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# Schema modes as a common mechanism of change in personality pathology and functioning: Results from a randomized controlled trial

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ABSTRACT

*Objective:* We aimed to empirically test whether schema modes are central to the change process in schema therapy, clarification-oriented psychotherapy, and treatment as usual, i.e., predictive of personality pathology, and global and social-occupational functioning.
*Method:* A multicenter randomized controlled trial was conducted (N = 139 men, N = 181 women) over the course of three years. Repeated assessments of schema modes, personality disorder (PD) severity and functioning (controlled for concurrent PD-pathology) were analyzed using a multilevel autoregressive model. Variables were person-centered to ensure that within-person changes were analyzed. Through a process of backward elimination, the schema modes predictive of the dependent variable (i.e., PD-severity and functioning) at a later point in time were identified while controlling for concurrent dependent variable levels. Bidirectionality was tested by assessing whether dependent variables predicted later schema modes.
*Results:* The Healthy Adult, Vulnerable Child, Impulsive Child, and Avoidant Protector predicted later personality pathology, with no bidirectionality observed for the first two. The Healthy Adult and Self-Aggrandizer predicted functioning at a later point in time, with no bidirectionality for Self-Aggrandizer. There was no moderation by treatment type for PD symptomatology, except for Self-Aggrandizer, which predicted functioning only in schema therapy.
*Conclusions:* The Healthy Adult and Vulnerable Child are central to the change process and appear to reflect common mechanisms of change. The Self-Aggrandizer might reflect a change mechanism specific for schema therapy. Our findings support the recent emphasis on these modes in schema therapy.

## 1. Introduction

In recent years, schema therapy (ST) has been developed and validated as an integrated treatment for chronic and pervasive problems such as personality disorders (PDs). Recent studies have provided empirical evidence for the effectiveness of ST across a variety of disorders (Jacob & Arntz, 2013; Taylor, Bee, & Haddock, 2017). However, despite growing evidence in favor of the effectiveness of ST, inconsistencies reported in the literature do not allow for an integrated model of its mechanisms of change (Taylor et al., 2017).

ST was developed by Young (1990) to provide a solution to the chronic and pervasive problems that characterize personality pathology. The central triad of ST consists of early maladaptive schemas, schema coping styles and schema modes (SMs). *Early maladaptive schemas* (EMSs) are defined as generalized and persistent cognitive structures regarding oneself and the environment that develop as a result of challenging early life experiences and hinder successful adaptation later in life. These maladaptive schemas can then be triggered by particular events such as having an argument or failing a test, which might lead to a negative emotional outburst (Young, Klosko, & Weishaar, 2003). To avoid or regulate such negative emotional outbursts, individuals adopt various *coping mechanisms*. Young (1990) hypothesized that these coping mechanisms originate from innate fight-freeze-flight responses. When faced with schema-associated threats and their resulting negative emotions, individuals may display avoidance, surrender or overcompensation-based patterns of coping. Such coping reactions provide short-term relief, but eventually become toxic by contributing to the maintenance of EMS through preventing disconfirmation, and instead, eliciting reinforcing experiences. The third, and relatively new, key concept in ST is that of a *Schema Mode*. Young (1990) theorized the existence of SMs to explain the frequent changes he observed in patients' moods and behaviors during sessions. In contrast with schemas, which are considered to have a stable, trait-like character, a SM refers to the temporary emotional-cognitive-behavioral state of the individual formed by a combination of EMSs,

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coping responses and/or healthy functioning. SMs are thought to result from EMS-activation, or the threat of activation, in combination with a coping response to that (potential) activation. Research has supported this model by demonstrating that the type of coping response mediates the relationship between EMS and SMs, with SMs, in turn, predicting psychopathological symptoms (van Wijk-Herbrink et al., 2018). SMs are linked to psychopathological symptoms, as most symptoms can be viewed as directly resulting from an activated schema mode (Young, 1990).

Initially, Young et al. (2003) described ten SMs that could be grouped into four main categories: (1) *The child modes* are acquired in childhood and reflect the child's primary response to unmet emotional needs. Child modes display various innate emotions such as anger, disgust and happiness. Vulnerable (relating to feelings of loneliness, abandonment, abuse, neglect), Angry, and Impulsive Child modes are central to this category. Most EMSs are directly reflected in the Vulnerable Child mode because it is the core cognitive structure that reflects the cognitive-emotional reactions given as a reaction to damaging early childhood experiences. Thus, this mode is generally viewed as the most essential mode in the healing process in therapy. (2) *The dysfunctional parent modes* are rooted in internalized representations of punitive, critical or demanding responses from the child's significant others. An individual whose Demanding or Punitive Parent modes are triggered may treat themselves as their caregivers treated them in the past. Through the activation of these modes, introjected dysfunctional parenting attitudes continue to impair the individual's current functioning. (3) *The maladaptive coping modes* (relating to avoidance, detachment or overcompensation) consist of coping behaviors that developed in order to deal with extreme stress caused by unmet childhood needs. Although these modes functioned as survival strategies in childhood, they become dysfunctional in adult life. (4) Lastly, *the healthy modes* of Happy Child and Healthy Adult display integrated, wise and healthy functioning, as well as joyful, spontaneous and creative behavior. As might be expected, the Healthy Adult mode is viewed as a critical mode in the improvement process of the individual during treatment.

### 1.1. Mechanism of change in schema therapy for personality disorders

Given the challenges in treating PDs (e.g., comorbidity, complex childhood trauma, insecure relationship patterns and higher therapy drop-out rates) and lack of knowledge regarding the mechanism of change in PD treatment in general (see Aafjes-van Doorn & Barber, 2018), very few studies actually provide consistent plausible and coherent evidence on the mechanism in change in treatments of personality disorders (Forster, Berthollier, & Rawlinson, 2014).

The first randomized controlled trial to examine the (cost-)effectiveness of ST compared to psychodynamically based transference-focused psychotherapy was conducted with borderline personality disorder patients (Giesen-Bloo et al., 2006). This led to the recognition of ST as an evidence-based treatment for borderline PD and increased research interest in the clinical effectiveness of ST in other populations (Bamelis, Evers, Spinhoven, & Arntz, 2014; Bernstein et al., 2012; Farrell, Shaw, & Webber, 2009; Nadort et al., 2009). However, the growing evidence in favor of ST has mostly focused on outcomes (Gibbons et al., 2009), neglecting the mechanisms of change. An understanding of these mechanisms is essential to identifying the features that determine the effectiveness of a therapy approach (Kazdin, 2007).

Early theorization of ST suggested that a change in EMSs would predict the change in symptoms. Therefore, changes in EMSs were proposed as a mechanism of change in ST (Young et al., 2003). However, the evidence suggesting that EMSs drive therapeutic change is poor (Nordahl, Holthe, & Haugum, 2005; Renner et al., 2013; Taylor et al., 2017). Moreover, Renner et al. (2018) recently concluded that the negative core beliefs that lie at the center of EMSs did not predict subsequent changes in symptoms. Rather, symptoms and core beliefs improved concurrently, which might imply that a third mechanism underlies changes in both EMSs and depressive symptoms. Another possible candidate for the mechanisms of change in ST is therapeutic alliance (Fassbinder, Schweiger, Martius, Brand-de Wilde, & Arntz, 2016; Lockwood & Perris, 2012; Spinhoven, Giesen-Bloo, van Dyck, Kooiman, & Arntz, 2007). This suggestion was partially confirmed by Renner et al. (2018), who found that symptoms and alliance ratings within each session were associated, such that changes occurred concurrently. However, they also found that alliance ratings did not predict later changes in symptoms. Tschacher, Zorn, and Ramseyer (2012) found that insight and clarification are crucial to facilitating the changes in schema-focused therapy. Thus, the preliminary evidence suggests that gaining awareness about the problems within a working relationship leads to the development of new adaptive behaviors (Arntz & van Genderen, 2009; Roediger, 2012). Lastly, Fassbinder et al. (2016) highlighted the emerging empirical evidence for the importance of experiential techniques (such as imagery rescripting) as a mechanism of change. Experiential techniques address the Vulnerable Child mode, helping patients to emotionally process childhood memories of traumatic or aversive events, and to correct the dysfunctional meanings attached to these memories. These techniques thus aim to introduce awareness and responsibility taking, which are primarily functions of the Healthy Adult mode. Therefore, the SM perspective might be effective in understanding how the changes observed during ST occur.

As pointed out by Lemmens et al. (2017), the main problem with current research on the mechanisms of change in psychotherapy is that the hypothesized mechanisms generally do not conform with empirical evidence. On the contrary, symptoms predict the purported mechanisms instead of the other way around. One of the reasons for this might be that the hypothesized mechanisms are assessed with trait instruments rather than state instruments, which fail to catch temporal relationships between variables. The failure to find evidence for EMSs/beliefs underlying change in ST may be related to the trait character of these constructs. Schema modes are state constructs and might therefore better reflect change processes. Contemporary ST focuses on SMs in the treatment of chronic and pervasive disorders (Bernstein, Arntz, & Vos, 2007; Lobbestael, Van Vreeswijk, & Arntz, 2007) for at least four reasons: Firstly, because modes reflect the current state of the individual, they are relatively easily recognized by patients and therapists. Secondly, the concept of modes renders the sudden changes in states observed inside and outside of therapy sessions understandable and expected. Thirdly, psychopathological problems and symptoms can be easily understood as resulting from mode activation. Finally, the concept of SMs makes the idiosyncratic formulation of a case possible, allowing the therapist to tailor the focus and choice of techniques to the individual patient's needs. Recently, the original SM formulations described by Young et al. (2003) have been expanded to account for a broader range of settings and personality disorders (Arntz & Jacob, 2012; Bamelis, Renner, Heidkamp, & Arntz, 2011; Bernstein et al., 2012).

### 1.2. The present study

Although the SM model has become essential in modern applications of ST, there is not yet enough evidence to claim that SMs are indeed central to the change processes in treatment, let alone answer the question of which modes are the most crucial. The ultimate aim of ST is to bring about a change in EMSs. However, the trait-like character of EMSs makes it unlikely that these changes underlie symptom reduction. It seems more likely that the initial change during treatment is brought about by changing SMs, as these represent the momentary state of the patient and are the primary focus of treatment. Based on the considerations about mechanisms of change discussed above, we particularly expect that two modes will be important in the changes process: the Vulnerable Child mode (VC) and Healthy Adult mode (HA).

The first step of discovering the mechanisms of change is to demonstrate that the hypothesized mechanisms are mediators of the relationship between the treatment interventions and outcome variables (Kazdin, 2007, 2009). The direction of causality is also essential; i.e., the

treatment should cause a change in the mediator and the outcome (via the mediator) but not vice versa, meaning that the direction of causality cannot be reversed (Kazdin & Nock, 2003). Furthermore, measuring the mediator and outcome at multiple time-points during treatment offers a way to explore the direction of causality by focusing on the temporal relationships between variables. Thus, well-designed randomized controlled trials with comparison groups and multiple assessments at different time points provide an opportunity to gather high-quality data regarding the mechanisms of change (Lemmens et al., 2017; Taylor et al., 2017). Such a study was conducted by Bamelis et al. (2014), who evaluated the effectiveness of ST compared to clarification-oriented psychotherapy (COP) and treatment as usual (TAU) in a multicenter randomized controlled trial with repeated assessments over three years. This design provides an opportunity to observe the interplay between SMs, PD symptoms, global functioning and social-occupational functioning, and explore whether these associations differ between treatments (DeRubeis, Gelfand, German, Fournier, & Forand, 2014; Lorenzo-Luaces, German, & Derubeis, 2015). Note that ST-theory does not state that the role of SMs in PDs and the change process during treatment are unique to ST. Rather, the theory is formulated in universal terms.

The present study tested three hypotheses. First, we assessed whether SMs were associated with the subsequent ratings of PD severity, global functioning, and social-occupational functioning. It was expected that the Vulnerable Child and Healthy Adult modes would be the main predictors, as suggested by schema theory. To test the temporal relationships between variables, a multilevel autoregressive design (with the previous assessment of the dependent variable forced into the equation as a predictor) was employed, and a process of backward elimination of non-significant SMs was used until a model remained with only significant predictors. All variables were person-centered to prevent contamination of within-person variance with between-person variance. The SMs that significantly predicted PD severity, global functioning, and social-occupational functioning at later occasions were identified in three different models. Because treatment primarily focused on PD-pathology (which was also the primary outcome of interest in the randomized control trial), we investigated variance in global, and social-occupational functioning that was unaccounted for by PD-severity. Our second hypothesis was exploratory and involved testing the absence of bidirectionality. Bidirectionality was explored for each SM in the final model to check for reverse causality. Our third and final hypothesis was also exploratory: we tested whether SMs can be viewed as a common mechanisms of change for personality disorders regardless of the treatment approach. Therefore, we tested the moderating effect of treatment (i.e., schema therapy, clarification-oriented psychotherapy and treatment as usual) for each of the outcome variables.

## 2. Method

### 2.1. Participants

The data were collected from 12 Dutch mental health institutes as part of a multicenter randomized controlled trial conducted between 2006 and 2011 by Bamelis et al. (2014). Therefore, the participants will only be briefly described here. The sample consisted of 139 (43.4%) male and 181 (56.6%) female patients between the ages of 20 and 58 ($M$ = 37.98, $SD$ = 0.50). Participants had received a primary diagnosis of at least one PD (Avoidant = 163 (50.9%), dependent = 36 (11.3%), obsessive-compulsive = 89 (27.8%), paranoid = 14 (4.4%), histrionic = 2 (0.6%), and narcissistic = 16 (5.0%) PDs) based on the Structured Clinical Interview for DSM-IV Axis II PDs (SCID II) (First, Spitzer, Gibbon, Williams, & Benjamin, 1994). The intraclass correlation between the independent blinded interviewers based on the assessment of 42 audiotaped interviews was 0.84, indicating a good interrater reliability. Any patients who had an IQ score lower than 80, were at immediate suicide risk, were involved in substance abuse with a need of clinical detoxification and/or had a (co-morbid) diagnosis of antisocial, schizotypal, schizoid or borderline PD were excluded from the sample. Written consent forms were obtained from all participants. The medical ethics committee of Maastricht University approved the study. All participants were assessed six times: the first assessment was a pre-treatment assessment, the next four assessments were conducted every six months for two years, and a final follow-up assessment was conducted a year later.

Both ST and COP were delivered by weekly individual sessions based on a standardized treatment protocol. Therapists received a four-day training at the beginning of the trial and received weekly local supervision and yearly national supervision. COP-therapists were all accredited client-centered therapist or trainees, whereas self-expressed interest in ST was considered enough to be included in the trial as a schema therapist. Treatment as usual primarily included psychodynamic psychotherapy (insight-oriented psychotherapy (42%), supportive therapy (32%)), cognitive-behavioral therapy (19%), and eye movement desensitization and reprocessing (1.5%). Local indication staff selected the TAU deemed to be optimal for the specific participant. A detailed description of the methodological issues regarding data collection, procedure and intervention can be found in Bamelis, Evers, and Arntz (2012), and Bamelis et al. (2014).

### 2.2. Materials

**Schema Modes.** Bamelis et al. (2011) modified the SMI (Young et al., 2007) into the Schema Mode Inventory II (SMI-II) to assess all relevant SMs for the six PDs that the study focused on. Eighteen SMs were measured using 18 subscales, each with between 6 (Attention-Approval Seeking) and 13 (Detached Protector) items rated on six-point Likert scales, reflecting the frequency of manifestation of the item. Higher scores on a specific subscale are associated with a more frequent manifestation of the schema mode. The reliability scores for the subscales were found to range from 0.85 (Enraged Child) to 0.95 (Lonely Child) (Bamelis et al., 2011). An overview of the modes measured by the SMI is presented in Table 1. Given the high correlation ($r$ = 0.90, $p$ < .001) between the Lonely Child mode and Abandoned and Abused Child mode, these two modes were averaged and entered into the analyses as Vulnerable Child.

**Personality Psychopathology.** Assessments of DSM-IV PDs used the ADP-IV questionnaire (Schotte et al., 2004), a self-report instrument that consists of trait and distress scales to measure PD symptoms based on the DSM-IV. Respondents indicate on seven-point Likert scales the degree to which each item applies to them. The reliability of trait subscales was found to range from 0.60 (schizoid PD) to 0.84 (avoidant PD) (Schotte et al., 2004). For the present study, the total score was used as an index of severity of PD pathology. This score was log-transformed to reduce skewness (Bamelis et al., 2014).

**Table 1**
SMs measured by SMI-2.

| Mode Type | Schema Modes |
|---|---|
| Child | Lonely Child |
| | Abandoned and Abused Child |
| | Angry Child |
| | Enraged Child |
| | Impulsive Child |
| | Undisciplined Child |
| | Dependent Child |
| Coping Surrender | Compliant Surrender |
| Coping Avoidance | Detached Protector |
| | Detached Self-soother |
| | Avoidant Protector |
| Coping Overcompensator | Self-aggrandizer |
| | Perfectionist Over-controller |
| | Suspicious Over-controller |
| | Attention and Approval Seeking |
| Parent | Punitive Parent |
| | Demanding Parent |
| Healthy | Healthy Adult |

### 2.3. Functioning

***Global Assessment of Functioning Scale (GAF).*** This is an assessor-based instrument to assess global functioning. The GAF score represents global functioning on a scale of 0–100, given the interference of psychiatric symptoms. Higher scores represent better levels of global functioning. GAF ratings were given by independent raters at each assessment after conducting a semi-structured interview developed to get insight into the level of functioning of the participant (Bamelis et al., 2014).

***Social and Occupational Functioning Assessment Scale (SOFAS).*** This is an assessor-based instrument that assesses social and occupational (or academic) functioning, whilst considering mental and physical health problems. Participants are rated on a scale of 0–100, and higher scores represent better functional capacity. Independent raters gave SOFAS ratings at each assessment after conducting a semi-structured interview developed to get insight into the level of social and occupational (or academic) functioning of the participant (Bamelis et al., 2014).

### 2.4. Statistical analysis

Multilevel autoregressive models with a restricted maximum likelihood estimator (REML) were utilized to test cross-lagged associations between SMs, ADP-IV, SOFAS, and GAF scores over subsequent measurements at baseline, 6, 12, 18, 24 and 36 months. Scores at each previous time point were used as lagged predictors of scores at the following time points. Moreover, the lagged outcome variables at the previous time points were added as covariates while predicting the outcome variables in each of the models to control for autocorrelation.

We aimed to identify the mechanisms of change during treatment at an individual level. Enders and Tofighi (2007) suggest basing the centering decision on the particular research question. Furthermore, Hamaker and Grasman (2014) advise using within-person centering if there is a definite interest in getting results that are interpretable at an individual level with less bias, while Wang and Maxwell (2015) recommend using within-person centering to obtain more accurate fixed effects and variance component estimates. Therefore, all variables were centered at the individual level to prevent the contamination of the effects of predictors on the dependent variables by between-subject variance. The normality and variability of the data were screened and outliers identified via histograms, boxplots and scatterplots. The results were robust with various ways of handling outliers. Therefore, the original data were utilized, including outliers.

Regarding the main analyses, three separate multilevel autoregressive models were built to identify the specific SMs that predicted ADP-IV, GAF, and SOFAS scores. For each model, repeated multilevel regression analyses were performed using stepwise backward elimination of the non-significant SMs, with a two-tailed significance level of .05 as a criterion. Because all treatments focused primarily on the PD-related problems of patients, we considered PD-severity (assessed with the ADP-IV) the primary outcome. For theoretical reasons related to our hypotheses, we chose to control GAF and SOFAS scores for PD-severity by analyzing the residuals after regressing these variables on the concurrent (person-centered) ADP-IV instead of adding PD-severity as a covariate. In other words, we investigated whether time-lagged SMs contributed to GAF and SOFAS scores above what is explained by patients' PD-severity at the time. Results revealed a robust SM structure for ADP-IV; the multilevel autoregressive model for GAF and SOFAS ended up with the same model.

Lastly, bidirectional relationships between the predictors and outcome variables of the main models were explored in separate models for each of the SMs that turned out to be significant predictors of ADP-4, GAF and SOFAS. Bidirectionality was checked for each SM separately. Thus, in every analysis, each of the significant SMs was employed as an outcome variable. This variable was predicted by lagged measurements of the other SMs included in the relevant model, the original outcome variable of the model (ADP-IV, GAF or SOFAS), and its own lagged measurement at the previous time-point. All analyses were conducted in SPSS version 23 for Windows.

## 3. Results

### 3.1. Preliminary analyses

Descriptive statistics of the SMs, global severity of PDs (ADP-IV), global assessment of functioning (GAF), and social-occupational functioning assessment (SOFAS) over three years with the baseline assessment are presented in Table 2.

### 3.2. Prediction of PD-severity, GAF, and SOFAS by SMs

Three separate SM models were built to identify the most crucial SM associates of ADP-IV, GAF, and SOFAS. The results of the autoregressive multilevel regression analyses after stepwise deletion of nonsignificant SMs are presented in Table 3. The first schema mode model for ADP-IV revealed significant effects for the time-lagged scores of Vulnerable Child (VC), Impulsive Child (IC), Avoidant Protector (AP), and Healthy Adult (HA). As expected, the time-lagged ADP-IV scores at the previous time point also significantly correlated with ADP-IV scores at the later time points. The interactions between treatment condition and SMs were not significant.

The second model aimed to identify the SMs that predicted SOFAS scores, specifically the variability in SOFAS scores that was not explained by current PD-pathology scores (i.e., the residual SOFAS scores). The strongest predictor of SOFAS scores was the SOFAS scores from the previous assessment. Out of the SMs, HA was the strongest predictor of SOFAS, followed by the Self-aggrandizer (SA) mode. The third model, which was built to identify the predictors of GAF scores, revealed the same results as those of the SOFAS model. The strongest predictor was GAF at previous assessments, followed by the HA and SA modes. There was a significant interaction between the treatment condition and the SA mode on both SOFAS ($F$ (2, 1122) = 3.31, $p$ = .036) and GAF ($F$ (2, 1122) = 3.60, $p$ = .028): the SA mode had a stronger predictive effect in the ST condition compared to the treatment as usual. After deletion of the nonsignificant interactions, the predictive effects of the SA mode on functioning differed significantly between the ST and the other conditions (Table 4). From Table 4, it is also clear that SA did not have notable relationships to GAF and SOFAS in COP and TAU, while the relationships were significant in ST.

### 3.3. Examination of bidirectionality

Nine separate autoregressive models were tested to explore possible bidirectionality. In each of these models, an SM was employed as an outcome variable. The other SMs that were significant in the prediction model and the former dependent variable of the pertinent ADP-IV, SOFAS or GAF models were entered as the predictor variables. The time-lagged version of the SM that was the dependent variable in the analysis (i.e., the SM at a previous time) was also included in the analyses to account for autoregression. For example, to assess possible bidirectionality for the VC mode and ADP-IV, the (person-centered) VC mode was entered into the model as the dependent variable, while time-lagged (person-centered) ADP-IV, VC, HA, IC, and AP scores of the previous assessment were entered as predictors.

### 3.4. Examination of bidirectionality of SMs and PDSs

The bidirectionality analyses for the first set of SMs that predicted ADP-IV scores are displayed in Table 5. The VC mode was predicted by the time-lagged scores of HA, as well as previous VC scores. IC mode was only predicted by time-lagged scores of the ADP-IV at the previous time-point. The AP mode was predicted by the time-lagged scores of VC, HA, and ADP-IV. Lastly, the only significant predictor of the HA mode was the time-lagged VC, indicating that only lower VC ratings predicted higher HA ratings at the next assessment.

**Table 2**
Descriptive characteristics of SMs, ADP-IV, GAF, SOFAS scores at baseline, treatment and follow-up levels.

| Variable | Baseline M (SD) | 6-month M (SD) | 12-month M (SD) | 18-month M (SD) | 24-month M (SD) | 32- month M (SD) |
|---|---|---|---|---|---|---|
| ADP-IV | 295.96 (69.58) | 274.94 (71.67) | 256.75 (76.29) | 246.61 (78.57) | 233.53 (80.16) | 231.17 (81.61) |
| Log transformed ADP-IV | 5.66 (0.25) | 5.59 (0.27) | 5.51 (0.32) | 5.46 (0.33) | 5.40 (0.35) | 5.38 (0.36) |
| GAF | 56.11 (8.82) | 59.39 (9.71) | 61.77 (11.45) | 64.26 (11.92) | 67.24 (12.73) | 69.64 (13.69) |
| SOFAS | 55.92 (9.82) | 59.18 (10.30) | 61.35 (11.18) | 63.64 (11.68) | 66.90 (12.38) | 69.54 (13.59) |
| Vulnerable Child | 3.40 (0.90) | 3.10 (0.94) | 2.88 (0.94) | 2.73 (0.97) | 2.57 (1.00) | 2.48 (0.99) |
| Angry Child | 2.72 (0.85) | 2.61 (0.86) | 2.42 (0.89) | 2.35 (0.87) | 2.24 (0.88) | 2.19 (0.84) |
| Enraged Child | 1.78 (0.76) | 1.68 (0.71) | 1.60 (0.69) | 1.56 (0.68) | 1.52 (0.68) | 1.52 (0.67) |
| Impulsive Child | 2.55 (0.86) | 2.41 (0.80) | 2.26 (0.78) | 2.25 (0.80) | 2.13 (0.75) | 2.10 (0.78) |
| Undisciplined Child | 2.93 (0.76) | 2.73 (0.76) | 2.64 (0.79) | 2.53 (0.80) | 2.44 (0.79) | 2.32 (0.76) |
| Dependent Child | 2.87 (0.89) | 2.72 (0.89) | 2.57 (0.91) | 2.43 (0.92) | 2.30 (0.93) | 2.23 (0.91) |
| Compliant Surrender | 3.82 (0.82) | 3.63 (0.88) | 3.44 (0.91) | 3.27 (0.90) | 3.14 (0.94) | 3.02 (0.98) |
| Detached Protector | 3.01 (0.86) | 2.77 (0.88) | 2.61 (0.90) | 2.50 (0.94) | 2.34 (0.87) | 2.34 (0.99) |
| Detached Self Soother | 2.96 (0.77) | 2.82 (0.79) | 2.64 (0.80) | 2.54 (0.86) | 2.43 (0.86) | 2.34 (0.88) |
| Avoidant Protector | 3.72 (0.93) | 3.49 (0.94) | 3.33 (0.98) | 3.14 (1.00) | 2.99 (1.00) | 2.91 (1.02) |
| Self-aggrandizer | 2.54 (0.80) | 2.43 (0.74) | 2.31 (0.67) | 2.30 (0.72) | 2.20 (0.71) | 2.24 (0.76) |
| Perfectionist over-controller | 3.70 (0.83) | 3.49 (0.83) | 3.34 (0.88) | 3.25 (0.94) | 3.09 (0.92) | 2.91 (0.94) |
| Suspicious Over-controller | 3.21 (0.99) | 3.06 (0.95) | 2.87 (0.94) | 2.72 (0.98) | 2.62 (1.00) | 2.63 (1.06) |
| Attention and Approval Seeking | 1.88 (0.81) | 1.87 (0.79) | 1.85 (0.74) | 1.90 (0.78) | 1.90 (0.76) | 1.92 (0.75) |
| Punitive Parent | 2.82 (0.91) | 2.65 (0.95) | 2.45 (0.96) | 2.31 (0.93) | 2.16 (0.89) | 2.03 (0.88) |
| Demanding parent | 3.82 (0.86) | 3.65 (0.87) | 3.50 (0.86) | 3.38 (0.90) | 3.19 (0.90) | 3.02 (0.91) |
| Healthy Adult | 3.00 (0.75) | 3.23 (0.84) | 3.39 (0.84) | 3.56 (0.91) | 3.74 (0.94) | 3.80 (0.96) |

The results of the main analyses and the examination of bidirectionality can be summarized in a final predictive model for the global severity of PDs (see Fig. 1). As is shown in the model, the VC and HA modes have unidirectional relationships with ADP-IV and AP ratings: they are not affected by previous ADP-IV or AP ratings and can thus be viewed as fundamental starting points of change. These SMs also have the highest numbers of predictive associations: The number of outgoing arrows is 4 for VC and 3 for HA; in contrast to 1 for AP and 1 for IC. The VC and HA modes also influence each other over time. The AP mode partially mediates the predictive effects of HA and VC, and has a bidirectional relationship with the ADP-IV. The position of the IC mode is rather isolated, but demonstrates a bidirectional relationship with the ADP-IV.

### 3.5. Examination of bidirectionality between SMs and SOFAS

Two separate analyses were conducted to explore bidirectionality for the second set of SMs that predicted SOFAS scores (See Table 6). The first analysis was conducted for the HA mode. HA was predicted by its own measurement at the previous time-point, as well as SA ratings and SOFAS scores. Besides its own measurement at a previous level, decreased manifestation of the SA mode and elevated SOFAS levels were predictive of increased HA ratings. The second analysis revealed that SA was only predicted by HA at the previous time point. That is, an increase in HA scores at the previous time point predicted a reduced manifestation of SA at a subsequent point. In both analyses, HA at the previous measurement was the strongest predictor.

The results of the main analyses and the examination of bidirectionality are summarized in the SOFAS predictive model. As can be seen in Fig. 2, HA has a central role in the model in that it predicts both SOFAS and SA scores at a later assessment and is also its own strongest predictor (outgoing arrows; HA: 3, SOFAS: 2, SA: 1). The only bidirectional relationship observed was between HA and SOFAS scores.

### 3.6. Examination of bidirectionality between SMs and GAF

The last set of multilevel analyses were conducted to explore bidirectionality between SMs and GAF scores (see Table 7). The results largely overlapped with those of the SOFAS analysis: HA was predicted by its own measurement and GAF at the previous time-point. SA was only predicted by HA at the previous time-point. Fig. 3 displays the predictive model for GAF. Similar to the SOFAS results, HA has a central role in the prediction of GAF (outgoing arrows; HA: 3, GAF: 2, SA: 1).

**Table 3**
Results of the multilevel autoregressive analysis for ADP-IV, GAF, and SOFAS.

| Outcome variable | Lagged predictor at the previous level | Estimate | SE | t | df | r[a] | 95% CI | | p |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | Lower | Upper | |
| ADP-IV | VC | 0.04 | 0.01 | 3.07 | 1183 | 0.09 | 0.02 | 0.07 | 0.002 |
| | IC | 0.04 | 0.01 | 3.66 | 1183 | 0.11 | 0.02 | 0.07 | < 0.001 |
| | AP | 0.03 | 0.01 | 2.48 | 1183 | 0.07 | 0.01 | 0.06 | 0.013 |
| | HA | −0.04 | 0.01 | −2.91 | 1183 | 0.08 | −0.06 | −0.01 | 0.004 |
| | Pre ADP-IV | 0.11 | 0.04 | 2.86 | 1183 | 0.08 | 0.03 | 0.19 | 0.004 |
| SOFAS | SA | −1.39 | 0.58 | −2.39 | 1130 | 0.07 | −2.53 | −0.25 | 0.017 |
| | HA | 1.70 | 0.38 | 4.45 | 1130 | 0.13 | 0.95 | 2.45 | < 0.001 |
| | Pre SOFAS | 0.14 | 0.03 | 4.59 | 1130 | 0.14 | 0.08 | 0.19 | < 0.001 |
| GAF | SA | −1.56 | 0.61 | −2.57 | 1130 | 0.08 | −2.75 | −0.37 | 0.010 |
| | HA | 1.53 | 0.40 | 3.81 | 1130 | 0.11 | 0.74 | 2.32 | < 0.001 |
| | Pre GAF | 0.17 | 0.03 | 5.47 | 1130 | 0.16 | 0.11 | 0.23 | < 0.001 |

*Note.* These results are of the final models that resulted after backward deletion of non-significant predictors. For SOFAS and GAF residual scores were taken, after regressing current ADP-IV on these variables (after person-based centering). VC = Vulnerable Child, HA = Healthy Adult, AP = Avoidant Protector, IC = Impulsive Child, SA = Self-Aggrandizer, ADP-IV = PD symptoms; SOFAS = Social/occupational functioning; GAF = General functioning; pre = previous assesment.

[a] Effect size r defined as $r = \sqrt{(t^2/(t^2 + d.f.))}$.

**Table 4**
The interaction effects of TAU and COP conditions compared to ST for ADP-IV, SOFAS, and GAF.

| Outcome variable | Main effects of treatment conditions, predictors at previous assessments and their interactions | Estimate | SE | *t* | 95% CI | | *p* |
|---|---|---|---|---|---|---|---|
| | | | | | Lower | Upper | |
| ADP-IV | COP | −0.003 | 0.01 | −0.25 | −0.02 | 0.02 | 0.80 |
| | TAU | −0.0004 | 0.01 | −0.06 | −0.02 | 0.02 | 0.96 |
| | ADP-IV | 0.10 | 0.06 | 1.70 | −0.02 | 0.22 | 0.09 |
| | Vulnerable Child | 0.06 | 0.02 | 2.98 | 0.02 | 0.10 | 0.003 |
| | Impulsive Child | 0.02 | 0.02 | 1.41 | −0.01 | 0.06 | 0.16 |
| | Avoidant Protector | 0.03 | 0.02 | 1.50 | −0.01 | 0.06 | 0.13 |
| | Healthy Adult | −0.03 | 0.02 | −1.56 | −0.07 | 0.01 | 0.12 |
| | COP x ADP-IV | 0.03 | 0.11 | 0.27 | −0.18 | 0.24 | 0.79 |
| | TAU x ADP-IV | 0.005 | 0.09 | 0.06 | −0.17 | 0.18 | 0.95 |
| | COP x Vulnerable Child | −0.02 | 0.04 | −0.47 | −0.10 | 0.06 | 0.64 |
| | TAU x Vulnerable Child | −0.04 | 0.03 | −1.31 | −0.11 | 0.02 | 0.19 |
| | COP x Impulsive Child | 0.07 | 0.04 | 1.76 | −0.01 | 0.15 | 0.08 |
| | TAU x Impulsive Child | 0.03 | 0.03 | 1.24 | −0.02 | 0.08 | 0.21 |
| | COP x Avoidant Protector | 0.002 | 0.04 | 0.06 | −0.08 | 0.08 | 0.95 |
| | TAU x Avoidant Protector | 0.008 | 0.03 | 0.30 | −0.05 | 0.06 | 0.77 |
| | COP x Healthy Adult | 0.03 | 0.04 | 0.66 | −0.05 | 0.10 | 0.51 |
| | TAU x Healthy Adult | −0.03 | 0.03 | −1.10 | −0.08 | 0.02 | 0.27 |
| SOFAS | COP | 0.16 | 0.52 | 0.30 | −0.86 | 1.17 | 0.76 |
| | TAU | 0.18 | 0.38 | 0.47 | −0.57 | 0.92 | 0.64 |
| | SOFAS | 0.12 | 0.04 | 2.88 | 0.04 | 0.21 | 0.004 |
| | Self-Aggrandizer | −2.85 | 0.81 | −3.52 | −4.44 | −1.26 | < .001 |
| | Healthy Adult | 2.24 | 0.56 | 4.00 | 1.14 | 3.34 | < .001 |
| | COP x SOFAS | 0.08 | 0.08 | 0.92 | −0.09 | 0.24 | 0.36 |
| | TAU x SOFAS | −0.01 | 0.07 | −0.22 | −0.14 | 0.11 | 0.82 |
| | COP x Self-Aggrandizer | 2.80 | 2.04 | 1.37 | −1.21 | 6.82 | 0.17 |
| | TAU x Self-Aggrandizer | 3.03 | 1.23 | 2.47 | 0.62 | 5.45 | 0.014 |
| | COP x Healthy Adult | −1.92 | 1.19 | −1.61 | −4.26 | 0.42 | 0.11 |
| | TAU x Healthy Adult | −0.60 | 0.82 | −0.73 | −2.21 | 1.01 | 0.47 |
| GAF | COP | 0.11 | 0.54 | 0.20 | −0.95 | 1.17 | 0.84 |
| | TAU | 0.19 | 0.39 | 0.48 | −0.58 | 0.96 | 0.63 |
| | GAF | 0.20 | 0.05 | 4.43 | 0.11 | 0.29 | < .001 |
| | Self-Aggrandizer | −3.09 | 0.84 | −3.66 | −4.75 | −1.44 | < .001 |
| | Healthy Adult | 1.68 | 0.59 | 2.87 | 0.53 | 2.83 | 0.004 |
| | COP x GAF | 0.03 | 0.09 | 0.30 | −0.15 | 0.21 | 0.76 |
| | TAU x GAF | −0.10 | 0.07 | −1.46 | −0.23 | 0.03 | 0.15 |
| | COP x Self-Aggrandizer | 2.29 | 2.13 | 1.08 | −1.87 | 6.47 | 0.28 |
| | TAU x Self-Aggrandizer | 3.41 | 1.29 | 2.65 | 0.89 | 5.94 | 0.008 |
| | COP x Healthy Adult | −2.10 | 1.24 | −1.69 | −4.53 | 0.34 | 0.09 |
| | TAU x Healthy Adult | 0.45 | 0.86 | 0.52 | −1.24 | 2.15 | 0.60 |

*After deletion of n.s. interactions*

| Outcome | Predictors | Estimate | SE | t | r[a] | 95% CI | | p |
|---|---|---|---|---|---|---|---|---|
| | | | | | | Lower | Upper | |
| SOFAS | COP | 0.17 | 0.52 | 0.32 | 0.01 | −0.85 | 1.18 | 0.75 |
| | TAU | 0.18 | 0.38 | 0.48 | 0.01 | −0.56 | 0.92 | 0.63 |
| | SOFAS | 0.13 | 0.03 | 4.46 | 0.13 | 0.07 | 0.19 | < .001 |
| | Self-Aggrandizer | −3.10 | 0.78 | −3.97 | 0.12 | −4.63 | −1.56 | < .001 |
| | Healthy Adult | 1.76 | 0.38 | 4.61 | 0.14 | 1.01 | 2.51 | < .001 |
| | COP x Self-Aggrandizer[b] | 4.24 | 1.84 | 2.31 | 0.07 | 0.63 | 7.85 | 0.021 |
| | TAU x Self-Aggrandizer[c] | 3.33 | 1.15 | 2.90 | 0.09 | 1.08 | 5.57 | 0.004 |
| GAF | COP | 0.13 | 0.54 | 0.24 | 0.01 | −0.93 | 1.19 | 0.81 |
| | TAU | 0.20 | 0.39 | 0.51 | 0.02 | −0.57 | 0.97 | 0.61 |
| | GAF | 0.17 | 0.03 | 5.38 | 0.16 | 0.11 | 0.23 | < .001 |
| | Self-Aggrandizer | −3.14 | 0.81 | −3.86 | 0.11 | −4.74 | −1.54 | < .001 |
| | Healthy Adult | 1.58 | 0.40 | 3.95 | 0.12 | 0.80 | 2.37 | < .001 |
| | COP x Self-Aggrandizer[d] | 3.96 | 1.92 | 2.07 | 0.06 | 0.20 | 7.72 | 0.039 |
| | TAU x Self-Aggrandizer[e] | 3.07 | 1.19 | 2.57 | 0.08 | 0.73 | 5.41 | 0.010 |

*Note.* SE = Standard Error, d.f = 1128), TAU = treatment as usual, COP = Clarification-oriented psychotherapy, ADP-IV = PD symptoms; SOFAS = Social/occupational functioning; GAF = General functioning. ST (schema therapy) is the reference.
Effect of Self-Aggrandizer within ST on SOFAS is −3.10 (s.e. 0.78, t = −3.97, r = 0.12, p < .001).
Effect of Self-Aggrandizer within ST on GAF is −3.14 (s.e. 0.81, t = −3.86, r = 0.11, p < .001).

[a] r = effect size ($r = \sqrt{(t^2/(t^2 + d.f.))}$).
[b] Effect of Self-Aggrandizer within COP is 1.15 (s.e. = 1.70, t = 0.68, r = 0.02, n.s.).
[c] Effect of Self-Aggrandizer within TAU is 0.24 (s.e. = 0.89, t = 0.27, r = 0.01, n.s.).
[d] Effect of Self-Aggrandizer within COP is 0.82 (s.e. = 1.77, t = 0.64, r = 0.01, n.s.).
[e] Effect of Self-Aggrandizer within TAU is −0.06 (s.e. = 0.93, t = −0.07, r = 0.002, n.s.).

**Table 5**
Results of the exploration of bi-directionality Schema Modes - PD symptoms.

| Outcome variable | Lagged predictor at the previous assessment | Estimate | SE | *t* | 95% CI | | *P* |
|---|---|---|---|---|---|---|---|
| | | | | | Lower | Upper | |
| VC | Vulnerable Child (VC) | 0.15 | 0.05 | 3.14 | 0.06 | 0.25 | 0.002 |
| | Impulsive child | 0.05 | 0.04 | 1.35 | −0.02 | 0.13 | 0.176 |
| | Avoidant protector | 0.08 | 0.04 | 1.95 | −0.001 | 0.16 | 0.052 |
| | Healthy Adult | −0.08 | 0.04 | −2.04 | −0.16 | −0.003 | 0.041 |
| | PD Symptoms | 0.14 | 0.13 | 1.10 | −0.11 | 0.40 | 0.270 |
| IC | Vulnerable Child | 0.07 | 0.04 | 1.81 | −0.01 | 0.15 | 0.070 |
| | Impulsive child (IC) | −0.05 | 0.03 | −1.54 | −0.11 | 0.01 | 0.124 |
| | Avoidant protector | 0.04 | 0.03 | 1.31 | −0.02 | 0.11 | 0.189 |
| | Healthy Adult | −0.04 | 0.03 | −1.08 | −0.10 | 0.03 | 0.281 |
| | PD Symptoms | 0.26 | 0.10 | 2.52 | 0.06 | 0.46 | 0.012 |
| AP | Vulnerable Child | 0.18 | 0.05 | 3.78 | 0.09 | 0.27 | < 0.001 |
| | Impulsive child | 0.03 | 0.04 | 0.84 | −0.04 | 0.11 | 0.404 |
| | Avoidant protector (AP) | 0.03 | 0.04 | 0.70 | −0.05 | 0.11 | 0.486 |
| | Healthy Adult | − 0.11 | 0.04 | −2.72 | −0.19 | −0.03 | 0.007 |
| | PD Symptoms | 0.26 | 0.13 | 2.02 | 0.01 | 0.51 | 0.043 |
| HA | Vulnerable Child | −0.11 | 0.05 | −2.14 | −0.22 | −0.01 | 0.033 |
| | Impulsive child | −0.04 | 0.04 | −0.89 | −0.12 | 0.04 | 0.373 |
| | Avoidant protector | −0.06 | 0.05 | −1.39 | −0.16 | 0.03 | 0.165 |
| | Healthy Adult (HA) | 0.06 | 0.04 | 1.23 | −0.03 | 0.14 | 0.218 |
| | PD Symptoms | −0.26 | 0.14 | −1.84 | −0.54 | 0.01 | 0.066 |

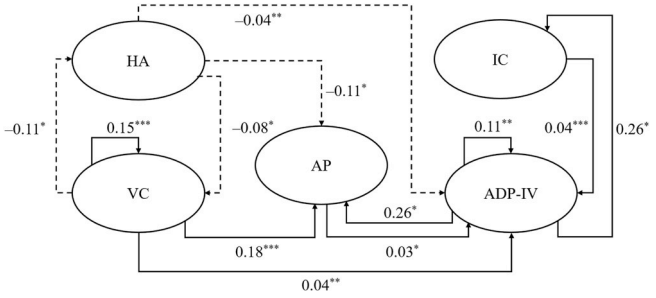


**Fig. 1.** Time-lagged relationships between schema modes and personality pathology (ADP-IV). Arrows represents significant time-lagged associations. Parameter values are displayed next to respective arrows with the significance level (*$p < .05$; **$p < .01$; ***$p < .001$). Significant positive associations are represented by continuous arrows, whereas negative associations are represented by dashed arrows. ADP-IV = PD symptoms; VC = Vulnerable Child; IC = Impulsive Child; AP = Avoidant protector; HA = Healthy Adult.

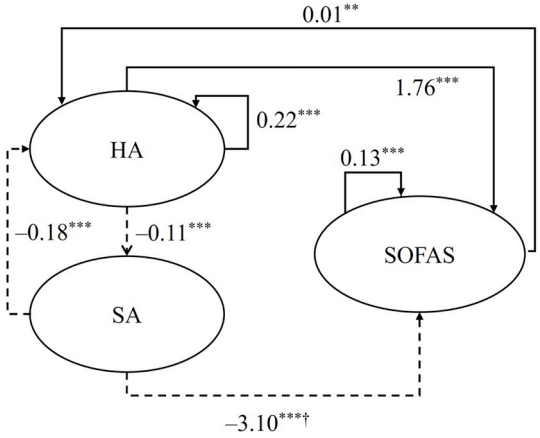


**Fig. 2.** Time-lagged relationships between schema modes and social-occupational functioning (SOFAS). *Note.* Arrows represents significant time-lagged associations. Parameter values are displayed next to respective arrows with the significance level (*p < .05; **p < .01; ***p < .001). Significant positive associations are represented by continuous arrows, whereas negative associations are represented by dashed arrows. SOFAS-scores were controlled for concurrent PD-pathology. SOFAS = Social and Occupational Functional Assessment Scale; SA = Self-Aggrandizer; HA = Healthy Adult. †Only within ST (the effect was N.S. in the other treatments).

## 4. Discussion

This study aimed to test the role of schema modes as a mechanism of change in schema therapy for PDs. We focused on the predictive effects of SMs on PD-pathology and functioning (i.e., global, social, and occupational) during a 3-year period using data from a large randomized control trial. We focused on the individual level by investigating temporal, time-lagged relations between these variables. Variables were repeatedly assessed and we used mixed regression to aggregate the results across participants after person-centering. We first identified the SMs that predicted later PD severity levels, and then identified the SMs that predicted subsequent global and social-occupational functioning.

The multilevel mixed regression analysis for the first model revealed that increased HA scores and decreased levels of three maladaptive SMs (i.e., VC, IC and AP) predicted improvement in PD-pathology. The second

**Table 6**
Results of the exploration of bi-directionality Schema Modes – social-occupational functioning (SOFAS).

| Outcome variable | Lagged predictor at the previous assessment | Estimate | SE | *t* | 95% CI | | *P* |
|---|---|---|---|---|---|---|---|
| | | | | | Lower | Upper | |
| HA | Self-Aggrandizer (SA) | −0.18 | 0.05 | −3.84 | −0.27 | −0.09 | < 0.001 |
| | SOFAS | 0.01 | 0.002 | 2.91 | 0.002 | 0.01 | 0.004 |
| | Previous HA | 0.22 | 0.03 | 6.92 | 0.15 | 0.28 | < 0.001 |
| SA | Healthy Adult (HA) | −0.11 | 0.02 | 5.39 | −0.15 | −0.07 | < 0.001 |
| | SOFAS | −0.002 | 0.002 | −1.40 | −0.005 | 0.001 | 0.160 |
| | Previous SA | −0.01 | 0.03 | −0.34 | −0.07 | 0.05 | 0.732 |

**Table 7**
Results of the exploration of bi-directionality Schema Modes - global functioning (GAF).

| Outcome variable | Lagged predictor at the previous assessment | Estimate | SE | *t* | 95% CI | | *P* |
|---|---|---|---|---|---|---|---|
| | | | | | Lower | Upper | |
| HA | Self-Aggrandizer (SA) | −0.19 | 0.05 | −4.13 | −0.29 | −0.10 | < 0.001 |
| | GAF | 0.01 | 0.002 | 4.30 | 0.005 | 0.02 | < 0.001 |
| | Previous HA | 0.20 | 0.03 | 6.34 | 0.14 | 0.26 | < 0.001 |
| SA | Healthy Adult (HA) | −0.11 | 0.02 | −5.23 | −0.15 | −0.07 | < 0.001 |
| | GAF | −0.002 | 0.001 | −1.41 | −0.005 | 0.001 | 0.159 |
| | Previous SA | −0.01 | 0.03 | −0.28 | −0.07 | 0.05 | 0.781 |

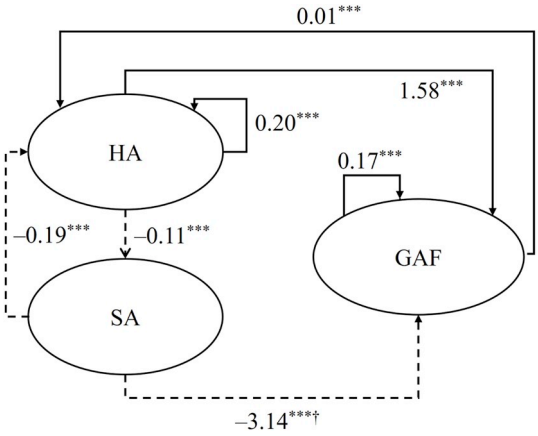


**Fig. 3.** Time-lagged relationships between schema modes and general functioning (GAF). Arrows represent significant time-lagged associations. Parameter values are displayed next to their respective arrows with the significance level (*p < .05; **p < .01; ***p < .001). Significant positive associations are represented by continuous arrows, whereas negative associations are represented by dashed arrows. GAF-scores were controlled for concurrent PD-pathology. GAF = Global Assessment of Functioning Scale; SA = Self-Aggrandizer; HA = Healthy Adult. †Only within ST (the effect was N.S. in the other treatments).

and third models (Figs. 2 and 3) identified the relevant SMs (i.e., HA, SA) for global and social-occupational functioning. Finally, we also found that treatment condition (i.e., schema therapy, clarification-oriented therapy and treatment as usual) did not moderate the relationships in the model for PD-pathology. Treatment did however moderate the relationship between Self-Aggrandizer and later functioning, with presence of such a predictive association in ST, and absence in COP and TAU.

In the first model, the VC mode was the most important predictor, predicting not only PD-pathology but also nearly all the SMs in the model (i.e., ADP-IV, HA, AP, VC). The second most crucial SM was HA, predicting three other variables in the model. Interestingly, there was a reciprocal time-lagged relationship between HA and VC (i.e., HA predicting later VC ratings, and VC predicting later HA ratings). Thus, VC and HA did not only have a central role in the change process of other variables, but also affected each other. Empowering the HA mode would be expected to lead to a decrease in VC, which in turn would result in a stronger HA mode at the next time point. Thus, the VC and HA modes appear to play the biggest role in the change in PD-pathology. It is also essential to emphasize that this finding does not simply suggest that healthier people benefit more from the treatment. Indeed, although the baseline score of the HA mode predicted baseline ADP-IV scores ($t = -12.43, p < .001$), baseline HA did not predict the change in ADP-IV scores[1] over time ($t = 0.19, p = .85$). This means that improvement in PD-pathology was not predicted by baseline assessment of (psychological) healthiness, but still, an intra-individual increase in healthiness predicts lower PD-pathology at a later point of assessment. Importantly, HA and VC were predictive of PD severity but the opposite was not observed, thus ruling out bidirectionality. This fulfills a precondition to identify HA and VC as mechanism of change (Kazdin, 2007, 2009). On the other hand, the predictive effects of the IC and AP modes on PD severity were bidirectional, meaning that increased levels of IC and AP were predictive of later PD-related pathology, and vice versa. The relationship between the IC mode and PD pathology was relatively isolated compared to the AP mode, which was associated with the other SMs in the model and partially mediated the effects of HA and VC on PD pathology.

The second and third models (Figs. 2 and 3) identified the relevant SMs for global and social-occupational functioning. As expected, the results were similar for both models: increased levels of HA and decreased levels of SA predicted healthier functioning at both global and social-occupational levels. Moreover, the relationship between the HA mode and healthy functioning was bidirectional, meaning that increases in HA influence functioning positively, while an increase in healthy functioning in turn stimulates further growth of the HA mode (Arntz & Jacob, 2012; Young et al., 2003). On the other hand, the predictive effect of SA was not bidirectional, meaning that decreased levels of SA preceded an increase in healthy functioning, but functioning did not have a significant effect on the level of SA. This finding should be interpreted with caution as the predictive effect of SA on functioning was not hypothesized and was only evident in ST. Nevertheless, this finding suggests that ST changes the relationship between SA and functioning, compared to other treatments (Kraemer, Wilson, Fairburn, & Agras, 2002). An explanation would be that ST works with the SA mode on a more profound level compared to the other treatments. As the SA has a central role in communication as an over-compensating coping mode, it might often cause ruptures in the therapeutic relationship. These ruptures are both welcomed and desirable in ST in order to empathically confront the patient with their maladaptive coping behaviors. Because of explicitly addressing the role of the SA mode in interpersonal situations, this mode might have played a more prominent role in the change process in ST than in the other treatments. Nonetheless, this post-hoc explanation about the ST-specific role of SA in the process of change in functioning needs further testing.

The present study is important as it addresses the lack of empirical knowledge regarding the mechanisms of change in the treatment of PDs (Aafjes-van Doorn & Barber, 2018; Fassbinder et al., 2016; Taylor et al., 2017). Our results indicate that independent of treatment model, the

[1] The same model was used as in the Bamelis et al. (2014) study, but now baseline HA and the interaction between time (as a dimensional predictor, with time log-transformed to create a linear relationship between time and outcome) (*footnote continued*) and baseline HA were added to the mixed regression. On request of an anonymous reviewer, we also assessed the predictive effect of HA baseline while controlling for the ADP-IV baseline per assessment. As predicted by the reviewer, this was significant at 6 months, indicating that psychologically healthier people have an initially faster response. However, baseline HA did not predict later improvements, thus pre-treatment HA does not predict an ultimately stronger response.

main focus of the treatment should be healing the vulnerable part of the patient – which was originally suggested as the ultimate aim of the schema therapy (Young, 1990; Young et al., 2003) – as well as strengthening the healthy side of the patient. Our results also provide insight into the mediating role of coping modes in the treatment process. With relatively high AP, the effect of HA and VC on PD pathology was weakened due to avoidance limiting the effects. However, AP only partially mediated the relationships between the VC and HA modes and PD-pathology, meaning that improvements in the VC and HA modes can at least partially directly influence PD-pathology. Theoretically, it could be argued that the Detached Protector mode (DP) should also be highly relevant to change in PD-severity, as the DP mode blocks access to the VC during treatment, while the AP implies situational and interpersonal avoidance (Arntz & Jacob, 2012). One possible reason for the AP mode dominating DP in the backward elimination process could be the relatively large proportion of participants in our sample with a primary diagnosis of Avoidant PD (50.9%). With other samples, different coping modes than AP might be found to be significant.

So far we have labeled SMs *mechanisms of change*. It is good to critically evaluate the degree to which this is justified. Using Kazdin's (2007, 2009) requirements for demonstrating mediators and mechanisms of change, the evidence can be summarized as follows:

(1). *Strong association.* We indeed found associations between treatment, mediators, and outcome. However, it should be noted that the RCT did not have an inactive control group, thus the evidence is limited in the sense that it is based on time-lagged relationships, not on between condition differences. Moreover, the strengths of the associations was modest. Whether strong associations (in the sense of large effect sizes) will ever be found in the field of treatment of PDs is, in our view, questionable.
(2). *Specificity*. Among the many schema modes, the contributions of the VC and HA modes turned out to be specific.
(3). *Consistency*. With one exception, the findings were consistent over 3 types of treatment. Nevertheless, replication is needed.
(4). *Experimental manipulation.* Although the claim is that ST explicitly addresses SMs, a clinical trial like that by Bamelis et al. (2014) will generally not be viewed as a direct experimental manipulation of the SMs. Testing whether direct manipulation of VC and HA modes leads to reduction of PD-pathology will be a challenge, given the definition of PDs, which makes assessment of immediate, short-term changes virtually impossible.
(5). *Time line.* As the current study used time-lagged variables, and tested reverse time-relationships, this criterion is clearly met: the hypothesized mechanisms preceded the outcome, and not the other way around.
(6). *Gradient.* Using linear models, the results support the requirement that larger changes in the mechanism should be related to larger changes in the clinical outcome.
(7). *Plausibility or coherence.* The hypothesized mechanisms were derived from the theory underlying ST and had the expected effects. Moreover, in a more general view, it is plausible that reductions of the maladaptive emotional-cognitive-behavioral state associated with adverse childhood experiences as well as strengthening healthy attitudes and behaviors lead to reduction of PD-pathology.

It can be concluded that five requirements were met to a reasonable degree. Requirement (1) was not unequivocally met as an inactive control group was missing, and requirement (4) was clearly not met. It should be noted that the requirements don't distinguish between *mediators* and *mechanisms of change*. Generally, with a mediator a variable is meant that accounts statistically for the change process, but does not (necessarily) explain the change, while with a mechanism of change a process is meant that is responsible for the change (Kazdin, 2007, 2009; Kraemer et al., 2002). Whether one would label SMs mechanisms of change might thus depend on the level of explanation one is looking for. Whereas for many clinicians and clinical researchers SMs might be mechanisms of change, fundamental researchers might want to see the mechanisms that explain the changes underlying the changes in SMs, such as learning and memory processes, brain mechanisms, and molecular processes. We started from the theoretical framework of ST and within that framework hypothesized SMs as mechanisms of change. This does not contradict that change processes on more fundamental levels play a role.

Although SMs have become increasingly popular for theoretical formulations of PDs in clinical practice (Lobbestael et al., 2007), to the best of our knowledge, this is the first time SMs have been tested as a mechanism of change in a randomized controlled trial. For this reason, replication of the present findings, especially with the trials that monitor treatment integrity to ensure the quality of interventions that are specific to the treatment approaches is necessary. Furthermore, some limitations should be kept in mind while interpreting our results. First, we tested temporal relationships and addressed the direction of the relationship between the VC-HA dyad and PD-Pathology. However, the SMs were not experimentally manipulated, and hence, the design of the study does not allow us to draw causal conclusions. Future studies with schema mode manipulations that specifically focus on the temporal relationships between SMs during treatment might be able to draw more robust inferences. Secondly, the data was collected using self-report measures, but the validity of assessing SMs through self-report should be approached with caution (Lobbestael, Arntz, Löbbes, & Cima, 2009; Nordahl et al., 2005; Young et al., 2003). Using other sources of information enables a more precise assessment of SMs. Thirdly, effect sizes were small (see Table 3), ranging from 0.07 to 0.11 for the ADP-IV (including the previous ADP-IV, $r = 0.08$), 0.07 to 0.14 for SOFAS (previous SOFAS, $r = 0.14$), and 0.08 to 0.16 for GAF (previous GAF, $r = 0.16$). However, we should keep in mind that the assessments are only samples of the fluctuating states of the participants, thus the analyses can only capture part of the true relationships between schema modes and PD-pathology. Moreover, the instruments have their psychometric limitations as to reliability and validity, which also limits the observable associations. Lastly, there might be other variables playing a role in the change process that are not captured by the SMI. Fourthly, only Cluster C, paranoid, histrionic, and narcissistic PDs were included in our sample, and these disorders were not equally distributed, limiting generalization of the findings to all PDs (see Bamelis et al., 2014). Fifthly, we compared schema therapy with clarification-oriented psychotherapy and treatment as usual. Further research comparing schema therapy with other evidence-based therapy approaches that are frequently mentioned in the treatment of PDs (i.e., dialectical behavioral therapy) is needed to identify universal mechanisms of change. Finally, another factor related to generalization is that the data was collected only from Dutch mental health institutions. Thus, the results might not be generalizable to cross-cultural populations. It would be interesting to explore whether the SM model for PD pathology is stable across different PDs, treatment styles and cultures other than the ones utilized in the present study.

To conclude, our study provides significant evidence that some SMs reflect common mechanisms of change in decreasing PD-related pathology and increasing healthy functioning. Healing the VC, or in broader terms, the vulnerable parts of the patient, and strengthening the HA (the healthy parts of the patient) should be the main focus in the treatment of PDs, regardless of the treatment approach. Clinically, it is also essential to address the disruptive effects of the AP, SA, and IC modes during treatment, as they appear to play an essential role in shaping subsequent PD-related psychopathology and functioning. With respect to ST, our findings provide empirical support for the recent shift towards a focus on SMs in clinical practice. SMs might be key to understanding how treatment brings about a decrease in PD severity and an increase in healthy function.

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## CRediT authorship contribution statement

**Duygu Yakın:** Conceptualization, Formal analysis, Writing - original draft, Visualization, Funding acquisition. **Raoul Grasman:** Methodology, Formal analysis, Writing - review & editing. **Arnoud Arntz:** Conceptualization, Methodology, Formal analysis, Writing - review & editing, Supervision, Funding acquisition.

## Declaration of competing interest

The authors declare no conflict of interest.

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