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Research report

Personality and cognitive vulnerability in remitted recurrently depressed patients

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ABSTRACT

Introduction: Personality disorders (PDs) have been associated with a poor prognosis of Major Depressive Disorder (MDD). The aim of the current study was to examine cognitive vulnerability (i.e., dysfunctional beliefs, extremity of beliefs, cognitive reactivity, and rumination) that might contribute to this poor prognosis of patients with PD comorbidity.

Methods: 309 outpatients with remitted recurrent MDD (SCID-I; HAM-D₁₇ ≤ 10) were included within two comparable RCTs and were assessed at baseline with the Personality Diagnostic Questionnaire-4⁺ (PDQ-4⁺), the Dysfunctional Attitude Scale Version-A (DAS-A), the Leiden Index of Depression Sensitivity (LEIDS), the Ruminative Response Scale (RRS), and the Inventory of Depressive Symptomatology-Self Report (IDS-SR).

Results: We found an indication that the PD prevalence was 49.5% in this remitted recurrently depressed sample. Having a PD (and higher levels of personality pathology) was associated with dysfunctional beliefs, cognitive reactivity, and rumination. Extreme 'black and white thinking' on the DAS was not associated with personality pathology. Brooding was only associated with a Cluster C classification ($t(308)=4.03, p < .001$) and with avoidant PD specifically ($t(308)=4.82, p < .001$), while surprisingly not with obsessive-compulsive PD.

Limitations: PDs were assessed by questionnaire and the analyses were cross-sectional in nature.

Conclusion: Being the first study to examine cognitive reactivity and rumination in patients with PD and remitted MDD, we demonstrated that even after controlling for depressive symptomatology, dysfunctional beliefs, cognitive reactivity, and rumination were associated with personality pathology. Rumination might be a pathway to relapse for patients with avoidant PD. Replication of our findings concerning cognitive vulnerability and specific PDs is necessary.

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1. Introduction

A consistent finding among patients with Major Depressive Disorder (MDD) is the high prevalence of personality disorders (PDs). Prevalence rates of PD comorbidity during MDD typically range between 40% and 80% (Friborg et al., 2014; Fournier et al., 2008; Fava et al., 2002; Hirschfeld, 1999). This wide variability can be explained by the use of different diagnostic instruments (interview or questionnaire), the diagnostic system used (DSM-III or DSM-IV) (Friborg et al., 2014), but also likely depends on the range of PDs and mood disorders included.

Few studies examined PD comorbidity prevalence after remission from MDD. Comorbid PD diagnoses appear to be low to moderately stable, and fluctuations over time have been suggested to represent the disorder itself, rather than a *mood state* effect of MDD (Costa et al., 2005; Grilo et al., 2004; Lopez-Castroman et al., 2012; Morey et al., 2010; Shea et al., 2002). However, it has been demonstrated that personality pathology is generally more stable when measured dimensionally (i.e., continuous levels of pathology; Durbin and Klein, 2006; Melartin et al., 2010; Samuel et al., 2011). There is ample evidence that having a comorbid PD is a negative prognostic factor for the course of MDD, which is reflected by a longer time to remission and increased risk of relapse up to six years (Grilo et al., 2010; Hollon et al., 2014; Skodol et al., 2011). However, the evidence is less clear for the influence of PDs on MDD treatment outcome, partly depending on design and analysis strategy (De Bolle et al., 2011; Mulder, 2006; Newton-Howes et al., 2006). MDD with PD

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comorbidity (i.e., higher scores on dimensional pathology measures) more than tripled the 10-year risk of mortality and suicide (Hansen et al., 2003), whereas the presence of a borderline PD was related to multiple instead of single suicide attempts over 10 years (Boisseau et al., 2013). Therefore, it is highly relevant to study whether modifiable cognitive vulnerability is associated with comorbid PDs, and might therefore contribute to a poor prognosis.

Within the cognitive model, latent dysfunctional beliefs (i.e., attitudes, schemas) are a potential cognitive vulnerability factor for relapse. All individuals are assumed to develop sets of beliefs about themselves and the world, based on experiences and life events (Beck, 1967). Once dysfunctional beliefs (e.g., I am worthless unless I am loved by others) become activated, they can start to dominate one's thinking and responding to situations. Negative automatic thoughts originate from the belief and in their turn trigger depressive feelings. Although several studies supported the notion that patients with higher dysfunctional beliefs are at increased risk of relapse (Bockting et al., 2006; Jarrett et al., 2012; Lewinsohn et al., 1999; Otto et al., 2007; Ten Doesschate et al., 2010), the predictive validity of schemas for the first onset of depression and the general role of schema-matching life events is less well validated (Charlton and Power, 1995; Parker et al., 2000). Patients with comorbid PDs generally endorse heightened levels of dysfunctional beliefs even in the absence of depression, which is most pronounced in Cluster C (Farabaugh et al., 2007; Ilardi and Craighead, 1999).

Besides the content of these beliefs, faulty information processing (e.g., overgeneralized thinking, selective abstraction, absolutistic thinking) as a result of the activated belief maintains the belief, and prevents disconfirming information from becoming incorporated into the cognitive structure. Therefore, it also might be the way patients think (e.g., cognitive biases) that renders them vulnerable for a recurrent course of MDD (Petersen et al., 2007; Beevers et al., 2003; Teasdale et al., 2001). Patients with borderline PD can be characterized by biased thinking, including a more negative perception of others (Sieswerda et al., 2013; Barnow et al., 2009), thought suppression (Geiger et al., 2014), overgeneralization (Van den Heuvel et al., 2012), and extreme thinking (Arntz and ten Haaf, 2012; Veen and Arntz, 2000).

Building on the cognitive model (Beck, 1967), Teasdale (1988) suggested that dysfunctional beliefs could also be activated by mild dysphoric mood in the remitted phase instead of matching life events (i.e., cognitive reactivity) to serve as a vulnerability factor for relapse in depression. Although the activation of dysfunctional beliefs by means of mood-induction has been frequently examined (e.g., Segal et al., 2006; Van Rijsbergen et al., 2013), it appears that cognitive reactivity can also be assessed using a self-report measure that instructs patients to recall how they responded during periods of mild dysphoric mood (i.e., Leiden Index of Depression Sensitivity; Van der Does, 2002). Ilardi and Craighead (1999) noted that patients with PDs are characterized by inner chronic distress, potentially serving as a natural primer to activate latent dysfunctional beliefs (i.e., cognitive reactivity). In line with this reasoning, one might expect cognitive reactivity after remission to be more strongly related to PDs than dysfunctional beliefs.

Alternatively, responding to dysphoric mood with a maladaptive repetitive focus on the causes, meaning and consequences of depressive symptoms (i.e., rumination; Nolen-Hoeksema, 1991) makes patients vulnerable for early relapse as well (Michalak et al., 2011; Nolen-Hoeksema, 2000). Especially the *brooding* component was related to the emergence of depressive symptoms (Trenor et al., 2003). In patients with acute MDD, rumination was associated with borderline PD features, but not with any specific PD ($n=257$; Abela et al., 2003; Watkins, 2009). The same was found in student samples without MDD (Baer and Sauer, 2011; Smith et al., 2006), although in

these student samples obsessive-compulsive PD features were also related to rumination (Smith et al., 2006). To our knowledge, no studies to date examined rumination in patients remitted from MDD with comorbid PDs.

The current study aims to examine potentially modifiable cognitive vulnerability after remission in patients with comorbid personality pathology (categorical as well as dimensional), and to extend findings by Ilardi and Craighead (1999) and Craighead et al. (2011). This is important since we know that patients with PDs show dysfunctional thinking even in the absence of depression. We are the first to examine a combination of cognitive vulnerability (i.e., extremity of beliefs, cognitive reactivity, and rumination) that could potentially mediate the effect of PDs on future relapse. Thereby, this study gives impetus to future prospective studies examining depression vulnerability in PD comorbidity. Moreover, current (acute) and relapse prevention psychotherapies (including CBT) might reduce cognitive vulnerability factors and thereby reduce risk of relapse. Nevertheless, differential effectiveness of relapse prevention strategies offered after remission for patients with and without PDs remains to be examined. We expected that the presence of comorbid PDs and higher levels of personality pathology (i.e., continuous) would be associated with all measured cognitive vulnerability (i.e., dysfunctional beliefs, cognitive reactivity and extremity) and rumination, and, due to the nature of the sample (i.e., remitted patients) more strongly to cognitive reactivity than to dysfunctional beliefs. When studying the classification of specific clusters, we expected dysfunctional beliefs to be related to all clusters (in line with Ilardi and Craighead (1999)). Given the mixed results for the association of specific PD clusters with extreme thinking and the absence of studies on cognitive reactivity and rumination, we explored their associations with specific PD clusters. Finally, we examined cognitive vulnerability in the three most prevalent PDs in the current sample in an exploratory fashion.

2. Methods

This study combines the baseline data of two randomized controlled trials; for readability referred to as Study A and Study B. Study A focused on Preventive Cognitive Therapy (PCT) in groups as an addition or alternative to antidepressant medication (ADM) versus ADM alone in the prevention of relapse in recurrent depression (Bockting et al., 2011a), whereas Study B studied an internet adaptation of PCT added to Treatment-As-Usual (TAU) versus TAU alone in the prevention of relapse in recurrent depression (Bockting et al., 2011b). Medical Ethical Committee for Mental Health Institutions (METiGG) approved both protocols and all patients provided written informed consent prior to participation.

2.1. Participants

In both studies, patients were included who had a) experienced at least two lifetime Major Depressive Episodes (MDEs), of which the last MDE was no longer than two years ago; b) current remission of the last MDE for at least two months, both defined according to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) and assessed with the Structured Clinical Interview for DSM-IV disorders (SCID-I; First et al., 1995) administered by trained interviewers; and c) a current score of ≤ 10 on the 17-item Hamilton Depression Rating Scale (HAM-D₁₇). Exclusion criteria were current mania, hypomania, a history of bipolar illness, any psychotic disorder (current and previous), organic brain damage, current alcohol or drug abuse, predominant anxiety disorder, and recent electroconvulsive therapy. Both studies included remitted patients, but differed to the extent that Study A only included patients who a) were currently on ADM for at least

six months, and b) did not receive psychotherapy more frequent than twice per month. In Study B, there were no restrictions with respect to both type and frequency of current care (i.e., psychotherapy, ADM, specialty care, no care).

2.2. Measures

Following inclusion and prior to receiving PCT, patients completed amongst others the following questionnaires online.

2.2.1. Dysfunctional Attitude Scale-version A (DAS-A)

In the current study, the Dutch adaptation of the DAS-A (Douma, 1991; Weissman, 1979) was used to assess rigid dysfunctional beliefs. An exemplary item is “People will probably think less of me if I make a mistake”. On the DAS-A patients rated their agreement with each item on a seven-point scale that ranged from ‘totally agree’ to ‘totally disagree’. The total number of extreme responses on the DAS (i.e., the sum of items endorsed on either the low or high end of the seven-point scale) was used as a measure of extreme response style on the DAS, similar to Beevers et al. (2003) and Jacobs et al. (2010). Lessor change in extreme responding over treatment predicted earlier return of depressive symptoms (Beevers et al., 2003). The DAS-A demonstrated excellent reliability in a previous study ($\alpha=.86$; Dozois et al., 2003), and had a reliability of $\alpha=.93$ in the current study.

2.2.2. Leiden Index of Depression Sensitivity (LEIDS)

The LEIDS is a self-report questionnaire that aims to measure cognitive reactivity to sad mood independent of mood induction (Van der Does, 2002). After imagining a mildly depressed mood, patients rated all 34-items on a scale that ranged from one ‘not applicable’ to five ‘strongly applicable’. An exemplary item is “When I feel sad, I feel I can afford less mistakes”. The LEIDS was found to be significantly associated ($r=.43$) with changes in dysfunctional beliefs following mood induction (Van der Does, 2002). Cronbach’s alpha in the current study was .87.

2.2.3. Ruminative Response Scale (RRS)

Rumination was assessed using the validated Dutch adaptation of the RRS, the RRS-NL (Raes and Hermans, 2007). An exemplary item is: “I think about how lonely I feel”. Patients rated their agreement on a scale that ranged from ‘almost always’ to ‘almost never’. The five-item subscale *brooding* was used, as this aspect of rumination appears to specifically reflect dysfunctional and maladaptive thinking and is strongly related to depression later in time (Trenor et al., 2003). In the current study, Cronbach’s alpha for the total RRS was .94, and .64 for the *brooding* subscale.

2.2.4. Personality Diagnostic Questionnaire 4+ (PDQ-4+)

The PDQ-4+ (Hyler, 1994) is a self-report personality questionnaire with 99 true/false items that directly correspond to personality disorders in the DSM-IV (American Psychiatric Association, 2000). Moreover, the total PDQ-4+ score reflects overall continuous personality pathology. The internal consistency of the PDQ-4+ ranged between .49 and .75 in a previous study (Hopwood et al., 2012). For internal consistencies in the current study, see Table 2. Lower internal consistencies of the PDQ-4+ have been attributed to the nature of PDs itself (Carr and Francis, 2010; McHoskey, 2001). A known limitation of the PDQ is its risk of false positives (Hyler et al., 1990). To further improve diagnostic accuracy, the threshold for diagnosing a personality disorder was increased by raising the number of criteria required for each disorder by one, which increased diagnostic power and higher agreement between the PDQ-4+ and the SCID-II in a previous study (Van Velzen et al., 1999).

2.2.5. Inventory of Depressive Symptomatology-Self Report (IDS-SR)

The Dutch translation of the 30-item IDS-SR (Rush et al., 1996) was used to assess levels of depressive symptomatology. The IDS-SR is a self-report measure on which patients rate their symptoms on a scale from zero to three. The IDS-SR rates all DSM-IV core symptom domains including mood, cognitive and psychomotor symptoms, but also commonly associated symptoms including anxiety. The IDS-SR had excellent internal consistency in a previous study ($\alpha=.92$; Rush et al., 2003). Cronbach’s alpha in the current study was .77.

2.3. Data analysis

In order to combine the baseline data from both studies, we first assessed potential differences between patient groups. Groups were compared on gender, age, age of onset, number of previous MDEs, last MDE severity, percentage of ADM use as well as clinical measures including HAM-D₁₇, DAS, LEIDS, RRS, PDQ-4+ (both continuous and categorical) and IDS-SR.

Subsequently, using SPSS version 20.0, multiple imputation by chained equations was used to account for the 8.8% of the data that were missing. The 40 imputations were combined according to Rubin’s rules (Rubin, 1987). Multiple imputation is a state-of-the-art technique, and preferred above other missing data approaches including case-wise deletion (Schafer and Graham, 2002).

As suggested, the threshold for PDQ-4+ personality disorder diagnosis was increased by one criterion for each disorder (Van Velzen et al., 1999). We then used univariate regression analysis to examine the association of personality (both continuous pathology and presence versus absence of a diagnosis) on the following dependent measures: dysfunctional beliefs, cognitive reactivity, and rumination (brooding). We also examined whether personality pathology was related to an extreme response style on the DAS-A. Because the number of extreme responses showed strong deviations from a normal distribution, we used the non-parametric Mann Whitney U test instead. Moreover, we studied whether the presence versus absence of personality Clusters (A, B or C) was specifically related to any of the dependent measures. Finally, we analyzed cognitive vulnerability in the three most prevalent PDs. In all models we checked whether residual depressive symptomatology changed the effect of personality pathology on the dependent variable by adding the IDS-SR as a covariate in the analysis.

3. Results

3.1. Preliminary analyses

In order to analyze the data of the two trials together, we first assessed differences between the two studies. Patients did not differ between trials with respect to gender, age, age of onset, number of previous MDEs, HAM-D₁₇ at inclusion, whether or not they received current psychotherapy, personality disorders (continuous pathology, absence versus presence of a disorder, absence versus presence of a cluster, total number of disorders), dysfunctional beliefs (including extreme responses; DAS), cognitive reactivity, brooding, and residual symptoms (IDS-SR) (all $p > .05$). Therefore, the baseline data of both trials was merged. However, as expected, current use of ADM was higher in Study A than in Study B (100% versus 51%, $\chi^2(1, N=307)=80.380, p < .001, \phi=.51$). Moreover, last depressive episode severity was somewhat higher in Study A than in Study B (Mann Whitney $U=8619.5; z=-3.38; p=.001, r=.19$; 36% severe versus 22% severe). Controlling for ADM use and last episode severity did not change any of the results.

Table 1
Baseline demographic and clinical characteristics (N=309).

Characteristic	n	Descriptive
Female (%)	309	68.6
Age	309	46.6 (10.6)
Married or cohabiting (%)	308	59.5
Patients on antidepressants (%)	307	68.6
Current psychotherapy (%)	255	55.5
Median previous MDEs (IQR)	309	4.0 (3.0)
Age of first onset	301	29.1 (12.7)
Severity last episode ^a	308	
Mild (%)		20.8
Moderate (%)		51.9
Severe (%)		27.3
Inclusion HAM-D ₁₇	309	3.6 (2.8)
Dysfunctional beliefs (DAS-A)	281	130.6 (31.2)
Extremity of dysfunctional beliefs (DAS-A)	281	5.7 (5.8)
Cognitive reactivity (LEIDS)	274	105.0 (16.0)
Brooding (RRS)	275	11.2 (2.8)
Depressive symptomatology (IDS-SR)	294	17.5 (10.7)
Continuous PD score (PDQ-4 ⁺)	283	24.2 (12.5)
Number of PDs (PDQ-4 ⁺)	283	1.1 (1.5)

Note. Descriptive characteristics represent mean $\pm$ SD unless stated otherwise. IQR=Interquartile Range, PD=personality disorder.

^a Last episode severity is based on the number of SCID-I depression symptoms (5 symptoms corresponds to mild, 6–7 symptoms corresponds to moderate, whereas 8–9 symptoms corresponds to severe depression).

Table 2
Frequency of PDs, average dimensional scores, and corresponding Cronbach's alphas (n=283).

Personality disorder	Frequency	Dimensional score (M $\pm$ SD)		Cronbach's alpha
		Across all items	Within PD	
Paranoid	26	44.4 (12.1)	5.5 (.65)	.66
Schizoid	14	36.6 (18.3)	5.3 (4.7)	.57
Schizotypal	17	43.8 (12.6)	6.2 (.39)	.66
Histrionic	4	42.5 (11.5)	6.0 (.0)	.40
Narcissistic	4	59.5 (16.2)	6.8 (.50)	.50
Borderline	18	49.7 (10.8)	6.8 (.94)	.64
Antisocial	1	68.0 (.0)	6.0 (.0)	.63
Avoidant	84	35.1 (11.7)	5.7 (.70)	.70
Dependent	7	50.6 (17.9)	6.6 (.79)	.65
Obsessive–compulsive	56	36.8 (12.1)	5.5 (.69)	.54
All items	283	24.2 (12.5)		.92

Table 3
Univariate regression models in the presence of a PD and continuous personality pathology on the cognitive measures (n=309).

Dependent variable	B	SE (B)	R ²	t	p	FMI
DAS-A						
PD	27.02	3.23	.19	8.37	<.001	.06
Continuous PD	1.50	.11	.40	13.42	<.001	.16
LEIDS						
PD	10.09	1.71	.11	5.90	<.001	.06
Continuous PD	.52	.07	.19	7.7	<.001	.18
Brooding						
PD	1.24	.35	.05	3.54	<.001	.20
Continuous PD	.07	.02	.09	4.38	<.001	.38

Note. FMI=Fraction Missing Information, DAS-A=Dysfunctional Attitude Scale Version A, LEIDS=Leiden Index of Depression Sensitivity.

3.2. Patients

In total 309 patients were included in both trials. Patients were predominantly female (68.6%) and were currently in remission as

Table 4
Univariate regression models in the presence of the PD clusters on the cognitive measures (n=309).

Dependent variable	B	SE (B)	R ²	t	p	FMI
DAS-A						
Cluster A	28.16	4.37	.12	6.45	<.001	.06
Cluster B	34.48	5.68	.12	6.07	<.001	.17
Cluster C	27.26	3.31	.19	8.24	<.001	.07
LEIDS						
Cluster A	8.15	2.39	.04	3.41	.001	.13
Cluster B	9.69	3.16	.04	3.07	.002	.25
Cluster C	9.90	1.75	.10	5.66	<.001	.06
Brooding						
Cluster A	.66	.47	.01	1.42	.155	.21
Cluster B	1.08	.63	.01	1.70	.090	.37
Cluster C	1.37	.34	.06	4.03	<.001	.13

Note. FMI=Fraction Missing Information, DAS-A=Dysfunctional Attitude Scale Version A, LEIDS=Leiden Index of Depression Sensitivity.

defined by the HAM-D₁₇ (M=3.6, SD=2.8) with a median of 4 previous MDEs (IQR=3.0). Table 1 presents the demographic and clinical characteristics of the complete sample.

3.3. Personality disorder prevalence

We found an indication that 49.5% of the patients in our sample had a comorbid personality disorder. Among all patients, 22.3% had one PD, 14.5% had two PDs and 12.7% had three or more PDs. Most patients had a diagnosis in Cluster C (39.6%) followed by Cluster A (16.6%) and Cluster B (8.1%). Avoidant PD was the most prevalent disorder (29.7%), followed by obsessive–compulsive PD (19.8%), and then paranoid PD (9.2%). See Table 2 for an overview of PD prevalence in the current sample.

3.4. Vulnerability for continuous and categorical personality pathology

As depicted in Table 3, both PD diagnosis and continuous pathology were significantly related to dysfunctional beliefs, cognitive reactivity and brooding. Continuous PD pathology accounted for 40% of variation in dysfunctional beliefs. The results did not change significantly when we controlled for residual depressive symptoms (see Supplemental Table 1).

Patients with a comorbid PD did not differ significantly from patients without comorbid PD on their level of extreme responses on the DAS-A (Mann Whitney U=11,770.25, z=−.18, p=.86).

3.5. Vulnerability by personality cluster

As depicted in Table 4, dysfunctional beliefs were significantly associated with the presence of a Cluster A, Cluster B, and Cluster C diagnosis. The same was true for cognitive reactivity. Most of the variance in both dysfunctional beliefs and cognitive reactivity was accounted for by having a Cluster C diagnosis, although more variance was explained in dysfunctional beliefs (R²=.19) than in cognitive reactivity (R²=.10). Only having a Cluster C diagnosis was significantly related to brooding scores. However, after controlling for residual symptoms, the presence of a Cluster A diagnosis was no longer related to cognitive reactivity. No other effects of controlling for residual symptoms were found (see Supplemental Table 2).

Finally, non-parametric tests revealed that there were no differences in extremity of thinking on the DAS-A between the presence versus absence of a PD diagnosis in Cluster A, Cluster B and/or Cluster C (all p>.10).

3.6. Vulnerability in the three most prevalent personality disorders

Finally, we assessed cognitive vulnerability in the three most prevalent PDs in the current sample (avoidant PD, obsessive-compulsive PD, and paranoid PD). Together, these three disorders comprised 72% of the total number of PDs diagnosed (231 diagnoses in total).

Being diagnosed with an avoidant PD was significantly associated with dysfunctional beliefs ($t(308)=8.10, p<.001, \text{Adj. } R^2=.18$), reactivity of these beliefs ($t(308)=5.73, p<.001, \text{Adj. } R^2=.10$), as well as brooding ($t(308)=4.82, p<.001, \text{Adj. } R^2=.08$). Similarly, paranoid PD was associated with dysfunctional beliefs ($t(308)=6.33, p<.001, \text{Adj. } R^2=.12$), and reactivity of these beliefs ($t(308)=2.91, p=.004, \text{Adj. } R^2=.03$). Although brooding was unrelated to having a Cluster A diagnosis in general, having a paranoid PD (Cluster A) was associated with brooding levels ($t(308)=2.13, p=.034, \text{Adj. } R^2=.02$). Remarkably, having an obsessive-compulsive PD was unrelated to brooding ($t(308)=1.61, p=.11$). Having an obsessive-compulsive PD was associated with higher levels of dysfunctional beliefs ($t(308)=5.30, p<.001, \text{Adj. } R^2=.09$) and cognitive reactivity ($t(308)=3.18, p=.002, \text{Adj. } R^2=.03$). After correction for residual symptoms with the IDS-SR, having a paranoid PD was no longer related to brooding ($t(308)=1.57, p=.11$) and cognitive reactivity ($t(308)=1.70, p=.09$). No other effects of controlling for residual symptoms were found.

4. Discussion

The central aim of the current study was to examine potentially modifiable cognitive vulnerability factors (i.e., dysfunctional beliefs, extremity of beliefs, cognitive reactivity, and rumination) in patients remitted from MDD with and without a comorbid PD, in order to unravel why these patients might be prone to a chronic and persistent course of MDD (e.g., Grilo et al., 2010; Hollon et al., 2014; Skodol et al., 2011). This study gives impetus to which cognitive vulnerability to examine as potential mediators of the effect of PDs on future relapse. Specifically addressing cognitive vulnerability factors might thereby reduce risk of relapse. Our findings indicate that PDs remain highly prevalent after remission from recurrent MDD (49.5% prevalence). This closely resembles findings by previous studies that reported a 48% (Personality Assessment Form; recurrent MDD sample; Pilkonis and Frank, 1988), 50% (SCID-II; primarily recurrent MDD sample; Farabaugh et al., 2007), and 51.9% (SCID-II; primarily non-recurrent MDD sample; Sato et al., 1994) PD prevalence after remission, as well as a study during the acute-phase of MDD that asked patients to recall their typical self (Fournier et al., 2008). In line with Farabaugh et al. (2007), avoidant PD, obsessive-compulsive PD, and paranoid PD were the most prevalent PDs in our remitted population.

We found that rumination was associated with both the presence of a PD and higher levels of PD pathology. A closer inspection revealed that rumination was associated with avoidant PD and not with obsessive-compulsive PD, as was also found in a student sample (Smith et al., 2006). Rumination might serve as a way of avoiding both cognitive and active problem solving, since it was found that rumination and avoidance (behavioral as well as cognitive and experiential) are associated (Cribb et al., 2006; Moulds et al., 2007). As rumination has also been linked to borderline PD dimensions in several previous studies (Smith et al., 2006; Watkins, 2009) we examined post-hoc whether this was also applicable to borderline PD in our patient group. Similar to paranoid PD, we found that borderline PD was related to rumination, however not over and above residual depressive symptomatology. This suggests that in these patients, rumination might be a reflection of depressive symptomatology instead of the PD itself. Since we assessed cluster classification in an exploratory fashion, future studies should attempt to replicate these findings. Moreover, since

effective relapse prevention interventions are available (Guidi et al., 2011; Vittengl et al., 2007), it is worthwhile to examine whether these interventions target rumination specifically.

As far as we know, this was the first study that examined cognitive reactivity in remitted patients with and without a PD. According to Beck's cognitive model applied to personality disorders (Beck and Freeman, 1990; Pretzer and Beck, 1996) and suggested by previous studies (Craighead et al., 2011; Ilardi and Craighead, 1999), we expected dysfunctional beliefs to be more easily activated in patients with PD comorbidity. We indeed found that cognitive reactivity was associated with both having a PD or higher levels of PD pathology, which suggests that PD pathology might serve as an innate primer or stressor for dysfunctional beliefs. Cognitive reactivity was associated with classification in all three PD Clusters, although the association with Cluster A disappeared after controlling for residual depressive symptoms.

Similar to our cognitive reactivity findings, and replicating previous studies (Farabaugh et al., 2007; Ilardi and Craighead, 1999), we also found that dysfunctional beliefs appear to represent an overarching cognitive vulnerability for all PD clusters. However, remarkable for a remitted population, dysfunctional beliefs showed a stronger association with PD levels (40% explained variance) than cognitive reactivity (19% explained variance). Even after controlling for residual symptomatology in our analyses, the association with dysfunctional beliefs remained the strongest. This could imply that due to the innate stress caused by the PD, the DAS itself is also a measure of cognitive reactivity in this group. Given the moderate association between the LEIDS and depressive symptomatology ($r=.30, p<.001$), comparable to the DAS, the LEIDS appears to be affected by state effects of depression as well.

Finally, we found that a dichotomous thinking style (i.e., rigid 'black and white thinking') was not specifically related to PDs or levels of PD pathology in our remitted patient group. Findings on the role of dichotomous thinking in PDs and borderline PD specifically (in the absence of MDD) have been mixed (Arntz and ten Haaf, 2012; Sieswerda et al., 2013; Veen and Arntz, 2000). A recent study demonstrated that instead of dichotomous thinking; negativistic thinking (i.e., general more negative evaluations of others) was typical for borderline PD (Sieswerda et al., 2013).

The cognitive model is not explicit about how and when early critical life events lead to an accumulation and consolidation of dysfunctional beliefs into MDD, PD or their combination (Beck and Freeman, 1990; Pretzer and Beck, 1996). It still has to be determined whether dysfunctional beliefs accumulate over time and consolidate into a PD, or whether they are a byproduct of the PD itself. The lack of differentiation in the associations of dysfunctional beliefs and cognitive reactivity with the PD clusters strongly suggests that this cognitive vulnerability might be an epiphenomenon of the PD (Craighead et al., 2011; Otto et al., 2007). Despite similar patterns, the low to moderate association ($r=.44; p<.001$) between dysfunctional beliefs (DAS) and their reactivity (LEIDS) does suggest that these questionnaires measure different constructs.

Strengths of the current study include use of a large recurrently depressed patient sample ($N=309$), relatively unaffected by state effects (i.e., depressive symptomatology) due to remission of the MDE, the use of several well-validated measures, and the examination of a combination of cognitive vulnerability including rumination on the level of both PD clusters and disorders. Several limitations should also be taken into account. First, we used a self-report instrument to diagnose PDs (i.e., the PDQ-4⁺) instead of the Semi-structured Interview for Personality Disorders (SCID-II). Although we adjusted the PDQ-4⁺ with one criterion to reduce over-diagnosis and, moreover, the prevalence of PDs in the current sample was comparable to other studies using remitted MDD samples (Farabaugh et al., 2007; Pilkonis and Frank, 1988; Sato et al., 1994), we cannot completely rule out that the PDQ-4⁺ overestimated the prevalence of PDs.

Second, some of the PDs had a low prevalence (i.e., antisocial PD, $n=1$; histrionic PD, $n=4$; and narcissistic PD, $n=4$), potentially related to our inclusion criteria (i.e., exclusion of (hypo)mania and alcohol and drug abuse); hence we were not able to examine these PDs specifically. Third, the current study used a cross-sectional approach to study vulnerability in patients with a comorbid PD. Therefore, we were unable to determine whether these cognitive vulnerabilities indeed predict a poor MDD prognosis (i.e., faster, more severe or persistent relapse) in these patients prospectively. Fourth, we cannot rule out that the concepts of PDs and cognitive vulnerability have the same underlying construct, which would explain their association. Overlapping items within the PDQ-4⁺ and the DAS could therefore also explain their association. Future studies should be attentive of this potential overlap. Finally, the inclusion criteria of our studies (i.e., highly recurrent group, absence of predominant anxiety disorder, between age of 18 and 65 years, fluency in Dutch,) might affect the generalizability of the results.

Future studies should attempt to replicate our findings on cognitive vulnerability and specific PDs. Since dysfunctional beliefs were more strongly associated with PDs after remission than cognitive reactivity, the question arises whether the assessment of mood-linked activation of dysfunctional beliefs is relevant in this specific group. Future studies should also examine whether rumination and other modifiable cognitive vulnerability mediate the effects that PDs have on time to relapse, in order to be able to better understand the mechanisms that drive relapse prevention strategies. Subsequently tailoring preventive interventions (i.e., specifically targeting rumination in Cluster C PD patients) might improve their efficacy. However, it remains to be examined whether relapse prevention strategies offered after remission have different effects on patients with and without comorbid PDs.

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Conflict of interest

None of the authors declares any conflict of interest.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.jad.2014.10.042>.

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