VC mode, similar to Yakin et al. (2020): a previous lower level of this mode predicted later increases in the HA mode. Remarkably, the autocorrelation was negative, implying that relatively low levels of the HA mode were followed by relatively high levels the next assessment. The BPDSI-IV of the previous assessment did not significantly relate to the HA mode, supporting a unidirectional causal interpretation. By contrast, the VC was significantly predicted by the previous BPDSI-IV level, indicating a recursive causal relation between the VC mode and BPDSI-IV, with similar effect sizes for both directions. In addition, the previous HA mode level predicted the VC mode, replicating the Yakin et al., finding that increases in the HA are followed by decreases in the VC mode. Similarly, the Detached Self-Soother was predicted by the previous BPDSI-IV, indicating a recursive relationship. In addition, the Detached Self-Soother was predicted by the previous VC assessment, and by itself at the previous assessment, though this autocorrelation was negative: high previous levels of this mode predicted relatively lower levels the next assessment. Lastly, the Impulsive Child mode was predicted by the previous BPDSI-IV assessment, indicating that it was rather the change in BPD-severity causing changes in this mode, than the opposite.

Table 1
Results of the replication model testing prediction of severity of BPD by schema modes.

Fixed effect	B	SE	t	df	p	r	r ²
Healthy Adult	-.107	.032	-3.369	1127	<.001	.10	.010
Vulnerable Child	.055	.026	2.161	1111	.031	.06	.004
Impulsive Child	-.008	.027	-.307	1114	.76	.01	<.001
Detached Self-Soother	.062	.020	3.048	1111	.002	.09	.008
Control variables							
BPDSI-IV	-.002	.002	-.675	1123	.50	.02	<.001
Time in S-TAU	-.096	.015	-6.402	55	<.001	.65	.423
Time in PGST	-.121	.018	-6.616	91	<.001	.57	.325
Time in IGST	-.169	.018	-9.371	84	<.001	.71	.504
Random effect (variance)		Estimate	SE	z		p	
Time (for cohort within site)		.0036	.0013	2.845		.004	
Intercept (for participant)		.258	.022	11.624		<.001	

Note. Effect size r represents the effect size in the fixed part and is calculated as $r = \sqrt{(t^2/(t^2 + df))}$, its squared value r² represents the variance explained. Degrees of freedom are based on Satterthwaite's approximations and rounded to integers.

S-TAU =Specialized Treatment as Usual, PGST = Predominantly Group Schema Therapy, IGST = Individual-Group Schema Therapy combined.

Table 2
Results of reversed Granger causality tests.

Healthy Adult							
Fixed effect	B	SE	t	df	p	r	r ²
BPDSI-IV	-.003	.003	-1.002	1096	.32	.03	.001
Vulnerable Child	-.119	.030	-3.988	1088	<.001	.12	.014
Impulsive Child	-.038	.031	-1.253	1084	.21	.04	.002
Detached Self-Soother	.021	.024	.877	1086	.38	.03	.001
Control variables							
Healthy Adult	-.115	.037	-3.144	1104	.002	.09	.008
Time in S-TAU	.039	.015	2.548	70	.013	.29	.084
Time in PGST	.073	.019	3.839	104	<.001	.35	.122
Time in IGST	.089	.019	4.770	91	<.001	.45	.203
Random effect (variance)		Estimate	SE	Z	p		
Time (for cohort within site)		.0026	.0011	2.292	.02		
Intercept (for participant)		.439	.037	11.865	<.001		
Vulnerable Child							
Fixed effect	B	SE	t	df	p	r	r ²
BPDSI-IV	.009	.004	2.317	1090	.021	.07	.005
Healthy Adult	-.106	.053	-2.003	1099	.045	.06	.004
Impulsive Child	.047	.044	1.070	1077	.29	.03	.001
Detached Self-Soother	.010	.034	.300	1080	.76	.01	<.001
Control variables							
Vulnerable Child	-.017	.043	-.385	1083	.70	.01	<.001
Time in S-TAU	-.093	.023	-4.065	69	<.001	.44	.194
Time in PGST	-.123	.028	-4.316	108	<.001	.38	.144
Time in IGST	-.189	.028	-6.777	96	<.001	.57	.325
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.0068	.0026	2.616	.009		
Intercept (for participant)		.883	.075	11.773	<.001		
Detached Self-Soother							
Fixed effect	B	SE	t	df	p	r	r ²
BPDSI-IV	.008	.004	2.152	1111	.032	.06	.004
Healthy Adult	-.033	.052	-.642	1122	.52	.02	<.001
Impulsive Child	.036	.043	.836	1096	.40	.03	.001
Vulnerable Child	.099	.042	2.341	1102	.019	.07	.005
Control variables							
Detached Self- Soother	-.104	.033	-3.141	1099	.002	.09	.008
Time in S-TAU	-.062	.021	-3.000	72	.004	.33	.109
Time in PGST	-.077	.026	-2.971	107	.004	.28	.078
Time in IGST	-.096	.025	-3.745	93	<.001	.36	.130
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.005	.002	2.244	.03		
Intercept (for participant)		.549	.050	10.980	<.001		
Impulsive Child							
Fixed effect	B	SE	t	df	p	r	r ²
BPDSI-IV	.007	.003	2.608	1088	.009	.08	.006
Healthy Adult	-.036	.037	-.979	1094	.33	.03	.001
Vulnerable Child	-.023	.030	-.750	1081	.45	.02	<.001
Detached Self-Soother	.018	.024	.777	1081	.44	.02	<.001
Control variables							
Impulsive Child	-.004	.031	-.144	1079	.89	.004	<.001
Time in S-TAU	-.070	.014	-5.095	99	<.001	.46	.212
Time in PGST	-.091	.017	-5.211	117	<.001	.43	.185
Time in IGST	-.108	.017	-6.359	97	<.001	.54	.292
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.0012	.0007	1.627	.10		
Intercept (for participant)		.598	.049	12.204	<.001		

Note. Effect size r represents the effect size in the fixed part and is calculated as $r = \sqrt{(t^2 / (t^2 + df))}$, its squared value r² represents the variance explained. Degrees of freedom are based on Satterthwaite's approximations and rounded to integers.

S-TAU =Specialized Treatment as Usual, PGST = Predominantly Group Schema Therapy.

IGST = Individual-Group Schema Therapy combined.

In sum, while BPD severity was predicted by previous changes in the VC, the HA, and the Detached Self-Soother mode, we did not find support for such an effect by the Impulsive Child mode. While unidirectionality was supported for the HA, which also influenced the VC over

time, the VC showed a recursive causal relationship over time with the index of BPD-severity. Despite differences with the findings by Yakın et al. (2020), the central relationships were replicated: the effects of HA and VC on the index of personality disorder severity, direct, and (for the

VC mode) indirect via an avoidant coping mode, and the mutual influences over time of the HA and VC on each other. Fig. 2 shows the relationships over time found by Yakin et al. in the left panel, and the findings in the present data in the right panel.

3.2. Interaction with condition

Because the IGST treatment arm was the most effective and resembles the (individual) ST in Bamelis et al. (2014) the most, it was taken as reference category in the tests of the interactions of effects of the four schema modes with treatment (see Table 3). With an uncorrected p-level of .05, two interactions were significant: compared to IGST, S-TAU showed a diminished effect of the HA on the next BPDSI score (NS within the S-TAU); while compared to IGST, PGST showed a positive effect of the Impulsive Child mode on the next BPDSI. However, Bonferroni correction for the eight explorative interaction tests lead to no significant findings at all (significance level $.05/8 = .00625$). Note that in the reference, IGST, the Impulsive Child mode had a negative (and significant) beta, in Table 3 shown as the main effect of that mode. This suggests an effect that is difficult to interpret as decreases of the Impulsive Child mode are followed by increases in BPD-severity.

In conclusion, interaction with treatment type tests suggested that the effect of changes in the HA mode were most profound in the IGST condition, in the expected direction, while the Impulsive Child mode showed an unexpected and difficult to interpret negative effect on the next BPDSI scores in the IGST condition, different from its effect in PGST and (marginally) in S-TAU. However, none of the interaction effects survives a correction for multiple testing.

3.3. Replication GAF & SOFAS prediction model

3.3.1. GAF prediction model

Table 4 presents the results of the replication model. To enhance interpretation, the time effects are given per treatment condition. Note that the time \times treatment interaction was not significant, $F(2, 726) = 1.899, p = .15$, despite the differences suggested in Table 4 (note that time effects were already partially taken out the GAF scores because these are controlled for the BPDSI). The results indicate independent significant effects of HA and Self-Aggrandizer (now as a main effect),

with small effect sizes, in the same direction as found by Yakin et al. (2020). Adding the interaction between treatment condition and Self-Aggrandizer resulted in a nonsignificant overall test of the interaction ($F(2, 1460) = .09, p = .91$), and nonsignificant differences between S-TAU and PGST on the one hand, and IGST on the other hand, in the effect of the Self-Aggrandizer mode on the GAF, see Table 4. Subsequently adding the HA \times Treatment interaction showed nonsignificant effects of this interaction, $F(2, 1462) = .80, p = .45$.

Test of reversed Granger causality yielded evidence that changes in GAF scores influence the HA, but not the Self-Aggrandizer over time, replicating Yakin et al. (2020), Table 5. Also replicating Yakin et al., the HA did significantly influence the next Self-Aggrandizer assessment. However, in contrast to Yakin et al.'s findings, the Self-Aggrandizer did not influence later HA scores.

3.3.2. SOFAS prediction model

Table 6 presents the results of the replication analysis of the SOFAS. The time \times treatment interaction was not significant, $F(2, 771) = 1.627, p = .20$, despite the differences suggested in the table. Without the treatment condition by Self-Aggrandizer interaction, the model replicated the time-lagged effects of the HA and the Self-Aggrandizer mode on the SOFAS. This interaction turned out to be NS, $F(2, 1463) = .21, p = .81$. The contrasts of IGST vs S-TAU and vs PGST of this interaction also failed to reach significance, Table 6. Similarly, when both interactions of HA and Self-Aggrandizer were added to the model, the interactions were not significant ($F(2, 1465) = 1.001, p = .37; F(2, 1461) = .22, p = .80$), see Table 6 for details.

Test of reversed Granger causality showed that changes in SOFAS scores influence the HA, but not the Self-Aggrandizer over time, replicating Yakin et al. (2020), see Table 7. Also replicating Yakin et al., the HA did significantly influence the next Self-Aggrandizer assessment. However, in contrast to Yakin et al.'s findings, the Self-Aggrandizer did not significantly influence later HA scores.

To summarize, most of the findings by Yakin et al. (2020) were replicated: HA and Self-Aggrandizer modes predicted later GAF and SOFAS, with reverse prediction of the HA mode by both GAF and SOFAS. Moreover, the HA mode predicted later Self-Aggrandizer, as in Yakin et al. In contrast to the previous findings, the effects of the Self-Aggrandizer were not moderated by treatment and the significant

Table 3

Results of the tests of treatment interactions with four schema modes of the replication model in the prediction of severity of BPD.

Fixed effect	B	SE	t	df	p	r	r ²
Treatment-Predictor interaction							
S-TAU x Healthy Adult	.178	.076	2.342	1112	.019	.07	.005
S-TAU x Vulnerable Child	.014	.057	.245	1102	.81	<.01	<.001
S-TAU x Impulsive Child	.117	.062	1.873	1122	.06	.06	.004
S-TAU x Detached Self-Soother	-.075	.049	-1.533	1118	.13	.05	.003
PGST x Healthy Adult	.151	.088	1.719	1104	.09	.05	.003
PGST x Vulnerable Child	.022	.065	.337	1108	.74	.01	<.001
PGST x Impulsive Child	.181	.070	2.574	1128	.010	.08	.006
PGST x Detached Self-Soother	-.044	.056	-.78	1108	.43	.02	<.001
Predictor within reference (IGST)							
Healthy Adult	-.230	.060	-3.805	1096	<.001	.11	.012
Vulnerable Child	.043	.047	.922	1100	.36	.03	.001
Impulsive Child	-.108	.048	-2.246	1108	.025	.07	.005
Detached Self-Soother	.116	.038	3.016	1109	.003	.09	.008
Control variables							
BPDSI-IV (previous)	-.001	.002	-.528	1118	.60	.02	<.001
Time in S-TAU	-.100	.015	-6.526	58	<.001	.65	.423
Time in PGST	-.108	.020	-5.480	118	<.001	.45	.203
Time in IGST	-.172	.019	-9.015	103	<.001	.66	.436
Random effect (variance)							
	Estimate	SE	z		p		
Time (for cohort within site)	.004	.0013	2.827		.005		
Intercept (for participant)	.259	.022	11.621		<.001		

Note. Reference treatment is IGST, individual-group schema therapy combined.

S-TAU = specialized treatment as usual, PGST = predominantly group schema therapy.

r = effect size of the fixed part ($r = \sqrt{t^2/(t^2 + df)}$).

Table 4
Results of the replication model testing prediction of GAF by schema modes.

Main effects only							
Fixed effect	B	SE	t	df	p	r	r ²
Healthy Adult	1.112	.453	2.454	1467	.014	.06	.004
Self-Aggrandizer	-1.498	.490	-3.055	1459	.002	.08	.006
Control variables							
GAF (previous)	-.038	.025	-1.543	1471	.12	.04	.002
Time in S-TAU	.102	.167	.608	267	.544	.04	.002
Time in PGST	.330	.223	1.480	324	.140	.08	.006
Time in IGST	.579	.217	2.669	273	.008	.16	.026
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.077	.063	1.222	.22		
With Self-Aggrandizer x treatment interaction (ISGT as reference)							
Fixed effect	B	SE	t	df	p	r	r ²
Healthy Adult	1.120	.454	2.476	1465	.014	.06	.004
Self-Aggrandizer	-1.711	.893	-1.916	1452	.056	.05	.003
S-TAU x Self-Aggrandizer	.438	1.134	.386	1462	.70	.01	<.001
PGST x Self-Aggrandizer	.080	1.264	.063	1455	.95	<.01	<.001
Control variables							
GAF (previous)	-.038	.025	-1.531	1469	.13	.04	.002
Time in S-TAU	.115	.170	.676	286	.50	.04	.002
Time in PGST	.319	.229	1.398	353	.16	.07	.005
Time in IGST	.560	.225	2.486	313	.013	.14	.020
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.0028	.0012	2.365	.02		
With Self-Aggrandizer & Healthy Adult x treatment interactions (ISGT as reference)							
Fixed effect	B	SE	t	df	p	r	r ²
Healthy Adult	1.003	.866	1.158	1461	.25	.03	.001
Self-Aggrandizer	-1.732	.902	-1.919	1448	.055	.05	.003
S-TAU x Healthy Adult	-.291	1.050	-.277	1461	.78	<.01	<.001
PGST x Healthy Adult	1.059	1.222	.867	1459	.39	.02	<.001
S-TAU x Self-Aggrandizer	.329	1.160	.284	1458	.78	<.01	<.001
PGST x Self-Aggrandizer	.340	1.292	.263	1452	.79	<.01	<.001
Control variables							
GAF (previous)	-.038	.025	-1.523	1467	.13	.04	.002
Time in S-TAU	.145	.173	.839	314	.40	.05	.003
Time in PGST	.218	.242	.901	430	.37	.04	.002
Time in IGST	.570	.231	2.467	346	.014	.13	.017
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.071	.061	1.164	.24		

Note. Main effects of intercept and treatment condition are not shown as these are not relevant. Effect size r represents the effect size in the fixed part and is calculated as $r = \sqrt{(t^2 / (t^2 + df))}$, its squared value r² represents the variance explained.

Degrees of freedom are based on Satterthwaite's approximations and rounded to integers.

S-TAU = Specialized Treatment as Usual, PGST = Predominantly Group Schema Therapy.

IGST = Individual-Group Schema Therapy combined.

autocorrelations in Yakin et al. were not significant in the present data. Figs. 3 and 4 shows the relationships over time found by Yakin et al. in the left panel, and the findings in the present data in the right panel.

4. Discussion

Previous research has demonstrated that specific schema modes function as mechanisms of change in the treatment of six different personality disorders, excluding BPD (Yakin et al., 2020). This study aimed to replicate these findings in a sample of BPD patients, employing Granger causality tests. We investigated whether mechanisms similar to those as previously found could be identified in the change of severity of BPD pathology using data from a large international RCT (Arntz et al., 2022). The most important aspects were replicated, especially the centrality of the HA and the VC in changes in the severity of BPD symptoms together with the Detached Self-Soother. Unlike findings by Yakin et al., the relationship between the VC and the BPD severity was reciprocal, and the Impulsive Child did not predict BPD severity at the subsequent measurement point but was only influenced by previous levels of BPD

severity. In terms of general social and occupational functioning, the SA and the HA predicting functioning at the subsequent measurement point was replicated. While the HA demonstrated a reciprocal relationship with functioning, no reciprocity was observed for Self-Aggrandizer.

A key finding from Yakin et al. (2020) was the reciprocal time-lagged relationship between the VC and the HA, with changes in both preceding a change in personality pathology. The influence of VC and HA on PD-pathology was unidirectional and observed across the three treatments. In terms of the criteria to establish mechanisms of change (Kazdin, 2007, 2009) the unidirectionality meets the timeline criterion as indicator of a mechanism of change, and the consistency of effects across three different therapeutic approaches strengthens the evidence for the consistency criterion as proposed to identify mechanisms of change. The current study replicated the finding that the HA and the VC modes are central in the model. These modes not only predicted borderline pathology but also demonstrated a reciprocal relationship and this effect was observed across all treatment conditions. Replicating these findings are essential for advancing evidence on consistency, an important precondition to establish mechanism of change. Interestingly,

Table 5
Results of reversed Granger causality tests of GAF.

Healthy Adult							
Fixed effect	B	SE	t	df	p	r	r ²
GAF	.005	.002	2.710	1122	.007	.08	.006
Self-Aggrandizer	-.051	.034	-1.491	1091	.14	.05	.003
Control variables							
Healthy Adult	-.027	.032	-.845	1103	.40	.03	.001
Time in S-TAU	.046	.015	3.010	63	.004	.35	.122
Time in PGST	.078	.019	4.001	103	<.001	.37	.137
Time in IGST	.108	.019	5.694	91	<.001	.51	.260
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.0028	.0012	2.365	.02		
Intercept (for participant)		.431	.036	11.972	<.001		
Self-Aggrandizer							
Fixed effect	B	SE	t	df	p	r	r ²
GAF	.001	.002	.578	1070	.56	.02	<.001
Healthy Adult	-.059	.029	-2.036	1087	.042	.06	.004
Control variables							
Self-Aggrandizer	.020	.031	.636	1084	.53	.02	<.001
Time in S-TAU	-.065	.012	-5.561	100	<.001	.49	.240
Time in PGST	-.070	.015	-4.553	111	<.001	.40	.160
Time in IGST	-.075	.015	-5.129	90	<.001	.48	.230
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.0006	.0005	1.177	.24		
Intercept (for participant)		.523	.043	12.163	<.001		

Note. Main effects of intercept and treatment condition are not shown as these are not relevant. Effect size r represents the effect size in the fixed part and is calculated as $r = \sqrt{(t^2/(t^2 + df))}$, its squared value r² represents the variance explained. Degrees of freedom are based on Satterthwaite’s approximations and rounded to integers. S-TAU = Specialized Treatment as Usual. PGST = Predominantly Group Schema Therapy. IGST = Individual-Group Schema Therapy combined.

in the present study the VC was less central (i.e., had smaller effect sizes) compared to the original findings and displayed a reciprocal relationship with BPD severity. In fact, BPD severity predicted all the modes except the HA in the subsequent measurement points. Thus, the role of the HA seems more central in the current study. Also, the Impulsive Child did not predict changes in BPD severity at the subsequent measurement point. The Impulsive Child is characterized by the pursuit of immediate, hedonic gratification without consideration of long-term consequences (Arntz & Jacob, 2013). Given the centrality of impulsive behaviors in the BPD pathology, it is noteworthy that the Impulsive Child did not play a central role. However, unlike the original model, the replication model includes the Detached Self-Soother. We considered replacing the Avoidant Protector Mode with the Detached Protector and the Detached Self-Soother as it was not measured in the current study. Ultimately, we chose to retain the Detached Self-Soother mode due to its significant contribution to the model. The Detached Protector and the Detached Self-Soother serve similar functions; both aimed at maintaining a safe distance from experiencing emotions and needs and from connecting to other people. However, the Detached Self-Soother might be especially relevant for BPD pathology, as it represents the highly dysfunctional active soothing behaviors in BPD, including substance use and other addictive actions. Additionally, these behavioral issues are more easily captured by self-report measures. Detached Self-Soother is a mode that is also involved in engaging impulsive activities like the Impulsive Child; however, the function is different: The Detached Self-Soother soothes difficult feelings, whereas the Impulsive Child Mode seeks immediate need gratification. It is possible that the Impulsive Child’s role in the model was partly explained by the Detached Self-Soother mode, and the overlap in impulsivity. Furthermore, the Detached Protector was not included in the model due to its high correlation with the Vulnerable Child mode in this dataset ($r = .78$). Given this strong collinearity, including both variables simultaneously is problematic (see supplemental analyses) and future research is needed

to more clearly delineate the specific contribution of the Detached Protector.

In terms of the interactions of the treatment conditions (i.e., predominantly group ST combined individual and group ST, and treatment as usual), an increase in the HA predicted a subsequent decrease in BPD pathology at the subsequent measurement point in IGST, which was the superior treatment, while these effects were weaker in the other two treatments. It is tempting to conclude that in IGST the role of the HA mode in recovery is specific for IGST; however, the interaction effect did not reach the Bonferroni corrected significance level, hence such a conclusion would be premature. In addition, a decrease in the Impulsive Child predicted an increase in BPD pathology at the subsequent measurement point in IGST, differing from PGST, a finding which we cannot explain. Nevertheless, interpreting these effects should be done with caution as they became non-significant following the application of the Bonferroni correction. In sum, the model representing the mechanisms of change did not exhibit significant variation across the different treatment conditions. This is important in terms of providing evidence to ensure the consistency of the findings regarding the schema modes as a common mechanism of change across various treatments.

In the second and third models, independent from the treatment condition, the Self-Aggrandizer also emerged as the strongest predictor of global, social and occupational functioning replicating the previous findings (Yakın et al., 2020). Similarly, the HA was identified as an important predictor of global, social and occupational functioning at the subsequent measurement point, replicating the findings of Yakın et al. In contrast, the connection between the HA and the Self-Aggrandizer was unidirectional in the current study meaning that having a stronger HA predicts lower levels of the Self-Aggrandizer at the subsequent measurement point but not vice versa. The strong predictive role of the Self-Aggrandizer in both models is noteworthy as the literature on the link between narcissistic traits and functioning is complex. Some researchers postulate that vulnerable narcissism is mostly associated with

Table 6
Results of the replication model testing prediction of SOFAS by schema modes.

Main effects only							
Fixed effect	B	SE	t	df	p	r	r ²
Healthy Adult	1.001	.444	2.254	1469	.024	.06	.004
Self-Aggrandizer	-1.066	.487	-2.191	1464	.029	.06	.004
Control variables							
SOFAS (previous)	-.038	.025	-1.508	1474	.13	.04	.002
Time in S-TAU	.106	.166	.637	294	.52	.04	.002
Time in PGST	.549	.222	2.472	356	.014	.13	.017
Time in IGST	.356	.216	1.634	299	.10	.09	.008
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.077	.059	1.305	.19		
With Self-Aggrandizer x treatment interaction (IGST as reference)							
Fixed effect	B	SE	t	df	p	r	r ²
Healthy Adult	1.012	.445	2.275	1468	.023	.06	.004
Self-Aggrandizer	-1.476	.889	-1.660	1458	.097	.04	.002
S-TAU x Self-Aggrandizer	.424	1.127	.376	1466	.71	.01	<.001
PGST x Self-Aggrandizer	.808	1.258	.642	1460	.52	.02	<.001
Control variables							
SOFAS (previous)	-.039	.025	-1.533	1472	.13	.04	.002
Time in S-TAU	.107	.169	.631	314	.53	.04	.002
Time in PGST	.574	.228	2.519	389	.01	.13	.017
Time in IGST	.321	.224	1.429	342	.15	.08	.006
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.077	.059	1.305	.19		
With Self-Aggrandizer & Healthy Adult x treatment interactions (IGST as reference)							
Fixed effect	B	SE	T	df	p	r	r ²
Healthy Adult	1.775	.853	2.080	1464	.38	.05	.003
Self-Aggrandizer	-1.339	.898	-1.491	1454	.14	.04	.002
S-TAU x Healthy Adult	-1.365	1.043	-1.309	1464	.19	.03	.001
PGST x Healthy Adult	-.353	1.215	.291	1463	.77	.01	<.001
S-TAU x Self-Aggrandizer	.098	1.152	.085	1462	.93	<.01	<.001
PGST x Self-Aggrandizer	.774	1.286	.602	1457	.55	.02	<.001
Control variables							
SOFAS (previous)	-.039	.025	-1.549	1469	.12	.04	.002
Time in S-TAU	.154	.172	.892	340	.37	.05	.003
Time in PGST	.531	.242	2.196	471	.029	.10	.010
Time in IGST	.264	.230	1.147	379	.25	.06	.004
Random effect (variance)		Estimate	SE	z	p		
Time (for cohort within site)		.073	.058	1.259	.21		

Note. Main effects of intercept and treatment condition are not shown as these are not relevant. Effect size r represents the effect size in the fixed part and is calculated as $r = \sqrt{(t^2 / (t^2 + df))}$, its squared value r² represents the variance explained.

Degrees of freedom are based on Satterthwaite's approximations and rounded to integers.

S-TAU = Specialized Treatment as Usual.

PGST = Predominantly Group Schema Therapy.

IGST = Individual-Group Schema Therapy combined.

poorer functioning while grandiose narcissism has adaptive components (Barry et al., 2015; Dashineau et al., 2019; Pincus et al., 2014). Salgó et al. (2021) found that schema modes related to externalizing pathology (i.e., Impulsive Child, Enraged Child, Angry Child, Self-Aggrandizer, Bully and Attack) are negatively associated with self-blame and self-judgment and positively associated with self-kindness. However, while elevated levels of self-compassion and reduced levels of self-criticism are associated with a more adaptive outlook, Salgó et al. also discussed that individuals with schema modes related to externalizing pathology cannot follow-up on the social consequences of their behaviors because of these traits. Therefore, they are unable to use self-reflection to make use of secondary emotions like shame and guilt in a socially adaptive way. Thus, the effects of the Self-Aggrandizer mode on functioning can be linked to lack of insight and self-reflection (i.e., the presence of the HA). Additionally, it is important to note that there is evidence suggesting that insight and clarification function as mechanisms of change in ST (Tschacher et al., 2012). Therefore, their absence may hinder therapeutic progress and block optimal functioning.

Furthermore, in a forensic setting, Keulen-de Vos et al. (2017) found that the Self-Aggrandizer mode was not associated with the interpersonal facets of psychopathy. This suggests that the impact of the Self-Aggrandizer on functioning may be widely influenced by the degree to which environmental factors are controlled and the extent to which individuals develop reflective insight into social dynamics.

There are several clinical implications of the present findings. First, the findings suggest that it might be beneficial if treatments focus on the vulnerable parts of the patient (in ST terms the VC mode) and help the patient to recover from issues that underlie these. Second, the findings indicate that next to a focus on vulnerability, therapy might focus on strengthening the adaptive and healthy parts (the HA mode). Moreover, avoidant (in particular dysfunctional self-soothing) coping might block the effects of addressing the vulnerable issues of the patient. In addition, for healthy functioning the results suggest that self-aggrandizer issues might need to be addressed. Thus, rather than emphasizing a singular treatment focus, the findings point towards that multiple foci may be more effective, at least in the treatment of complex personality

Table 7
Results of reversed Granger causality tests of SOFAS.

Healthy Adult								
Fixed effect	B	SE	t	df	p	r	r ²	
SOFAS	.004	.002	1.994	1126	.046	.06	.004	
Self-Aggrandizer	-.055	.034	-1.628	1093	.10	.05	.003	
Control variables								
Healthy Adult	-.017	.031	-.547	1103	.59	.02	<.001	
Time in S-TAU	.048	.015	3.119	64	.003	.36	.130	
Time in PGST	.080	.019	4.096	104	<.001	.37	.137	
Time in IGST	.110	.019	5.786	91	<.001	.52	.270	
Random effect (variance)		Estimate	SE	z	P			
Time (for cohort within site)		.0028	.0012	2.372	.02			
Intercept (for participant)		.431	.037	11.649	<.001			
Self-Aggrandizer								
Fixed effect	B	SE	t	df	p	r	r ²	
SOFAS	.001	.002	.374	1060	.71	.01	<.001	
Healthy Adult	-.056	.029	-1.969	1089	.049	.06	.004	
Control variables								
Self-Aggrandizer	.019	.031	.623	1087	.53	.02	<.001	
Time in S-TAU	-.064	.012	-5.541	99	<.001	.49	.240	
Time in PGST	-.069	.015	-4.521	110	<.001	.40	.160	
Time in IGST	-.075	.015	-5.080	88	<.001	.48	.230	
Random effect (variance)		Estimate	SE	z	P			
Time (for cohort within site)		.0006	.0005	1.176	.24			
Intercept (for participant)		.523	.042	12.452	<.001			

Note. Main effects of intercept and treatment condition are not shown as these are not relevant. Effect size r represents the effect size in the fixed part and is calculated as $r = \sqrt{t^2 / (t^2 + df)}$, its squared value r² represents the variance explained. Degrees of freedom are based on Satterthwaite’s approximations and rounded to integers. S-TAU = Specialized Treatment as Usual. PGST = Predominantly Group Schema Therapy. IGST = Individual-Group Schema Therapy combined.

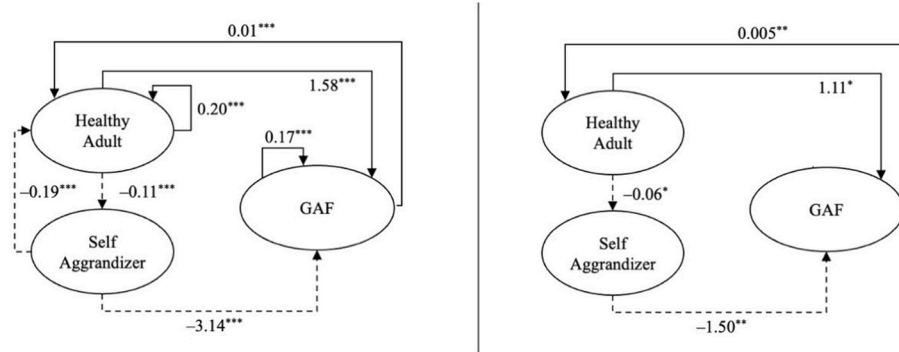


Fig. 3. Comparison of the time-lagged relationships between schema modes and general functioning over time: Yakın et al., 2020 (left panel) vs. current findings (right panel).

Note. Arrows represents significant time-lagged associations. Parameter values are displayed next to respective arrows with the significance level (*p < .05; **p < .01; ***p < .001). Significant positive associations are displayed by continuous arrows, whereas negative associations are displayed by dashed arrows. GAF: Global Assessment of Functioning Scale.

psychopathology. Although the findings suggest that addressing other schema modes that are thought to be central to BPD, such as the Punitive (Parent) mode and the Angry Child mode, is not important, such an interpretation would go too far. The findings do suggest however that if addressing such modes does not ultimately lead to changes in VC and the HA modes, the effects would be limited. Lastly, although the individual patient was the starting point of the analyses, results were pooled over participants, hence they represent general relationships. This implies that idiosyncratic processes are not addressed by the present results and that clinicians need to keep an eye on them.

4.1. Limitations

The findings of this replication study provide further evidence that schema modes act as a mechanism of change, not only in ST, but in psychological treatment of PDs in general. However, the study’s limitations should still be kept in mind when interpreting the results. Although we replicated a significant reciprocal relationship between the HA and the VC, we still cannot definitively determine whether interventions targeting the VC mode and respectively the HA mode, would result in subsequent changes in the HA mode and respectively the VC mode. Within-subject associations may be influenced by third variable confounds, even when the associations are assessed over time. While we

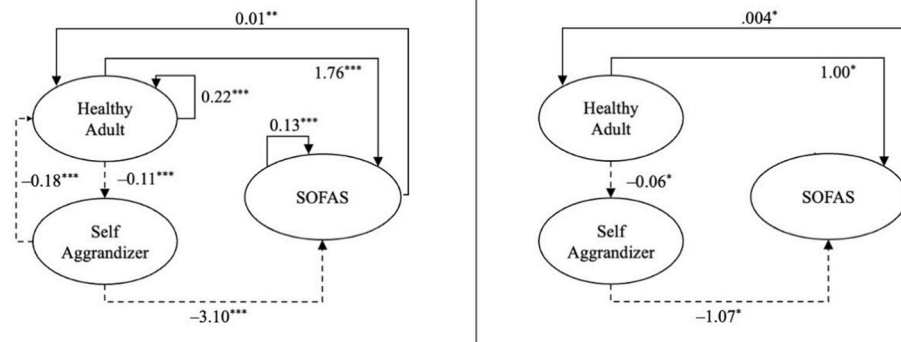


Fig. 4. Comparison of the time-lagged relationships between schema modes and social and occupational functioning over time: Yakın et al., 2020 (left panel) vs. current findings (right panel).

Note. Arrows represents significant time-lagged associations. Parameter values are displayed next to respective arrows with the significance level (* $p < .05$; ** $p < .01$; *** $p < .001$). Significant positive associations are displayed by continuous arrows, whereas negative associations are displayed by dashed arrows. SOFAS: Social and Occupational Assessment of Functioning Scale.

have identified mechanisms of change, these mechanisms still may manifest differently across individuals in terms of timing and the variability of the change process (Kazdin, 2009). Additionally, the data were collected using self-report measures, which might have their limitations as assessment of schema modes. It is important to note that the observed effect sizes were small, ranging from .06 to .10 across outcome measures (see Tables 1, 4 and 5). This likely reflects, in part, limitations in measurement. Fluctuations in schema modes were captured solely via self-report, which may only partially reflect the underlying dynamics between schema modes, PD severity, and functioning. In addition, the relatively long-time lags between assessments were planned to reliably capture changes in BPD severity but have probably reduced the effect sizes. However, whether larger effect sizes can realistically be expected in this field remains an open question. Therapeutic change in personality disorders may be better characterized by subtle, gradual shifts over time rather than strong, linear associations between specific constructs. Moreover, some of the relationships over time were reciprocal, which when Kazdin's criteria are used, preclude that the pertinent variable represents a mechanism of change. However, the criterion of unidirectionality might be too strict. In systems theory, reciprocal causality is an acknowledged form of causality. Thus, reductions in the VC mode might lead directly, or indirectly (through HA and Detached Self Soother), to reduced BPD-severity. This causal effect is not necessarily contradicted by reductions in BPD-severity having a causal effect on the VC mode. For example, a stronger HA mode might lead to reductions in emotional instability, identity problems, and interpersonal problems, which in turn leads to the VC mode being less often triggered. Lastly, there are several limitations related to the implementation of the clinical trial that are discussed in detail in Arntz et al. (2022).

5. Conclusions

We replicated the findings underlying the importance of the VC and the HA as mechanisms of change in treatment of personality disorders, using a sample with BPD psychopathology. Additionally, we replicated the finding that the Self Aggrandizer and the HA function as a mechanism of change in therapy in terms of improved general, social and occupational functioning. The present study's multicultural sample is particularly important, as it strengthens the consistency of the previous findings. While earlier research by Yakın et al. (2020) provided evidence for schema modes as a common mechanism of change, the current findings elevate this evidence across cultures and generalizes it to BPD, an important and severe personality disorder.

CRediT authorship contribution statement

Duygu Yakın: Writing – review & editing, Writing – original draft, Visualization, Methodology, Conceptualization. **Julia Uijtewaal:** Formal analysis. **Puk Plooi:** Formal analysis. **Gitta A. Jacob:** Data curation. **Christopher W. Lee:** Writing – review & editing, Data curation. **Odette Manon Brand-de Wilde:** Data curation. **Eva Fassbinder:** Writing – review & editing, Data curation. **R. Patrick Harper:** Data curation. **Anna Lavender:** Writing – review & editing, Data curation. **George Lockwood:** Writing – review & editing, Data curation. **Ioannis A. Malogiannis:** Data curation. **Florian A. Ruths:** Data curation. **Ida A. Shaw:** Data curation. **Gerhard Zarbock:** Data curation. **Joan M. Farrell:** Data curation. **Arnoud Arntz:** Writing – review & editing, Visualization, Supervision, Methodology, Formal analysis, Data curation, Conceptualization.

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

During the preparation of this work the authors used OpenAI in order to improve readability. After using this tool/service, the authors reviewed and edited the content as needed and takes full responsibility for the content of the published article.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests:

This is a secondary data analysis from an earlier study. Funding & Support information for the primary study:

Arnoud Arntz reports a relationship with Netherlands Organisation for Health Research and Development that includes: funding grants. Arnoud Arntz reports a relationship with the Netherlands Foundation for Mental Health that includes: funding grants. Gitta A. Jacob reports a relationship with Else Kroner-Fresenius Foundation that includes: funding grants. Christopher W. Lee reports a relationship with Australian Rotary Health Research Fund that includes: funding grants. Ioannis A. Malogiannis reports a relationship with The Greek Society of Schema Therapy that includes: funding grants. Ioannis A. Malogiannis reports a relationship with The First Department of Psychiatry of the Medical School of the University of Athens that includes: funding grants. Gerhard Zarbock reports a relationship with The Institut für Verhaltenstherapie Ausbildung Hamburg that includes: funding grants. Florian A. Ruths & Anna Lavender reports a relationship with The South London and

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jbtep.2025.102074>.

Data availability

The data that has been used is confidential.

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