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General, Specific and Unique Cognitive Factors Involved in Anxiety and Depressive Disorders

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Abstract Comorbidity among anxiety and depressive disorders is the rule rather than the exception. The Integrative Hierarchical Model proposes that each of these disorders contains general (common to all), specific (common to some) and unique components. However, research into this model is limited and hampered by small (clinical) sample sizes. The aim of the present study is to investigate the incremental validity of the cognitive constructs Anxiety Sensitivity, Pathological Worry and Cognitive Reactivity to sad mood over and above the personality traits neuroticism and extraversion. Symptomatic ($N = 1,111$) and remitted ($N = 834$) patients were selected from the 2,981 participants of the Netherlands Study of Depression and Anxiety (NESDA). Results revealed both specific and unique cognitive components of anxiety and depression. Across symptomatic and remitted groups, Anxiety Sensitivity was specific to social anxiety disorder and panic disorder, Aggression Reactivity was a unique component of dysthymia, and Rumination on Sadness was unique to major depressive disorder. We conclude that cognitive constructs have additional value in understanding anxiety and depressive disorders. Moreover, they prove to be more than mere epiphenomena of current disorders.

Introduction

Anxiety and depressive disorders not only have high prevalences, but also high comorbidity rates (Angst 1996; de Graaf et al. 2002; Kessler et al. 1994; Sartorius et al. 1996). This has resulted in an ongoing debate about the breakdown of disorders as postulated in DSM-IV-TR (APA 2000). There is considerable overlap between anxiety and depressive disorders, which is also expressed in a certain degree of similarity in symptomatology, etiology, vulnerability factors and therapeutic interventions.

Etiological models have followed these developments and introduced common higher order factors accounting for comorbidity. The most promising psychological model explaining the findings reported in research and matching the experiences in clinical practice is the Integrative Hierarchical Model (IHM) for anxiety and depression introduced by Mineka et al. (1998). This model, a revised version of the tripartite model (Clark and Watson 1991), has recently attracted a lot of interest (e.g., Prenoveau et al. 2010). According to this model each disorder contains a general, a specific, and a unique component. The general component refers to a factor that anxiety and depressive disorders have in common, the specific component is shared with certain disorders but not all, and the unique component is an aspect characteristic of a particular disorder differentiating it from all the others. Hence, the model addresses the comorbidity issue while still

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acknowledging the heterogeneity of the disorders (Kotov et al. 2007; Mineka et al. 1998).

In line with its predecessor—the tripartite model—the general component of IHM consists of the personality trait Neuroticism. Elevated scores across anxiety and depressive disorders have been reported numerous times and studies investigating a hierarchical structure within the emotional disorders have confirmed its position as a higher order factor (Norton and Mehta 2007; Norton et al. 2005; Sexton et al. 2003). Extraversion is another influential Big Five personality trait. In both Clark and Watson's tripartite model as well as Barlow's Three Factor Model, extraversion constitutes a component unique to depression. This position was questioned however after several studies demonstrated links with Social Anxiety Disorder (SAD) (e.g., Brown et al. 1998; Norton and Mehta 2007) and to a lesser extent Generalized Anxiety Disorder (GAD) (Norton et al. 2005). While not in line with the original hierarchical models it does fit well within the IHM where it reflects a specific component.

Potential candidates for specific or unique components can be found within the wide array of cognitive constructs which includes factors such as rumination on sadness, self-focused attention, and attentional bias to threat. Some of these constructs, like self-focused attention, were originally linked to one or several disorders but as research progressed have later been established as more general components (Ingram 1990). Others are still awaiting further clarification. Three of these cognitive constructs which, though related to trait anxiety or neuroticism, cannot be totally accounted for by these general traits are anxiety sensitivity (AS), pathological worry (PW) and cognitive reactivity to sad mood (CR) (Cox et al. 1999; Meyer et al. 1990; Reiss et al. 1986; Van der Does 2002). AS has been defined as the fear of anxiety related sensations resulting from beliefs held about their potentially harmful physical, psychological or social consequences (Reiss and McNally 1985; Reiss et al. 1986). It is viewed as a dispositional characteristic which is relatively stable over time (for an overview see p. 68 of R. A. Peterson and Plehn 1999) and has mainly been linked to panic disorder (PD). PW on the other hand is the key feature of GAD (DSM-IV-TR; APA 2000) and can be defined as an unwanted, uncontrollable, aversive cognitive activity associated with negative thoughts and emotional discomfort (Borkovec 1994). It is considered a trait like construct and is thought to share the same underlying cognitive process as rumination (Watkins 2008). Contrary to AS and PW where the main focus is on anxiety disorders, the concept of CR has so far only been investigated in relation to depression. CR is defined as the extent to which dysfunctional schemas are activated when mood decreases. It is reported to successfully distinguish euthymic individuals with and without a history of

depression with the remitted group showing consistently higher reactivity (Merens et al. 2005; Miranda et al. 1998, Moulds et al. 2008; Van der Does 2002, 2005; Williams et al. 2008). Furthermore, high CR has also shown to increase the risk of depressive relapse (Segal et al. 1999, 2006).

Although all three constructs are mainly linked to one specific form of psychopathology which suggests that they are unique factors in the IHM model, there are also studies reporting otherwise. AS for example, has besides its connection to PD also been linked to all the other anxiety disorders as well as depression (Otto et al. 1995; Rector et al. 2007; Rodriguez et al. 2004). Similar findings have been reported for PW (Chelminski and Zimmerman 2003; Starcevic 1995; Starcevic et al. 2007) suggesting its relationship with GAD is not an exclusive one. CR has thus far only been studied in relation to depressive disorders, specifically those with a history of depression, where theoretically its influence is expected to be the most pronounced.

Studies investigating the role of cognitive constructs within hierarchical models are limited. PW has been mainly studied as a measure representing a key characteristic of GAD, but GAD is more encompassing than unwanted, uncontrollable, aversive cognitive activity and this cognitive process may have a wider relevance as a predictor of GAD, but also of related disorders (Watkins 2008). To our knowledge, both PW and CR have thus far not been tested as predictors in a hierarchical model. AS on the other hand has been included in a few such studies, where it differentiated PD from other anxiety and depressive disorders (Norton and Mehta 2007; Norton et al. 2005). However, studies did not investigate the direct contributions of lower order factors of AS and were hampered by either the use of analogue or small clinical samples. Moreover, only a couple of cognitive constructs were used and outcome measures consisted of symptoms, not actual psychiatric diagnoses.

The main aim of the present study is to investigate the incremental validity of the cognitive constructs AS, PW and CR in predicting specific depressive and anxiety disorders over and above the Big Five personality traits of neuroticism and extraversion. We will investigate this question both in symptomatic patients and in participants who are in remission, hence exploring whether elevated scores on cognitive constructs merely reflect an epiphenomenon of current psychopathology or not. We will address several limitations from previous studies by using both clinical and healthy samples. Moreover, the availability of a very large sample permits us to investigate multiple personality traits and cognitive constructs simultaneously in order to investigate their relative predictive power, while also taking comorbidity into account. The

sample comes from diverse settings (community, primary care, and specialised mental health care) and includes patients in various stages of illness, enhancing generalizability.

Method

Participants and Setting

Participants were selected from the Netherlands Study of Depression and Anxiety (NESDA), an ongoing 8-year multi-site naturalistic, longitudinal cohort study including 2,981 adult subjects aged 18 through 65 years. The sample consists of 687 healthy controls, 2,294 persons with a lifetime diagnosis of depression or anxiety disorder of which 1,342 have a current diagnosis (past month). In order to be representative of those with depressive and anxiety disorders respondents in different stages of the developmental history of the disorders (normal, high familial risk, subthreshold disorders, first and recurrent episodes) and from different health care settings (community, primary care and specialized mental health care) were included. The NESDA community sample had been previously identified in two population based studies: the Netherlands Mental Health Survey and Incidence Study (NEMESIS; Bijl et al. 1998) and the Adolescents at Risk of Anxiety and Depression (ARIADNE) study (Landman-Peeters et al. 2005). The recruitment of primary care patients took place through 65 general practitioners—using a three-stage screening method—and the recruitment of psychiatric outpatients through 17 mental health care institutions.

Across recruitment settings uniform in and exclusion criteria were used. A general inclusion criterion was an age of 18 through 65 years. An exclusion criterion was a primary psychotic, obsessive compulsive, bipolar or severe addiction disorder. In addition patients who were not fluent in Dutch were excluded. A more extensive description of the rationale, method and recruitment strategy can be found elsewhere (Penninx et al. 2008).

In the current study the psychological disorders of interest are anxiety disorders SAD, GAD, PD (with or without agoraphobia), and depressive disorders dysthymia (DD) and Major Depressive Disorder (MDD). Two psychiatric groups will be investigated; patients with a diagnosis at time of assessment (current diagnosis) and patients who fulfilled criteria of the disorder of interest at some point in life but did not meet full DSM-IV criteria in the past month (remission). Using a current sample dovetails nicely with existing literature allowing results to be easily compared. The remission sample provides an opportunity to replicate the findings of the current sample and to challenge the possibility of results merely reflecting

epiphenomena of current disorders. This was further safeguarded by controlling for residual symptoms.

Measures

Within the scope of NESDA many different measures have been administered. Only the ones relevant to the present study will be discussed here.

Assessment of Psychiatric Diagnoses

Depressive disorders (DD, MDD) and anxiety disorders (SAD, GAD, PD) were diagnosed using the Composite Interview Diagnostic Instrument (CIDI-WHO lifetime version 2.1; Ter Smitten et al. 1998). The CIDI is a worldwide used instrument which classifies diagnoses according to DSM-IV criteria (APA 1994). It has shown high interrater reliability (Wittchen et al. 1991), high test–retest reliability (Wacker et al. 2006) and high validity for depressive and anxiety disorders (Farmer et al. 1987; Wittchen 1994; Wittchen et al. 1989). The CIDI was conducted by specially trained clinical research staff.

Assessment of General Factors

Neuroticism The personality trait neuroticism was measured using the NEO Five-Factor Inventory (NEO-FFI) personality questionnaire (Costa and Mccrae 1995). This 60-item questionnaire contains a subscale for Neuroticism consisting of 12-items which are scored on a five-point scale ranging from 'strongly agree' to 'strongly disagree'. Scores ranged from 1 to 5 for each item, and the total score for each domain ranged from a minimum of 12 to a maximum of 60. The psychometric characteristics are satisfactory (Costa and Mccrae 1995). In the present study internal consistency was satisfactory with $\alpha = 0.75$.

Assessment of Specific Factors

Extraversion The personality trait extraversion was measured in the same manner as neuroticism using a subscale of the NEO-FFI personality questionnaire. Internal consistency in the present study reached an adequate level with $\alpha = 0.78$.
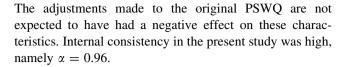
Anxiety Sensitivity Anxiety sensitivity was assessed using the Anxiety Sensitivity Index −16 items (Peterson and Reiss 1992; Reiss et al. 1986). This self-report questionnaire indicates the degree to which respondents are concerned about possible negative consequences of anxiety-related sensations. Items are scored on a 5-point Likert scale ranging from '0 = very little' to '4 = very much'. Total scores range from 0 to 64. The ASI has high levels of

internal consistency, good test–retest reliability, and good validity (R. A. Peterson and Plehn 1999; Reiss et al. 1986). Evidence from previous studies concerning the lower-order factorial structure of the ASI was nonconclusive (Cox et al. 1996; Schmidt and Joiner 2002; Vujanovic et al. 2007; Zinbarg et al. 1997). Consequently, the NESDA data were screened beforehand to determine the best fitting factor structure. Considering the large sample size of NESDA this is expected to prove a reliable method. Exploratory factor analyses presented 4 factors with eigenvalues greater than 1, of which 2 showed relatively poor internal consistency. This is in line with previous findings by Vujanovic et al. (2007) who also reported low internal consistency for 2 of the 4 factors. To maintain good internal consistency factors were combined to form 2 factors: a physical concerns factor (ASI-phc) and a social-cognitive concerns factor (ASI-scc). This was based both on theoretical and on statistical grounds. Theoretically it was most logical to combine factors of which the content was related. This theoretical solution matched the statistically emerging solution of a factor analysis with a forced 2 factor solution. The two factors explained 54.6% of the total variance in ASI scores. Items 7 and 13 were left out as both items showed very low loadings on each of the 2 factors and reliability analyses revealed that removal would improve the internal consistency. Removal of these two items did not affect the factor structure. The internal consistency of the 2 factors used in this study are adequate with $\alpha = 0.89$ for the physical concerns factor (items 3, 4, 6, 8, 9, 10, 11, 14) and $\alpha = 0.80$ for the social-cognitive concerns factor (items 1, 2, 5, 12, 15, 16). The subdivision into two factors makes AS a candidate for a unique as well as a specific factor.

Assessment of Unique Factors

Pathological Worry Pathological worry was operationalized using the Penn State Worry Questionnaire (PSWQ; Meyer et al. 1990). Items consist of statements related to worry, each rated on a 5-point Likert-scale ranging from '1 = not at all typical of me' to '5 = very typical of me'. The PSWQ consists of two subscales: a 'General worry' subscale (11 items) and a 'Not-worry' subscale (5 items) (van Rijsoort et al. 1999). The 'General worry' subscale is the strongest of the two (Brown et al. 1992; Meyer et al. 1990; van Rijsoort et al. 1999) and only this subscale was administered in the NESDA study. Psychometric properties of this Dutch 11-item version are not available but the original PSWQ has been proven to be a valid measure of trait worrying unaffected by the content of the worry (Davey 1993; Molina and Borkovec 1994) with high internal consistency, good test–retest reliability and unaffected by social desirability (Meyer et al. 1990).

The adjustments made to the original PSWQ are not expected to have had a negative effect on these characteristics. Internal consistency in the present study was high, namely $\alpha = 0.96$.

Cognitive Reactivity to Sad Mood Cognitive reactivity to sad mood was measured using the revised version of the Leiden Index of Depression Sensitivity (LEIDS-R; Van der Does 2002; Williams et al. 2008). The LEIDS-R is a self-report instrument which has been found to reliably discriminate between never-depressed and recovered depressed groups (e.g., Firk and Markus 2009; Merens et al. 2005; Moulds et al. 2008; Van der Does 2002). LEIDS-R scores also correlate with biological vulnerability markers of depression: response to acute tryptophan depletion (Booij and Van der Does 2007) and a serotonin transporter gene polymorphism (Antypa et al. 2010).

The LEIDS-R consists of 34 items divided over six subscales: Hopelessness/Suicidality Reactivity (HOP), Acceptance/Coping (ACC), Aggression Reactivity (AGG), Control/Perfectionism (CTR), Risk Avoidance (RAV) and Rumination on Sadness (RUM). Participants are asked to indicate whether and how their thinking patterns change when they experience mild dysphoria by scoring each item on a 5-point Likert-scale ranging from 0 'not at all' to 4 'very strongly' applicable to me. In the present sample internal consistencies of the subscales ranged from $\alpha = 0.62$ (ACC) to $\alpha = 0.86$ (HOP).

Covariates

Assessment of Demographic and Personal Characteristics

Detailed sociodemographic and socioeconomic data were collected, of which sex, age and number of years of education, were used in the present study.

Assessment of Symptom Levels

In order to control for residual symptoms in the remission sample, two broad and well established measures of anxiety and depressive symptomatology were used:

Anxiety The 21-item Beck Anxiety Inventory (BAI) (Beck et al. 1988) provides a reliable and valid assessment of anxiety symptomatology. In previous research it has shown high internal consistency, high test–retest reliability and good concurrent and discriminant validity (Ferguson 2000). Internal consistency in the present study was high with $\alpha = 0.95$.

Depression Depressive symptoms were measured using the Inventory of Depressive Symptomatology (IDS) a

30-item self report questionnaire. Psychometric properties are satisfactory (Rush et al. 1996). In the present study internal consistency was satisfactory with $\alpha = 0.91$.

Procedure

The study protocol was approved centrally by the Ethical Review Board of the VU University Medical Centre and subsequently by local review boards of each participating centre. NESDA subjects were assessed during a 4-h clinic visit at one of the seven field centre locations.

Once full verbal and written information about the study was given, written informed consent was obtained from all participants of the baseline assessment. Interviews were administered with computer-assisted personalized interviewing (CAPI) procedures with data entry checks on outliers and routing. When necessary the interview was taken in more than one session to avoid fatigue. Specially trained research assistants conducted the assessments. All interviews were taped to monitor data-quality and interviewer performance. In addition, the data monitoring centre routinely carried out data quality checks to review missing data and check for inconsistencies. After completion of the assessment respondents were compensated with a small incentive (gift certificate of 15 euros and payment of travel costs) for their time and cooperation.

Statistical Analyses

First scores on all measures were compared between participants meeting criteria for current disorder and participants who did not meet these criteria, as well as comparing participants with a disorder in remission to a control group with no lifetime disorders.

The ability of measures to (independently) predict an anxiety or depressive diagnosis was investigated within the total sample of participants who fulfilled criteria for a current anxiety or depressive disorder as well as in a group with a disorder in remission (cp. Gibb et al. 2007). A benefit of this analysis is that it includes a built-in psychiatric control group. Binominal logistic regression was conducted to assess this relative specificity of cognitive constructs using a hierarchical procedure. Demographics (gender, age and number of years of education) were entered as covariates by using forced entry into the model. In order to test whether the cognitive constructs would uphold their predictive value after correcting for neuroticism and extraversion, personality traits were also entered into the model using forced entry. The independent variables of interest, namely PSWQ, ASI-phc, ASI-scc and the six LEIDS-R-subscales were entered last, using a stepwise backward procedure (likelihood ratio) with a removal probability of 0.01. A stepwise backward procedure was

chosen over a stepwise forward procedure because the latter has a higher risk of Type II error due to suppressor effects (Field 2005).

Analyses were conducted for both the current diagnosis and diagnosis in remission group, with the only difference being that in the remission models BAI and IDS were added as covariates to control for residual symptoms.

A significance level of 0.01 was applied in order to control the familywise error rate and reduce the chances of a Type I error. To guard against multicollinearity the VIF score for each variable in each model was examined. No VIF statistic for any variable was found with a value above 2.7 (tolerance not below 0.37), suggesting that multicollinearity was not a problem for these regression models (Menard 1995; Myers 1990). All analyses were conducted using PASW Statistics package 17.0 (SPSS, INC, Chicago, Illinois, 2009).

Results

Sample Description

Demographic information on the sample and detailed information on the personality traits and cognitive constructs can be found in Tables 1 and 2. The NESDA sample consists of 2,981 adult subjects aged between 18 and 65 years, of which complete data including NEO-FFI, PSWQ, ASI and LEIDS-R scores were available for 2,590 participants (86.9%). The study sample mean age is 42.3 years (SD = 13.1), and 67.2% is female. Almost half (43.2%) of the participants has one or more current anxiety or depressive disorders and three-quarters (75.6%) has a lifetime anxiety or depressive disorder.

Comparison of Healthy Controls with Acute and Remitted Diagnostic Groups

When comparing participants with a current disorder ($N = 1,111$) to the group without current psychopathology ($N = 1,462$) the former group scores significantly higher ($P < 0.001$) on the personality traits (except for extraversion where the relationship is reversed) and all cognitive constructs. Cohen's d effect size is 0.33 (small effect) for LEIDS-R-acc and all others vary between 0.56 and 1.37 (large effect).

With regard to demographics, a significant difference ($P < 0.001$) for education was found, with the current disorder group completing fewer years of education ($d = 0.27$). Differences in gender and age were not significant.

Similar results were found when comparing participants with a disorder in remission ($N = 834$) to participants who had never experienced psychopathology in their lives

Table 1 Comparison of participants with a current disorder and those without current psychopathology

Current diagnosis	No current disorder (N = 1,462)	SAD (N = 462)[a]	GAD (N = 322)[a]	PD (N = 422)[a]	DD (N = 228)[a]	MDD (N = 647)[a]
Gender: female	67.4%	66.0%	64.3%	69.9%	64.0%	66.2%
Age	42.5 (13.7)	41.7 (11.9)	42.7 (11.9)	42.2 (12.0)	44.4 (11.5)	42.3 (12.2)
Education (years)	12.7 (3.2)	11.7 (3.3)	11.6 (3.3)	11.4 (3.3)	11.5 (3.5)	11.6 (3.1)
Neuroticism	31.3 (8.4)	43.8 (6.6)	44.1 (6.5)	41.6 (7.3)	44.5 (6.0)	43.1 (6.6)
Extraversion	39.8 (6.7)	31.7 (6.4)	32.3 (6.2)	34.4 (7.0)	30.5 (6.9)	32.1 (6.6)
PSWQ	25.2 (10.4)	39.2 (9.6)	42.0 (8.8)	38.3 (10.0)	41.5 (8.5)	39.5 (9.7)
ASI-phc	4.6 (4.7)	9.5 (6.8)	9.7 (6.6)	12.0 (7.0)	9.4 (6.5)	8.6 (6.5)
ASI-scc	4.8 (3.1)	9.6 (4.5)	9.2 (4.7)	9.3 (4.9)	9.5 (4.8)	8.7 (4.7)
LEIDS-R-rum	7.0 (4.7)	12.3 (4.6)	12.5 (4.8)	11.3 (5.0)	13.3 (4.2)	12.6 (4.5)
LEIDS-R-hop	2.9 (3.2)	7.9 (5.1)	8.1 (5.4)	6.9 (5.2)	9.2 (5.1)	8.2 (5.2)
LEIDS-R-acc	1.2 (1.8)	2.0 (2.3)	2.1 (2.4)	2.0 (2.5)	2.0 (2.4)	2.0 (2.4)
LEIDS-R-agg	3.5 (3.4)	7.0 (5.1)	6.7 (4.8)	6.2 (5.2)	7.8 (5.1)	6.7 (4.9)
LEIDS-R-ctr	4.6 (3.7)	7.2 (3.8)	7.0 (3.9)	6.8 (4.1)	7.2 (3.8)	6.9 (4.0)
LEIDS-R-rav	6.4 (4.4)	11.8 (4.5)	11.5 (4.6)	10.7 (4.9)	12.1 (4.5)	11.4 (4.5)
N current disorders	0.0	2.3 (1.2)	2.7 (1.1)	2.3 (1.2)	3.0 (1.1)	2.2 (1.1)

[a] Due to comorbidity participants in the disorder columns might overlap

Table 2 Comparison of participants who are in remission and those without any psychiatric history

Diagnosis in remission	No psychiatric history (N = 628)	SAD (N = 192)[a]	GAD (N = 194)[a]	PD (N = 188)[a]	DD (N = 157)[a]	MDD (N = 694)[a]
Gender: female	62.3%	69.8%	72.7%	77.1%	74.5%	71.9%
Age	41.5 (14.6)	43.7 (12.5)	43.4 (12.5)	40.4 (12.4)	46.6 (11.6)	43.2 (12.5)
Education (years)	12.8 (3.2)	12.4 (3.3)	12.5 (3.2)	12.4 (3.2)	12.2 (3.7)	12.6 (3.2)
Neuroticism	27.1 (7.4)	35.7 (8.2)	36.5 (7.7)	35.9 (7.5)	36.0 (7.3)	34.8 (7.8)
Extraversion	42.0 (6.2)	36.7 (6.5)	37.4 (6.3)	38.4 (6.7)	36.4 (6.7)	37.9 (6.7)
PSWQ	20.7 (8.8)	28.7 (10.1)	31.5 (10.4)	29.6 (10.0)	31.2 (10.3)	29.0 (10.2)
ASI-phc	3.3 (3.8)	6.2 (5.5)	6.1 (5.0)	7.6 (5.9)	6.0 (5.1)	5.4 (5.2)
ASI-scc	3.8 (2.4)	6.3 (3.5)	6.0 (3.5)	6.1 (3.7)	6.0 (3.4)	5.5 (3.3)
LEIDS-R-rum	4.8 (4.0)	9.1 (4.8)	9.3 (4.7)	9.0 (4.8)	9.6 (4.4)	8.9 (4.5)
LEIDS-R-hop	1.6 (2.3)	4.5 (3.9)	4.4 (3.7)	4.2 (3.8)	4.8 (4.0)	4.1 (3.6)
LEIDS-R-acc	0.9 (1.6)	1.4 (1.9)	1.6 (2.2)	1.5 (1.9)	1.8 (2.4)	1.5 (2.1)
LEIDS-R-agg	2.6 (2.7)	4.7 (4.0)	4.7 (3.6)	3.8 (3.3)	5.1 (3.9)	4.4 (3.7)
LEIDS-R-ctr	3.4 (3.1)	5.5 (3.8)	6.3 (4.2)	5.3 (3.3)	6.3 (4.0)	5.6 (3.9)
LEIDS-R-rav	4.4 (3.6)	8.5 (4.3)	8.9 (4.4)	8.3 (4.5)	8.7 (4.1)	8.2 (4.3)
N disorders in remission	0.0	2.4 (1.1)	2.6 (1.0)	2.4 (1.0)	2.8 (0.9)	1.8 (0.9)

Participants in remission = History of psychopathology, but none of the current diagnoses

[a] Due to comorbidity participants in the disorder columns might overlap

(N = 628), with highly significant (P < 0.001) differences for all personality traits and cognitive constructs. LEIDS-R-acc had a Cohen's d effect size of 0.29 (small effect) and all others varied between 0.50 and 0.96 (large effect). Results were all in the expected direction.

In demographics, a significant difference was observed in the gender distribution ($\varphi = -0.10$) indicating that the remission group contained a higher percentage of females. Further, there was a negligible effect (d = 0.13, P = 0.02) for age reflecting a slightly older remission sample. No difference was found for number of years of education. Demographic variables were included as covariates in all analyses, and between group differences in demographics were thereby controlled for.

Hierarchical Regression Analyses Investigating Incremental Validity

Binomial logistic regression was conducted for each of the disorders. Logistic coefficients, Wald statistic, odds ratio and 95% confidence intervals from each logistic regression analysis are presented in Tables 3, 4, 5 and 6. As shown above, psychopathology groups scored significantly higher than healthy controls (with the exception of extraversion where—as expected—an inverse relation was reported). Because the present analysis is conducted in participants with an acute disorder or a disorder in remission only (excluding healthy controls), associations in an unexpected direction merely reflect that the predictor is not so important for that particular disorder in comparison to the other disorders it is compared to. These associations indicate that the predictor is less dominant in a particular disorder and does not imply that the predictor constitutes a protective factor for that disorder. Therefore below only predictors with a significant contribution to the model in the expected direction will be discussed.

Table 3 Prediction of current anxiety disorders by personality factors and cognitive constructs, controlled for demographics

	SAD				GAD				PD			
	B	Wald test	OR	95% CI	B	Wald test	OR	95% CI	B	Wald test	OR	95% CI
Forced entry												
Sex	0.12	0.57	1.11	0.85–1.46	0.26	2.99	1.30	0.97–1.74	−0.21	1.95	0.82	0.61–1.09
Age	−0.01	1.53	0.99	0.98–1.00	0.01	2.76	1.01	1.00–1.02	−0.001	0.03	1.00	0.99–1.01
Education	−0.01	0.17	0.99	0.95–1.03	−0.03	1.31	0.98	0.94–1.02	−0.06	6.85**	0.95	0.91–0.99
Neuroticism	0.06	18.70***	1.06	1.03–1.09	0.04	7.87**	1.04	1.01–1.07	−0.02	2.90	0.98	0.96–1.00
Extraversion	−0.04	12.87***	0.96	0.94–0.98	0.00	0.04	1.00	0.98–1.03	0.03	6.43*	1.03	1.01–1.06
Stepwise backward												
PSWQ	−0.03	8.54**	0.98	0.96–0.99	0.05	32.23***	1.05	1.03–1.07				
ASI-phc									0.11	108.44***	1.12	1.10–1.14
ASI-scc	0.08	25.16***	1.08	1.05–1.12								

SAD: $R^2 = 0.13$ (Nagelkerke). Model $\chi^2(7) = 108.66$, $P < 0.001$; GAD: $R^2 = 0.12$ (Nagelkerke). Model $\chi^2(6) = 94.40$, $P < 0.001$; PD: $R^2 = 0.18$ (Nagelkerke). Model $\chi^2(6) = 156.03$, $P < 0.001$

In the stepwise backward section of the model, non-significant cognitive factors are not reported

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

Table 4 Prediction of current depressive disorders by personality factors and cognitive constructs, controlled for demographics

	DD				MDD			
	B	Wald test	OR	95% CI	B	Wald test	OR	95% CI
Forced entry								
Sex	−0.02	0.02	0.98	0.70–1.37	−0.04	0.08	0.96	0.73–1.27
Age	0.03	13.88***	1.03	1.01–1.04	0.002	0.09	1.00	0.99–1.01
Education	−0.01	0.23	0.99	0.94–1.04	−0.06	9.53**	0.94	0.90–0.98
Neuroticism	0.05	9.76**	1.05	1.02–1.08	0.01	0.48	1.01	0.98–1.03
Extraversion	−0.05	14.29***	0.95	0.92–0.98	−0.04	10.74**	0.96	0.94–0.99
Stepwise backward								
ASI-phc					−0.04	19.16***	0.96	0.94–0.98
LEIDS-R-rum					0.07	15.02***	1.07	1.03–1.11
LEIDS-R-hop					0.06	10.68**	1.06	1.02–1.10
LEIDS-R-agg	0.06	11.91**	1.06	1.03–1.09				

DD: $R^2 = 0.13$ (Nagelkerke). Model $\chi^2(6) = 91.79$, $P < 0.001$; MDD: $R^2 = 0.15$ (Nagelkerke). Model $\chi^2(8) = 133.93$, $P < 0.001$

In the stepwise backward section of the model, non-significant cognitive factors are not reported

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

Table 5 Prediction of anxiety disorders in remission by personality factors and cognitive constructs, controlled for demographics, BAI and IDS

	SAD				GAD				PD			
	B	Wald test	OR	95% CI	B	Wald test	OR	95% CI	B	Wald test	OR	95% CI
Forced entry												
Sex	0.01	0.004	1.01	0.70–1.46	−0.02	0.01	0.98	0.68–1.42	−0.13	0.42	0.88	0.58–1.31
Age	−0.001	0.01	1.00	0.99–1.01	0.01	1.79	1.01	1.00–1.02	−0.03	15.56***	0.97	0.96–0.99
Education	−0.01	0.19	0.99	0.94–1.04	0.002	0.01	1.00	0.95–1.06	−0.01	0.11	0.99	0.94–1.05
BAI	−0.004	0.07	1.00	0.96–1.03	0.01	0.66	1.01	0.98–1.05	0.04	3.93*	1.04	1.00–1.07
IDS	−0.003	0.05	1.00	0.97–1.03	−0.03	3.56	0.97	0.95–1.00	−0.01	0.75	0.99	0.96–1.02
Neuroticism	0.03	2.98	1.03	1.00–1.07	0.06	16.82***	1.07	1.03–1.10	0.02	1.23	1.02	0.99–1.05
Extraversion	−0.04	6.78**	0.96	0.93–0.99	0.001	0.01	1.00	0.97–1.03	0.01	0.69	1.01	0.98–1.05
Stepwise backward												
PSWQ	−0.04	9.61**	0.96	0.94–0.99								
ASI-phc									0.10	28.99***	1.10	1.06–1.14
ASI-scc	0.11	15.91***	1.12	1.06–1.18								
LEIDS-R-agg									−0.10	11.95***	0.91	0.86–0.96

SAD: $R^2 = 0.06$ (Nagelkerke). Model $\chi^2(9) = 35.04$, $P \leq 0.001$; GAD: $R^2 = 0.04$ (Nagelkerke). Model $\chi^2(7) = 23.53$, $P = 0.001$; PD: $R^2 = 0.13$ (Nagelkerke). Model $\chi^2(9) = 74.18$, $P < 0.001$

In the stepwise backward section of the model, non-significant cognitive factors are not reported

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

Table 6 Prediction of depressive disorders in remission by personality factors and cognitive constructs, controlled for demographics, BAI and IDS

	DD				MDD			
	B	Wald test	OR	95% CI	B	Wald test	OR	95% CI
Forced entry								
Sex	−0.38	3.18	0.68	0.45–1.04	−0.23	1.13	0.80	0.53–1.21
Age	0.03	14.25***	1.03	1.02–1.05	0.01	0.61	1.01	0.99–1.02
Education	−0.02	0.63	0.98	0.92–1.03	0.01	0.07	1.01	0.95–1.07
BAI	−0.01	0.38	0.99	0.96–1.02	−0.01	0.09	0.99	0.95–1.04
IDS	0.02	1.69	1.02	0.99–1.05	0.03	2.35	1.03	0.99–1.06
Neuroticism	0.01	0.21	1.01	0.98–1.04	0.01	0.10	1.01	0.97–1.04
Extraversion	−0.03	3.62	0.97	0.94–1.00	−0.01	0.33	0.99	0.96–1.03
Stepwise backward								
ASI-phc					−0.06	10.47**	0.94	0.90–0.98
LEIDS-R-rum					0.09	11.21***	1.09	1.04–1.15
LEIDS-R-agg	0.08	9.44**	1.08	1.03–1.13				

DD: $R^2 = 0.08$ (Nagelkerke). Model $\chi^2(8) = 44.70$, $P < 0.001$; MDD: $R^2 = 0.08$ (Nagelkerke). Model $\chi^2(9) = 38.04$, $P < 0.001$

In the stepwise backward section of the model, non-significant cognitive factors are not reported

* $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$

Prediction of Current Diagnosis by Cognitive Constructs After Correction for Personality Traits

In the first series of analyses only participants with a current diagnosis were included ($N = 1,111$). Results of the binomial logistic regressions are presented in Tables 3 and 4. These tables also show model fit and explained variance (Nagelkerke R^2) for each model.

After correction for demographics and personality traits results reveal PSWQ as a predictor of GAD, ASI-physical concerns of PD and ASI-social-cognitive concerns of SAD. Hence, indicating that these cognitive constructs possess additional predictive value over and above neuroticism and extraversion. Inspection of the LEIDS-R-subscales reveals that the hopelessness subscale (LEIDS-R-hop) predicts MDD as does the rumination subscale

(LEIDS-R-rum). Finally, the LEIDS-R-aggression is a predictor for DD.

The omnibus binomial logistic regression model indicated a significant model overall for each of the disorders (see Tables 3, 4) as well as a reasonable proportion of explained variance (Nagelkerke R^2—varied from 0.12 to 0.18). Considering the type of analysis—using a built in psychiatric control group and thus exclusion of healthy controls—these figures indicate that a reasonable proportion of the variance can be explained by each of the models.

When comparing these final models to the versions without the cognitive constructs,—only including demographics and the personality traits of neuroticism and extraversion-, all models significantly improved their fit as a result of adding the cognitive constructs. For SAD the improvement was χ^2(2, $N = 462$) = 28.20, $P < 0.001$, for GAD χ^2(1, $N = 319$) = 33.93, $P < 0.001$, for PD χ^2(1, $N = 419$) = 124.60, $P < 0.001$, for DD: χ^2(1, $N = 226$) = 11.85, $P = 0.001$ and for MDD χ^2(3, $N = 644$) = 57.95, $P < 0.001$.

Prediction of Disorders in Remission by Cognitive Constructs After Correction for Personality Traits

In contrast to the previous models that aimed to predict current diagnosis, the next models aim to investigate whether the established relationships will hold up when disorders are in remission without current diagnoses being able to dominate the relationship. In order to do so all participants with no history of depressive or anxiety disorders and all participants currently suffering from a disorder were excluded from the analyses, resulting in a sample of $N = 834$. Moreover, in order to correct for residual symptoms the covariates BAI and IDS where added to the models. Results are presented in Tables 5 and 6. These tables also show model fit and explained variance (Nagelkerke R^2) for each model.

Results for the anxiety disorders were very similar to what was reported for current diagnosis: ASI-social-cognitive concerns predicted SAD and ASI-physical concerns PD. Surprisingly PSWQ was the only exception as its contribution became non-significant in the prediction model of GAD where its influence seems to be completely overruled by neuroticism which now is a highly significant predictor.

Results for the depressive disorders show LEIDS-R-agg as predictor for DD and LEIDS-R-rum as a predictor of MDD. Most notable is the non-significant contribution of LEIDS-R-hop, which did add significantly to MDD's current disorder model.

All models are significant overall (see Tables 5, 6) although the explained variance is slightly lower than for

the current disorders. Nagelkerke varied from 0.04 to 0.13 with PD scoring at the higher and GAD at the lower end.

When comparing these final models to the versions without the cognitive constructs—only including demographics, the BAI and IDS, and the personality traits of neuroticism and extraversion—all models, except for GAD where neuroticism dominated, significantly improved their fit as a result of adding the cognitive constructs. For SAD the improvement is χ^2(2, $N = 192$) = 21.20, $P < 0.001$, for PD χ^2(2, $N = 187$) = 42.13, $P < 0.001$, DD χ^2(1, $N = 157$) = 9.20, $P < 0.01$ and for MDD χ^2(2, $N = 693$) = 20.30, $P < 0.001$.

Repeating Analyses with Comorbidity Correction

As comorbidity among anxiety and depressive disorders is very common this could have influenced the results. In order to check this, analyses for both the current and remission sample were re-run, controlling for comorbidity by adding to each model the different diagnoses (dummy coded variables: absent/present) as covariates (data not shown). Hence, in each sample five binomial logistic regression analyses were run. The models consisted of demographics, personality traits and the different diagnoses with the exception of the index disorder (forced entry), and the cognitive variables (stepwise backward procedure). The same was done for the remission sample with the addition of BAI and IDS (forced entry) to control for residual symptoms.

Compared to the analyses without comorbidity correction, results in the current diagnosis sample showed only a minor difference in the model of DD: neuroticism no longer made a significant contribution. The models on disorders in remission showed no differences. Overall the results are very similar to the analyses without correction for comorbidity, with the same significant cognitive predictors still present in the models, indicating that the reported results are very robust and not critically confounded by comorbidity.

Discussion

The aim of this study was to investigate the incremental validity of the cognitive constructs AS, PW and CR in predicting depressive and anxiety disorders over and above the personality traits of neuroticism and extraversion, while controlling for sociodemographic characteristics. This was investigated in both symptomatic and remitted patients.

Results from the symptomatic group revealed that both specific and unique cognitive components exist: Anxiety Sensitivity is a specific component involved in both SAD and PD, and Pathological Worry a unique component for

GAD. Within the depressive disorders, Aggression Reactivity (LEIDS-R-agg) is unique for DD, and Rumination on Sadness (LEIDS-R-rum) and Hopelessness Reactivity (LEIDS-R-hop) are unique factors in MDD. As expected, odds ratios were small, but when considering that we applied a stringent test by using a psychiatric control group, these results are noteworthy and robust.

The subscales of AS have unique aspects; social-cognitive concerns (ASI-scc) is solely related to SAD and physical concerns (ASI-phc) to PD. At a higher-order level both factors capture the same underlying mechanism—the fear of anxiety related sensations. The involvement of AS in both PD and SAD was no surprise, as misinterpretation of bodily sensations and maladaptive cognitions such as the fear to lose control are well known to play a prominent role in these disorders (e.g., Clark 1988; Clark and Wells 1995).

PW is also a unique component. Note that 'uniqueness' does not mean that the construct is not involved in other disorders, but that it has a dominant role in one disorder. As pointed out by Mineka et al. (1998) "symptom specificity must be viewed in relative rather than absolute terms.". So even though PW is elevated in many disorders, it has a more defining role in GAD, which is in line with both cognitive models and the DSM classification (APA 2000). Two other unique components are Rumination on Sadness and Hopelessness Reactivity, which are both linked to MDD. This relationship is unique in the sense that their predictive value of MDD is over and above that of personality traits and anxiety constructs, and remains limited to this one disorder. Further, this finding is consistent with previous research highlighting the core role of rumination and hopelessness in maintaining and predicting depression (e.g., Alloy et al. 1999; Nolen-Hoeksema 2000).

Contrary to the above findings, the relationship between DD and Aggression Reactivity (LEIDS-R-agg) was somewhat unexpected. Irritability is a common symptom of DD but has not been investigated as a (cognitive) vulnerability factor. For example, Fava et al. (1997) reported increased anger attacks among people with dysthymia or atypical depression compared to normal controls. More recently, irritability was examined as a potential subtype of MDD in the general population and it was found that the presence of irritability (vs. its absence) in MDD is associated with higher comorbid dysthymia and lifetime persistence of symptoms (Fava et al. 2010). Further, a recent large-scale longitudinal study showed that irritability during adolescence predicts an adult diagnosis of dysthymia, and to a lesser extent GAD and MDD, over a period of 20 years (Stringaris et al. 2009). The present finding expands on this literature by showing a unique association between aggression reactivity and dysthymia, but not major depression. It is noteworthy that dysthymia was also related to lower extraversion in our sample. At first glance this

pattern of results is suggestive of an introvert profile characterizing the dysthymic patient consistent with older psychoanalytic theories focusing on the central role of anger in depressive disorders. In this orientation difficulties with the expression of anger are thought to cause intra-psychic conflicts that lead to anger being directed inwards (Busch 2009). As a result, defence mechanisms, such as passive aggression, are triggered and maintained in dysthymic patients (Bloch et al. 1993). It should be noted however, that aggression reactivity is conscious, self-reported aggression.

The findings found in acute patients also appeared in the remission group, and the similarities are striking. With the exception of LEIDS-R-hop and PW all cognitive constructs uphold their position in the prediction models. An explanation for the non-significant contribution of LEIDS-R-hop to MDD can be found in a recently conducted study. This study showed that only remitted depressed participants with a history of suicidal ideation during their prior depressive episode are likely to experience high hopelessness reactivity scores during remission, whereas participants without such history do not show elevated hopelessness (Antypa et al. 2010). Consequently, the level of LEIDS-R-hop during remission seems to be dependent upon prior suicidal ideation status. This implies that although hopelessness was not represented in the overall MDD remission model it could still be a unique component for a specific subgroup. Another cognitive predictor conspicuous by its absence is PW. In the remission model of GAD, PW was no longer a significant predictor. This is most likely due to the high shared variance with neuroticism (e.g., Wells 1994). The cognitive predictors that continued to make a significant contribution to the remission models are AS (specific component: SAD and PD), LEIDS-R-agg (unique component: DD) and LEIDS-R-rum (unique component: MDD). The fact that these constructs uphold their position in the remission models, even when corrected for current symptomatology, supports the idea that these constructs are not merely epiphenomena of current disorders. Whether the results reflect scarring or more stable vulnerability factors cannot be derived from the present data.

The present study has several strengths such as the large (clinical) sample size, the recruitment from diverse settings, the replication in a remission group and the inclusion of several affective disorders and cognitive constructs.

There are also some limitations. The cross-sectional design limits insight into the direction of relationships. This was partly circumvented by also investigating remitted patients, however longitudinal data are needed to investigate cause and effect relationships. Another limitation lies in the selection of cognitive constructs. There are many other cognitive constructs which would have been

interesting to include, such as intolerance of uncertainty, and experiential avoidance. Thirdly, the self-report nature of some of the instruments used in the present study has obvious downsides. Most of these measures are well established and accepted in research, however. The only exception is the LEIDS-R, which has a relatively short history. Although the present golden standard for CR measurement involves a mood induction (Scher et al. 2005), this has also its drawbacks and the success rates of inducing sadness also varies. Furthermore, as mentioned above, the support for the LEIDS-R as a valid measure of CR is accumulating (e.g., Moulds et al. 2008). Finally, the categorical diagnosis/no-diagnosis approach is necessarily accompanied by the issue of subsyndromal symptoms. In the remission sample the BAI and IDS were added as control variables in order to correct for residual symptoms. These measures however, do not cover the entire symptomatology spectrum of anxiety in particular and thus the presence of some residual (anxiety) symptoms cannot be excluded. The current diagnosis sample did not allow for a similar approach to correct for subsyndromal (comorbid) symptoms as this would have corrected for the severity of the current index disorder itself and hence analyses would have provided non-informative results. Therefore it cannot be excluded that subsyndromal comorbid symptoms might account for some of the associations of current disorders with global or specific cognitive factors. Overall the findings support the Integrative Hierarchical Model. Moreover, when considering the additional value of cognitive constructs in understanding anxiety and depressive disorders, one can conclude that reliance on a few general measures does not do justice to the complexity of these psychopathologies. On a more practical level the results imply that although a patient might no longer meet (symptomatic) criteria of a disorder, cognitive mind sets might still be latently present, increasing the risk for relapse.

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