



Research report

Cognitive vulnerability differentially predicts symptom dimensions of depression



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ABSTRACT

Background: We examined the association of cognitive vulnerability to depression with changes in homogeneous measures of depressive symptoms.

Methods: Baseline and 1-year follow-up data were obtained from 2981 participants of the Netherlands study of depression and anxiety. Multivariate regression analyses were carried out on cognitive reactivity, locus of control and implicit and explicit self-depressive associations in combination with negative life events. The purpose of this analysis was to predict changes on the mood/cognition and anxiety/arousal subscales of the inventory of depressive symptomatology - self report.

Results: Cognitive reactivity, locus of control and explicit self-depressive associations were independently associated with changes in depressive symptoms after adjustment for covariates and baseline severity (all $p < 0.01$). Negative life-events interacted with cognitive vulnerability to depression to predict depressive symptoms. Locus of control ($b_1 = 0.16$, $SE = 0.02$, $\eta^2 = 0.01$; $b_2 = 0.10$, $SE = 0.02$, $\eta^2 = 0.004$, $F = 8.69$, $p < 0.01$) and explicit self-depressive associations ($b_1 = 0.10$, $SE = 0.03$, $\eta^2 = 0.02$; $b_2 = 0.02$, $SE = 0.04$, $F = 7.50$, $p < 0.01$) were more strongly associated with the cognitive (b_1) than the somatic (b_2) symptom dimension of depression.

Limitations: The study sample is over-inclusive of depressed patients. Therefore it might be problematic generalizing the findings to the general population.

Conclusion: Cognitive etiological factors may play a role in a “cognitive” subtype of depression. The findings strengthen the notion that homogeneous measures of depressive symptoms enable a greater degree of discrimination between subtypes than a multidimensional conception of depression.

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1. Introduction

Major depressive disorder (MDD) causes a major burden for modern society and it is predicted that the burden of MDD will be the largest of all diseases by 2030 (World Health Organization, 2008). In recent years, pharmacological (Kirsch et al., 2008) and psychotherapeutic (Cuijpers et al., 2010) interventions have reported disappointing results. The complex and heterogeneous nature of the construct major depression may contribute to these modest results (Kendler and Gardner, 1998; Lichtenberg and Belmaker, 2010; Lux and Kendler, 2010; Parker, 2005). Treatments that work for one specific individual might not work for another individual, resulting in an attenuated treatment effect. Therefore, unaccounted heterogeneity in symptoms of depression may arrest our knowledge about the etiology and the effective treatment of MDD. Heterogeneity in depressive symptoms has particularly gained attention in psychosomatic research (de Jonge, 2011). In the field of general psychology

there is a recent movement to address syndrome heterogeneity by assessing intermediate phenotypes across current diagnostic criteria (Insel and Cuthbert, 2009; Sanislow et al., 2010). Heterogeneity in depression leads to decreased clinical specificity and a loss of statistical power. Dichotomizing results in the dismissal of valuable information, which may lead to biased results (Shorter and Tyrer, 2003). A dimensional model of psychopathology resolves both issues by assuming that symptom severity follows a continuum rather than a dichotomy. Furthermore, dimensional models assume that psychopathology consists of several co-existing symptom domains, thereby allowing for multidimensionality (Watson, 2005). Accordingly, two factors of the IDS-SR have been optimized with Rasch analysis to serve as homogeneous measures of depressive symptom dimensions (Wardenaar et al., 2010). The mood/cognition subscale of the IDS-SR contains symptoms of depressed mood, affect and cognition, e.g. ‘sad mood’ (referred to in this document as the cognitive symptom dimension of depression). The anxiety/arousal subscale of the IDS-SR contains symptoms of anxiety, somatic arousal and somatic complaints, e.g. sympathetic arousal (referred to in this document as the somatic symptom dimension of depression). These homogeneous measures of depressive symptom dimensions may be useful

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to identify multiple etiological pathways that lead to depression (Parker, 2005).

Cognitive vulnerability to depression plays an important role in the etiology of MDD (Alloy et al., 1999). The concept of a negative thinking style regarding oneself, the world and the future was first introduced by Beck (1963) and had a major impact in clinical and research settings. Hereafter, several cognitive themes have been highlighted in influential theories such as: experienced control in stressful situations (helplessness theory; Abramson et al., 1978) and negative predictions about future consequences of one's behavior and a resulting negative self-image (hopelessness theory; Abramson et al., 1989). The Temple-Wisconsin cognitive vulnerability to depression project (Alloy et al., 2000) and the Oregon Adolescent Depression Project (Lewinsohn et al., 1998) have previously examined the effect of cognitive vulnerability in a longitudinal design using multiple measures, demonstrating that cognitive vulnerability is multifaceted. These measures assessed participant's explicit negative self-evaluations, attitudes and inferential style. It was not assessed whether vulnerability measures relating to implicit associations provide incremental risk in developing depressive symptoms. More importantly, it is unknown whether cognitive vulnerability to depression is differentially predictive of more homogeneous symptom dimensions of depression. Recently, Iacoviello et al., 2010 argued that prodromal and residual symptoms of depression represent the core of the disorder. These primary symptoms (e.g. sad mood, concentration loss) resemble symptoms from the cognitive symptom dimension. Therefore, cognitive vulnerability to depression is expected to be more predictive of cognitive symptoms. The diathesis-stress model of depression states that vulnerability predisposes individuals to experience psychopathology, particularly when activated by stress (Monroe and Simons, 1991). This is why cognitive vulnerability is expected to put individuals at risk for the development of depressive symptomatology when they are faced with a stressful life event.

The aims of this project are twofold: (1) to examine whether different measures of cognitive vulnerability are independently predictive of depressive symptoms and (2) to examine the impact of cognitive vulnerability on the development of homogeneous symptom dimensions of depression. We expect that: (1) all measures of cognitive vulnerability to depression are independently positively associated with the development of overall symptoms of depression; (2) these associations are moderated by negative life events; (3) the prospective association of cognitive vulnerability with the cognitive symptom dimension is significantly larger than with the somatic symptom dimension of depression.

2. Method

2.1. Study design

2.1.1. The Netherlands study of depression and anxiety

Data were derived from the baseline and 1-year follow-up assessment of The Netherlands Study of Depression and Anxiety (NESDA), an ongoing longitudinal cohort study designed to study the long term course and consequences of depression and anxiety. Baseline data were used as predictor variables and covariates; the 1-year follow-up data were used as moderators and outcome variables. A detailed description of the NESDA design and sampling procedure is provided elsewhere (Penninx et al., 2008).

2.1.2. Sample

The baseline assessment was completed by 2981 participants, of which 2455 (82%) completed the 1 year follow-up assessment. Subjects were recruited from three different settings: the

community, primary care and mental health care. At the baseline assessment 2329 participants had a lifetime depressive and/or anxiety disorder and 652 participants had no history of any depressive and/or anxiety disorder.

2.1.3. Procedure

Recruitment maintained the following inclusion and exclusion criteria: an age of 18 through 65, proficiency in the Dutch language and no diagnosis of a psychotic disorder, obsessive compulsive disorder, bipolar disorder or severe addiction disorder. The study protocol was approved by the ethical review board of each participating center. All subjects signed an informed consent before participating in the study. The baseline assessment started in September 2004 and ended in February 2007. The lifetime version of the composite international diagnostic interview was used to establish diagnoses of (current) mood and anxiety disorders. The 1-year follow-up assessment started in September 2005 and ended in February 2008.

2.2. Measures

2.2.1. Depressive symptom dimensions

The Inventory of Depressive Symptomatology Self Report (IDS-SRRush, Gullion et al., 1996) was used to assess depressive symptom dimensions at baseline and 1-year follow-up. The 1-year follow-up measures were used as outcome variables and the baseline measure were used as covariates. The IDS-SR contains all symptoms of depression as defined by the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition* (American Psychiatric Association, 2000) and symptoms commonly associated with depression. The IDS-SR has demonstrated satisfactory psychometric qualities with good internal consistency ($\alpha=0.92-0.94$), good convergent validity and high sensitivity to change in previous research (Rush et al., 1996; Trivedi et al., 2004). Principal component analysis and confirmatory factor analysis of the IDS-SR in the NESDA sample have indicated a three-factor model of which two factors (the mood/cognition factor and the anxiety/arousal factor) have been optimized with Rasch analyses to function as homogeneous measures of depressive symptoms dimensions (Wardenaar et al., 2010).

2.2.2. Cognitive symptom dimension of depression

The mood/cognition subscale of the IDS-SR comprises 11 equally-weighted items rated on a three-point scale, containing symptoms of depressed mood, affect and cognition. The sum score (range 0–22) of this subscale was used as a measure of cognitive symptoms of depression. Internal consistency was $\alpha=0.86$ at baseline and $\alpha=0.88$ at follow-up.

2.2.3. Somatic symptom dimension of depression

The anxiety/arousal subscale of the IDS-SR comprises 8 equally-weighted items rated on a three-point scale, containing symptoms of anxiety, somatic arousal and somatic complaints. The sum score (range 0–16) of this subscale was used as a measure of somatic symptoms of depression. Internal consistency was $\alpha=0.78$ at baseline and follow-up.

2.2.4. Cognitive reactivity

Cognitive reactivity is the activation of depressive cognitions during periods of low moods (Scher et al., 2005). The Leiden Index of Depression Sensitivity-Revised (LEIDS-R) is a questionnaire that intends to measure such dysfunctional attitudes during low mood without a mood-induction procedure (Van der Does and Williams, 2003; Van der Does, 2002). The LEIDS-R is a self-report questionnaire that contains 34 equally weighted items rated on a five-point scale.

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