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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-$D_{17} \leq 10$) 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.

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 (Treynor 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-D_{17}). 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