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Research paper

Negative memory bias as a transdiagnostic cognitive marker for depression symptom severity

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A B S T R A C T

Background: Negative memory bias is a strong risk factor for the development and maintenance of depression. Recent evidence also found negative memory bias in other mental disorders. Here, we aim to: 1) assess the presence and strength of negative memory bias in a range of (comorbid) mental disorders, 2) investigate which disorder-specific symptoms are associated with negative memory bias, and 3) test whether negative memory bias might be a transdiagnostic mechanism.

Methods: Negative memory bias was measured in patients with at least one diagnosis of a stress-related disorder (n = 86), a neurodevelopmental disorder (n = 53), or both (n = 68), and 51 controls. Depression, anxiety, attention-deficit/hyperactivity disorder, and autism spectrum disorder symptom severity was assessed using questionnaires. Groups were compared on negative memory bias and the associations between negative memory bias and symptom severity were made using linear regression models.

Results: All patient groups showed stronger negative memory bias than the controls. Negative memory bias was individually associated with all symptom severity indices, but when added into a single model, only the association with depressive symptom severity remained. This persisted after controlling for diagnostic group.

Limitations: Due to the cross-sectional sectional study design, we could only look at the associations between negative memory bias and disorder-specific symptoms and not at the direction of the effects.

Conclusions: Negative memory bias is characteristic of a depressotypic processing style and present in different mental disorders. It might play a mechanistic role in the development of (subclinical) co-occurrence between mental disorders.

1. Introduction

The prevalence of mental disorders is high; approximately 46% of the general population will be affected by a mental disorder at some point during their life (Kessler *et al.*, 2005). Moreover, co-occurrence of different mental disorders is more the rule than the exception: about 28% of psychiatric patients is diagnosed with two or more mental disorders and 17% of patients meet the criteria for three or more diagnoses (Kessler *et al.*, 2005). The most common mental disorders can roughly be categorised into two groups. Attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) are disorders that develop in early childhood and frequently persist into adulthood and are therefore considered neurodevelopmental disorders (Thapar *et al.*, 2017). Mood, anxiety, and substance use disorders are considered stress-related disorders, because they are all associated with an atypical response to stress (Sharma *et al.*, 2016).

There is high, but not well-defined, comorbidity within and between these categories. For example, studies have shown that the comorbidity between ADHD and ASD ranges between 14-78% (Gargaro *et al.*, 2011), for depression and anxiety disorders this is 40-80% (De Graaf *et al.*, 2003), and for ASD and depression this is 53-77% (Hofvander *et al.*, 2009; Joshi *et al.*, 2013). This suggests that different mental disorders might have common causes (Harkin *et al.*, 2016). Given this high prevalence and comorbidity, as well as the consequential personal and societal burden of mental disorders (Vigo *et al.*, 2016), it is important to identify mechanisms involved in the development and maintenance of different mental disorders.

In this study, we examined negatively biased memory processing as a potential shared underlying mechanism in both stress-related and neurodevelopmental disorders. Negative cognitive biases are defined as the involuntary, preferential processing of negative information and occur in different cognitive domains, such as attention, interpretation, and memory (Gotlib and Joormann, 2010). These negative cognitive biases have most frequently been studied in depressed individuals

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(Beck and Bredemeier, 2016; Joormann and Stanton, 2016; LeMoult and Gotlib, 2019; Miskowaik and Carvalho, 2014) and have been associated with greater depressive symptom severity and increased risk of relapse (Johnson *et al.*, 2007). Beck's original cognitive model of depression explains the link between negative cognitive biases and depression (Beck, 1974; Beck, 2008). According to this model, adverse experiences in childhood lead to the development of dysfunctional cognitive schemas, which form the core of cognitive vulnerability. This cognitive vulnerability is a latent characteristic that can be activated by stressful events and contributes to the development of mental disorders.

In depression, the most stable type of negative cognitive bias is memory bias (Gotlib and Krasnoperova, 1998; Gupta and Kar, 2012; Marchetti *et al.*, 2018; Matt *et al.*, 1992; Watkins *et al.*, 1996), meaning that negative information is recalled better and more frequently than neutral or positive information (Gotlib and Joormann, 2010). This is especially true for self-relevant information such as self-descriptive adjectives (Benau *et al.*, 2019; Del Valle and Mateos, 2018; Matt *et al.*, 1992; Symons and Johnson, 1997). Negative memory bias is considered an important risk factor for the development and maintenance (Hamilton and Gotlib, 2008) as well as the recurrence (LeMoult *et al.*, 2016) of depression (Bradley *et al.*, 1995; Bradley and Mathews, 1983; Harmer *et al.*, 2009; Matt *et al.*, 1992; Vrijsen *et al.*, 2014).

Although mostly studied in depression, negative memory bias is also present in other mental disorders, such as anxiety disorders (Coles and Heimberg, 2002; Kalenzaga and Jouhaud, 2018), eating disorders (Nikendei *et al.*, 2008), ASD (Henderson *et al.*, 2009; Freeth *et al.*, 2010), and substance use disorder (Wiers *et al.*, 2015), and it has also been related to ADHD symptom severity (Vrijsen et al., 2018b). In addition, Vrijsen and colleagues (2017) found that a stronger negative memory bias is associated with a higher number of current mental disorders. This implicates that negative memory bias can be a transdiagnostic marker and possible mechanism involved in the development of different mental disorders and their comorbidity.

So far, the majority of studies on negative memory bias have focused on single mental disorders, defined according to the classic International Classification of Diseases (ICD) or Diagnostic and Statistical Manual of Mental Disorders (DSM) diagnostic systems. In order to be able to translate research findings into targeted treatments, it is important to look beyond the traditional classifications and explicitly focus on possible common causes and causes of comorbidity (Curthbert and Insel, 2013; Insel, 2014; Watkins *et al.*, 2015). In order to do so, we require more research in large, naturalistic patient samples.

Therefore, we recruited a heterogenic, naturalistic psychiatric patient sample. Comorbidity was allowed and assessed and we did not sample on specific disorders (see Methods). This sample consists of patients with one or more stress-related disorders (mood, anxiety, and substance use disorders) and/or neurodevelopmental disorders (ADHD and ASD), as well as a healthy control group. The three aims of our exploratory study were: 1) to assess the presence and strength of negative memory bias in different mental disorders, 2) to investigate the associations between different disorder-specific symptom severity indices (depression, anxiety, ADHD, and ASD) and negative memory bias, and 3) to investigate whether negative memory bias is related to disorder-specific symptom severity above and beyond diagnostic classifications and depressive symptoms.

First we examined if negative memory bias was also present in neurodevelopmental disorders. If that would be the case, there would be evidence for its transdiagnostic nature. Patients were divided into three different groups based on their clinical classification: a group of patients with only stress-related disorders, a group of patients with only neurodevelopmental disorders, and a group of patients with both types of disorders. This allowed us to study negative memory bias in comorbid disorders whilst still keeping in line with the current research tradition of categorising disorders based on shared characteristics and underlying (genetic) mechanisms. For the second and third research aims, we examined negative memory bias across the classifications – hence using a transdiagnostic approach – by examining the association between negative memory bias strength and disorder-specific symptom levels in all patients.

2. Methods

2.1. Participants

This study was part of a naturalistic psychiatric cohort called MIND-Set (Measuring Integrated Novel Dimensions in Neurodevelopmental and Stress-related mental disorders). The aim of the MIND-Set cohort is to gain a better understanding of unique and shared mechanisms in stress-related and neurodevelopmental mental disorders by studying these disorders on different biological, neurocognitive, and behavioural levels using the same methodology. MIND-Set was initiated by the Department of Psychiatry of the Radboud University Medical Centre and the Donders Institute for Brain, Cognition, and Behaviour in Nijmegen, The Netherlands.

All patients at the outpatient clinic of the Department of Psychiatry who were 18 years or older and with a clinical diagnosis of a current mood disorder and/or anxiety disorder and/or substance use disorder and/or ADHD and/or ASD were eligible to participate. Patients with a current psychosis, sensorimotor handicaps, inadequate command of the Dutch language, a full-scale IQ estimate of below 70, and/or mental incompetence to sign the informed consent form were excluded from participation.

To answer the research aims as formulated in the introduction, patients (N = 207) were divided into three groups based on their diagnoses: 1) a stress-related disorders group (SR group, n = 86), only consisting of patients with one or more mood and/or anxiety and/or substance use disorders, 2) a neurodevelopmental disorders group (ND group, n = 53), only consisting of patients with ADHD and/or ASD, and 3) a comorbid group (CM group, n = 68), consisting of patients with at least one stress-related and at least one neurodevelopmental disorder. See Table 1 for an overview of the (comorbid) diagnoses in each group.

In addition, a control group (n = 51) was included, consisting of individuals without a current or past mental disorder. The control participants were matched to the patients in terms of gender identification, age, and educational level. The absence of mental disorders was assessed through a telephonic screening interview, using the same diagnostic instruments as for the patients (see below). The total patient group and control group did not differ significantly in gender identification ($\chi^2(1) = 2.74$, $p = .098$), age ($t(256) = 0.67$, $p = .504$), or education level ($\chi^2(3) = 2.71$, $p = .438$). The SR, ND, CM, and control group also did not differ significantly in gender identification ($\chi^2(3) = 2.82$, $p = .420$) or education level ($\chi^2(9) = 11.37$, $p = .251$), but they did differ significantly in age ($F(3) = 3.20$, $p = .024$). Control participants received a monetary compensation of €66 for participation. Written informed consent was obtained from all participants. The MIND-Set study was approved by the local ethical committee ('Commissie Mensgebonden Onderzoek Arnhem-Nijmegen') in July 2017.

2.2. Diagnostic procedure

Patients were diagnosed by trained clinicians. Mood and anxiety disorders were diagnosed with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 2002). Substance use disorder was diagnosed according to the DSM-IV with the Measurements in the Additions for Triage and Evaluation and Criminality (MATE-Crimi, subsections 1, 3, 4, 9, and Q1; Schippers et al., 2011). ADHD was diagnosed according to the DSM-IV with the Diagnostic Interview for Adult ADHD (DIVA; Kooij, 2010), and ASD was diagnosed using the Dutch Interview for Diagnosing Autism Spectrum Disorders (in Dutch: 'Nederlands Interview ten behoeve van Diagnostiek

Table 1
Overview of the (comorbid) disorders, gender identification, age, education level, and mean depressive (IDS-SR), anxiety (ASI), ADHD (CAARS), and ASD (AQ-50) symptom severity scores in the stress-related (SR), neurodevelopmental (ND), comorbid (CM), and control group. The mean number of positive and negative endorsed and recalled words that were used to calculate the negative memory bias index score are also presented for each group.

	SR	ND	CM	Controls
Stress-related disorders (n)	86	-	-	-
Mood disorder (n)	43	-	-	-
Anxiety disorder (n)	9	-	-	-
Substance use disorder (n)	1	-	-	-
Mood and anxiety disorder (n)	18	-	-	-
Mood and substance use disorder (n)	10	-	-	-
Anxiety and substance use disorder (n)	1	-	-	-
Mood, anxiety, and substance use disorder (n)	4	-	-	-
Neurodevelopmental disorders (n)	-	53	-	-
ADHD (n)	-	27	-	-
ASD (n)	-	17	-	-
ADHD and ASD (n)	-	9	-	-
Stress-related and neurodevelopmental disorders (n)	-	-	68	-
Mood disorder and ADHD (n)	-	-	7	-
Mood disorder and ASD (n)	-	-	3	-
Anxiety disorder and ADHD (n)	-	-	6	-
Anxiety disorder and ASD (n)	-	-	9	-
Substance use disorder and ADHD (n)	-	-	6	-
Substance use disorder and ASD (n)	-	-	1	-
Mood and anxiety disorder and ADHD (n)	-	-	2	-
Mood and anxiety disorder and ASD (n)	-	-	8	-
Mood and substance use disorder and ADHD (n)	-	-	5	-
Anxiety and substance use disorder and ADHD (n)	-	-	1	-
Mood, anxiety, and substance use disorder and ADHD (n)	-	-	4	-
Mood and substance use disorder, ADHD and ASD (n)	-	-	2	-
Mood disorder, ADHD, and ASD (n)	-	-	5	-
Anxiety disorder, ADHD, and ASD (n)	-	-	2	-
Mood and anxiety disorder, ADHD, and ASD (n)	-	-	4	-
Controls (n)	-	-	-	51
Gender (% female)	44%	45%	43%	57%
Mean age (SD)	45 (14.8)	40 (12.4)	38 (12.5)	40 (15.3)
Education level[a]				
None	4%	4%	6%	0%
Low	11%	10%	16%	10%
Medium	32%	44%	48%	44%
High	53%	42%	30%	46%
Mean IDS-SR score (SD)	40 (13.7)	24 (9.5)	35 (10.8)	5 (4.4)
Mean ASI score (SD)	18 (10.3)	13 (8.9)	16 (8.9)	7 (4.9)
Mean CAARS score (SD)	38 (12.9)	39 (12.5)	43 (10.0)	11 (7.7)
Mean AQ-50 score (SD)	118 (18.0)	123 (25.6)	131 (19.3)	94 (11.0)
Mean number positive endorsed words (SD)	4.6 (2.1)	6.1 (2.0)	5.2 (2.0)	8.7 (1.3)
Mean number negative endorsed words (SD)	3.4 (2.6)	1.4 (1.7)	2.7 (2.8)	0.1 (0.4)
Mean number positive recalled words (SD)	2.8 (1.3)	2.8 (1.5)	3.0 (1.6)	3.8 (1.6)
Mean number negative recalled words (SD)	2.5 (1.5)	2.4 (1.6)	2.5 (1.5)	2.6 (1.4)
Mean negative memory bias index score (SD)	0.34 (0.37)	0.17 (0.25)	0.25 (0.35)	0.01 (0.06)

*$p < .05$.
[a] Education level is the highest education someone finished with a diploma and is calculated conform the HELIUS study (Stronks *et al.*, 2013).

Autismespectrumstoornissen'; NIDA; Vuijk, 2016) according to the DSM-V.

2.3. Disorder-specific symptoms

All patients and control participants filled out an online self-report questionnaire that has previously been proven to be useful to assess demographics (Stronks *et al.*, 2013) as well as several other self-report questionnaires to assess different disorder-related symptoms. Depressive symptom severity was assessed using the 30-item Inventory of Depressive Symptomatology – Self Rating questionnaire (IDS-SR; Rush *et al.*, 1996), which had good internal consistency in our sample: Cronbach's $\alpha = .87$. The 16-item Anxiety Sensitivity Index questionnaire (ASI; Rodriguez *et al.*, 2004) was used as a measure for anxiety sensitivity. The ASI has adequate psychometric properties to assess an individual's concerns about the negative consequences associated with anxiety, even in individuals without an anxiety disorder (Powers *et al.*, 2016). Anxiety sensitivity has been linked to the presence (Allan et al., 2014) and development (Deacon and Abramowitz, 2006) of anxiety disorders as well as to cognitive biases (Clerkin et al., 2015). Furthermore, it is linked to the negative valence systems domain of the RDoC matrix. In our sample, the ASI had good internal consistency: $\alpha = .87$. ADHD symptom severity was assessed using the 26-item Conners' Adult ADHD Rating Scale (CAARS; Conners *et al.*, 1999) and ASD symptom severity was measured with the 50-item Autism Spectrum Quotient (AQ-50; Baron-Cohen *et al.*, 2001). They respectively showed good ($\alpha = .88$) and excellent ($\alpha = .90$) internal consistency in our sample. Higher scores on the questionnaires indicate more severe symptoms. The total score of each questionnaire was used for the data analyses.

2.4. Negative memory bias

The computerised Self-Referent Encoding Task (SRET; Derry and Kuiper, 1981) was used to assess self-referent negative memory bias (Derry and Kuiper, 1981; Dobson and Shaw, 1987). This is an implicit learning task that consists of an encoding phase followed by a recall phase. During the encoding phase, twelve positive and twelve negative

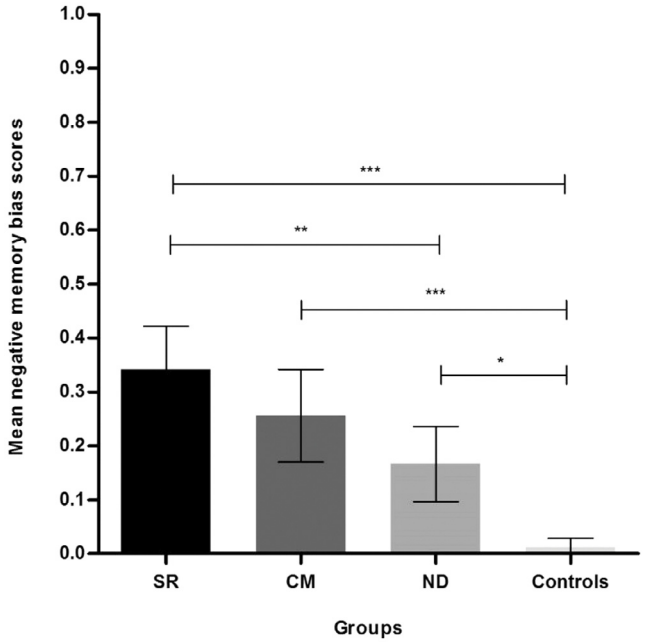

Figure 1. Mean negative memory bias scores for the stress-related (SR), comorbid (CM), neurodevelopmental (ND), and control group. The error bars show the standard error of the mean.
* $p < .05$, ** $p < .01$, *** $p < .001$.

possibly self-descriptive adjectives (Dutch translations of the Affective Norms for English Words database; ANEW; Bradley and Lang, 1999) were individually presented on a computer screen in a fixed randomised order. These words were aimed at triggering positive and negative cognitive schemas (Beck, 1987; Young, 1990). An example of a positive word is 'loving' and an example of a negative word is 'useless'. The valence of these words was confirmed by 99 independent volunteers (79% female, *M* age = 29 years, *SD* age = 15.12 years) who rated all words on a scale of 1 (*extremely negative*) to 10 (*extremely positive*). The valence of the positive words ($M = 6.02$, $SD = 0.49$) was significantly more positive than the valence of the negative words ($M = 2.69$, $SD = 0.72$), $t(98) = -34.57$, $p < .001$).

In the current study, we instructed participants to indicate how well each word described them on a five-point scale ranging from 1 (*not very well*) to 5 (*extremely well*). If a word was scored with a 4 or a 5, it was considered to be endorsed as self-descriptive. After a two-minute distraction task (Digit Symbol Substitution Task; Royer, 1971), the recall phase started. Participants were asked to type in as many words they remembered from the encoding phase (spelling mistakes were permitted and all responses that did not exactly match the presented words were checked by the experimenter).

The first 112 participants performed the task without a time restriction. To ensure that our version of the task was in line with other studies using the SRET (Gotlib *et al.*, 2004; Gerritsen *et al.*, 2012; Van Oostrom *et al.*, 2012; Vogel *et al.*, 2014; Vrijsen *et al.*, 2017, Vrijsen et al., 2018b) and since we noticed that participants typed in their answers within minutes after the start of the recall phase, we added a time restriction of three minutes to create more uniformity in the procedure. To account for primacy and recency effects, the first and last two words of the encoding phase were excluded from the task results.

In line with a broad range of studies using the SRET (Gerritsen *et al.*, 2012; Gotlib *et al.*, 2004; Van Oostrom *et al.*, 2012; Vogel *et al.*, 2014; Vrijsen *et al.*, 2017, Vrijsen et al., 2018b), a negative memory bias index score was calculated by dividing the number of endorsed and correctly recalled negative words by the total number of endorsed and recalled words. The advantage of calculating the negative memory bias index in this way, is that it controls for differences in overall rates of endorsement (Symons and Johnson, 1997).

2.5. Statistical analyses

An ANCOVA was used to test for differences in mean negative memory bias score between the SR, ND, CM, and control group whilst controlling for gender identification, age, and education level. Partial correlation analyses, controlling for age, gender identification, and education level, were used to look at the correlations between the symptom severity questionnaires. We used linear regression models to examine the associations between the different disorder-related symptom severity indices and negative memory bias. Then, we further explored these relationships by examining the associations between the disorder-specific symptom severity indices and negative memory bias whilst controlling for diagnostic category and depressive symptoms. The partial correlation and all linear regression models were only performed in patients and included gender identification, age, and education level as covariates.

3. Results

3.1. Negative memory bias across the groups

See Table 1 for an overview of gender identification, age, and education level per group. The mean number of positive and negative endorsed and recalled words per group are also presented in Table 1 as they were used to calculate the negative memory bias index score (mean scores also in Table 1).

The four groups (SR, ND, CM, and controls) differed significantly in strength of negative memory bias, $F(6, 238) = 6.70$, $p < .001$, Cohen's $f = .42$. A Tukey post-hoc test revealed that the SR group had the highest mean negative memory bias score, which was significantly higher than the ND group ($p < .01$, Cohen's $d = .54$) and the control group ($p < .001$, $d = 1.23$). Both the CM and ND groups had significantly higher negative memory bias scores than the control group ($p < .001$, $d = .94$ and $p < .05$, $d = .84$, respectively) (see Figure 1).

In order to control for the possible influence of individuals with remitted depression in the CM and ND groups on the mean negative memory bias score (LeMoult *et al.*, 2016), we repeated this analysis after removing the patients with remitted depression from the CM and ND groups. There was still a significant difference in negative memory bias strength between the four groups, $F(6,197) = 6.83$, $p < .001$, Cohen's $f = .46$. A Tukey-post hoc test revealed that the SR group still had the highest mean negative memory bias score, which was significantly higher than the ND ($p < .05$, $d = .69$), and control ($p < .001$, $d = 1.23$) groups. The CM group also had a significantly higher negative memory bias score than the control group ($p < .001$, $d = .96$). The mean negative memory bias score of the ND group was no longer significantly higher than the control group ($p = .439$, $d = .68$), which was most likely due to the smaller group size (ND group was now $n = 24$). However, the effect size was still medium and comparable to that of the SR versus ND pairwise comparison.

Considering the relatively large number of patients and control participants who did not show a negative memory bias at all (a negative memory bias score of 0), we explored if the diagnostic groups (SR $n = 47$, ND $n = 18$, and CM $n = 28$) differ on negative memory bias strength. We performed the same analysis as above again, but only including the patients with a negative memory index score larger than 0. Interestingly, there was no significant difference between the groups anymore, $F(5,80) = 1.46$, $p = .212$.

3.2. Associations between negative memory bias and disorder-specific symptom severity

The mean scores of each symptom severity questionnaire are presented in Table 1. The correlations between the symptom severity

Table 2
Results from the linear regression analyses relating IDS-SR (depressive symptoms), CAARS (ADHD symptoms), AQ-50 (ASD symptoms), and ASI (anxiety sensitivity) scores to negative memory bias.

	B	SE B	β	p-value	R^2
Model 1				< .001	.383
Constant	-.216	.112		.055	
IDS-SR	.009	.002	.359	< .001	
Model 2				.001**	.093
Constant	-.249	.133		.062	
CAARS	.007	.002	.262	< .001	
Model 3				.046*	.050
Constant	-.242	.179		.180	
AQ-50	.002	.001	.155	.030*	
Model 4				.051	.048
Constant	-.025	.113		.827	
ASI	.005	.003	.154	.035*	
Model 5				< .001	.166
Constant	-.435	.177		.015*	
IDS-SR	.008	.002	.320	< .001	
CAARS	.003	.002	.118	.146	
AQ-50	.001	.001	.069	.331	
ASI	-.002	.003	-.060	.459	

Note: gender, age, and education level were also added to each model. Education level was a significant ($p < .05$) predictor for negative memory bias in all models.
*$p < .05$, ** $p < .01$.

questionnaires were as follows: IDS-SR and AQ-50, $r = .18$, $p < .05$, IDS-SR and ASI, $r = .50$, $p < .001$, IDS-SR and CAARS, $r = .47$, $p < .001$, ASI and AQ-50, $r = .08$, $p = .252$, ASI and CAARS, $r = .40$, $p < .001$, CAARS and AQ-50, $r = .30$, $p < .001$. To assess the associations between negative memory bias and the disorder-specific symptom severity indices in the patient groups, we performed five separate linear regression analyses. In the first four models, we associated either depressive symptoms (IDS-SR score), anxiety sensitivity (ASI score), ADHD symptoms (CAARS score), or ASD symptoms (AQ-50 score) with the strength of negative memory bias. In the fifth model, we associated negative memory bias with all the symptoms. Here, the order of the symptom severity scores was based on the results from the previous correlation analyses.

The results showed that all psychiatric symptom severity indices were significantly associated with negative memory bias (Table 2, models 1-4). However, when all symptom severity scores were included in one model, only depressive symptoms were significantly associated with negative memory bias strength (Table 2, model 5).

Because only depressive symptom severity was significantly associated with negative memory bias, we looked at the distribution of depressive symptoms across the SR, CM, ND, and control group. We found that mean IDS-SR scores differed significantly between these groups, $F(6,238) = 65.32$, $p < .001$, Cohen's $f = 1.28$. The subsequent Tukey post-hoc test showed indeed that all groups differed significantly from each other, with the SR group showing the highest symptom scores, followed by the CM group, the ND group, and the control group showing the lowest depressive symptom scores (see Figure 2). The IDS-SR cut-off scores (Rush *et al.*, 1996) revealed that all patient groups (so even the ND group) had an average score indicating at least moderate depression.

3.3. Associations between negative memory bias and disorder-specific symptom severity, above and beyond diagnostic classification

To further explore the associations between the disorder-specific symptom severity indices and negative memory bias, we first assessed the relationship between negative memory bias and depressive symptoms whilst controlling for diagnostic category (SR, CM, and ND recoded into two dummy variables: CM vs. SR and ND vs. SR), which appeared significant (Table 3, model 1). We then performed three linear regression analyses to examine the relationships between the other disorder-specific symptoms and negative memory bias whilst controlling for diagnostic classification. In three additional models, we also added depressive symptom severity.

The results showed that without depressive symptoms added to the regression model, negative memory bias and ADHD symptoms, ASD symptoms, and anxiety sensitivity were significantly associated above and beyond diagnostic category (Table 3, models 2a, 3a, and 4a). However, when depressive symptoms were added, only depressive symptom severity was significantly associated with negative memory bias (Table 3, models 2b, 3b, and 4b). Negative memory bias was thus associated with depressive symptoms above and beyond diagnostic category.

4. Discussion

By examining negative bias in different mental disorders, we aimed to investigate whether negative memory bias is a potential shared neurocognitive mechanism involved in the development of (comorbidity between) different stress-related and neurodevelopmental disorders. Our first aim was to assess the presence and strength of negative memory bias in different mental disorders. We found that all patient groups showed a negative memory bias that was stronger than in the control group. Our second aim was to investigate the associations between different disorder-specific symptom severity indices and negative memory bias. We showed that while depressive symptoms, anxiety sensitivity, ADHD, and ASD symptoms were all associated with negative memory bias strength, only depressive symptoms showed a unique association with negative memory bias that was not explained by variance in the other symptom clusters. Our third aim was to investigate whether negative memory bias is related to disorder-specific symptom severity above and beyond diagnostic classifications and depressive symptoms. We found that only depressive symptoms were uniquely associated with negative memory bias regardless of the diagnostic group differences. The results thus indicate that negative memory bias is present in different mental disorders, independent of depression classification. Moreover, negative memory bias may be a transdiagnostic marker for depression, also in patients with neurodevelopmental disorders in which depression is often overlooked and difficult to diagnose (Chandrasekhar and Sikich, 2015).

Negative memory bias was present in all disorder groups, which is in line with previous findings showing that negative memory bias might be characteristic of multiple mental disorders (Coles and Heimberg, 2002; Dalgleish and Watts, 1990; Freeth *et al.*, 2010; Henderson *et al.*, 2009; Kalenzaga and Jouhaud, 2018; Mogg et al., 1987; Nikendei *et al.*, 2008; Vrijsen *et al.*, 2017; Wiers *et al.*, 2015). When only selecting patients with at least some level of negative memory bias (i.e. a negative memory bias score larger than 0), the differences between the disorder groups disappeared. This could indicate that once the positive memory bias is missing, the negative memory bias might be independent of diagnostic classification. However, it is important to note that this was a post-hoc analysis in smaller subgroups, limiting the informativeness of this result.

When we looked at the relationships between negative memory bias and the different disorder-specific symptom severity indices, we found that only depressive symptom severity, as measured with the IDS-SR, was uniquely associated with negative memory bias. This is in line with the findings of Del Valle and Mateos (2018) who showed that self-referent negative memory bias was present in individuals with subclinical depression independent of primary diagnosis. Our findings are also in line with a recent study by Beevers *et al.* (2019) who studied adults whose symptoms ranged from no symptoms of depression to clinical levels of depression. Using a similar task as we used here, they found that depression symptoms explained a large part (34-45%) of the variance in negative self-referent processing. Interestingly, this was not the

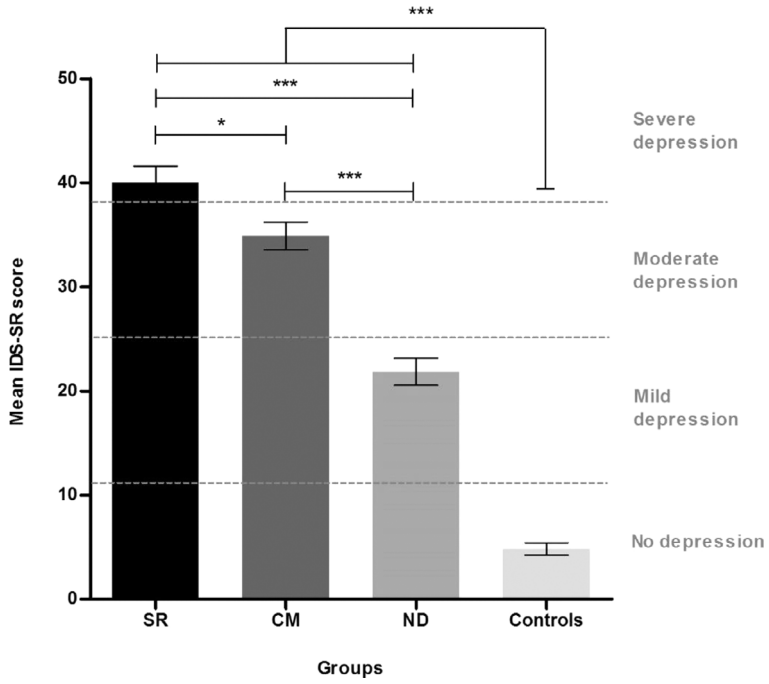

Figure 2. Mean IDS-SR scores (measuring severity of depression symptoms) for the stress-related (SR), the comorbid (CM), neurodevelopmental (ND), and control group, including the IDS-SR cut-off scores (Rush *et al.*, 1996). The error bars show the standard error of the mean.
* $p < .05$, *** $p < .001$.

case for negative attention bias, confirming the earlier notion that negative memory bias is the negative cognitive bias that is strongest related to depression and depressive symptoms (Marchetti *et al.*, 2018). Further research is necessary to find out which specific factors in the IDS-SR are associated with negative memory bias.

On the one hand, our findings confirm the already well-established relationship between negative memory bias and depression (Bradley et al., 1995; Bradley and Mathews, 1983; Harmer et al., 2009; Matt et al., 1992; LeMoult and Gotlib, 2019). On the other hand, we showed that this relationship extends beyond the depression diagnosis border. Our study therefore ties in well with recent initiatives pushing a transdiagnostic or even classification-free approach to psychiatry and mental health. Recent initiatives, such as the National Institute of Mental Health's Research Domain Criteria project (RDoC), aim for a transdiagnostic approach to discover the fundamental underlying mechanisms of psychopathology with the ultimate goal to improve personalised healthcare (Insel, 2014; Cuthbert and Insel, 2013; Watkins *et al.*, 2015).

The results from our study contribute to this step towards precision psychiatry. As Harmer and colleagues have shown, negative memory bias can be altered as a function of pharmacological treatment and these changes are visible way before subjective depression symptoms show a change (Harmer *et al.*, 2003; Harmer *et al.*, 2009; Harmer *et al.*, 2017). Hence, we suggest that negative memory bias could be used to index depressive symptoms transdiagnostically. Since norm scores are not available for the SRET, its outcome cannot be used for screening. However, negative memory bias may be used as intraindividual screening of the course of an individual's treatment in a more objective way than (self-report) questionnaires can. In addition, non-pharmacological treatments to alter negative memory bias, such as memory bias modification (Hertel and Mathews, 2011; Lang *et al.*, 2009; Vrijsen *et al.*, 2018a) and neuromodulation of memory processes, might be strong candidates for transdiagnostic treatment and prevention of comorbid depression. Although these are promising possible clinical applications, more research is required.

This study has strengths and limitations. A strength is the large, naturalistic sample, facilitating the generalisation of the findings to the clinical population. Another strength is the use of the SRET to measure negative memory bias, which is frequently used and hence allows us to compare the current findings to other memory bias results. A limitation might be that the stress-related and comorbid disorders groups only included patients with *current* mood and anxiety disorders. This means that patients with remitted depression could be included in any of the three patient groups. Patients with remitted depression and ADHD and/or ASD were hence categorised into the neurodevelopmental disorders group. Given that individuals with remitted depression often still show a negative memory bias (LeMoult *et al.*, 2016), one might consider it a possibility that the patients with remitted depression caused the high negative memory bias scores in patients in the neurodevelopmental disorders group. However, we showed that the effect sizes remained the same after removing the patients with remitted depression from the comorbid and neurodevelopmental disorders groups, meaning that remitted depression most likely did not cause the stronger negative memory bias in this group. Second, due to the cross-sectional study design, we were only able to look at the associations between negative memory bias and disorder-specific symptoms and not at the direction of the effects. To gain more insight in the mechanistic role of negative memory bias in the development of psychiatric problems across mental disorders and the comorbidity between them, a longitudinal study predicting symptom development by negative memory bias over time in a heterogeneous sample is necessary.

In conclusion, negative memory bias appears to be driven by a depressotypic processing style, not only in patients with depression, but also in patients with (comorbid) neurodevelopmental disorders. We therefore propose negative memory bias as a transdiagnostic cognitive marker for (comorbid) depression in mental disorders in general.

Author contributions

All authors have contributed to this article and concur on its content.

Table 3
Results from the linear regression analyses that examined the associations between IDS-SR (depressive symptom severity), CAARS (ADHD symptom severity), AQ-50 (ASD symptom severity), and ASI (anxiety sensitivity) and negative memory bias whilst controlling for diagnosis clusters (and IDS-SR in models 2a to 5b).

	B	SE B	β	p-value	R^2
Model 1 (IDS-SR)				< .001	.355
Constant	40.018	3.816		< .001	
CM vs. SR	-14.684	2.060	-.473	< .001	
ND vs. SR	-4.727	1.933	-.164	.015	
Negative memory bias	10.369	2.385	.263	< .001	
Model 2a (CAARS)				< .001	.354
Constant	38.857	3.994		< .001	
CM vs. SR	2.248	2.156	.081	.298	
ND vs. SR	5.645	2.023	.217	.006**	
Negative memory bias	9.827	2.496	.277	< .001	
Model 2b (CAARS)				< .001	.593
Constant	17.535	4.340		< .001	
CM vs. SR	10.072	2.097	.361	< .001	
ND vs. SR	8.163	1.773	.314	< .001	
Negative memory bias	4.302	2.260	.121	.058	
IDS-SR	.533	.066	.593	< .001	
Model 3a (AQ-50)				.002**	.319
Constant	115.737	7.097		< .001	
CM vs. SR	7.351	3.830	.150	.056	
ND vs. SR	13.914	3.594	.306	< .001	
Negative memory bias	11.428	4.434	.184	.011*	
Model 3b (AQ-50)				< .001	.379
Constant	99.661	8.746		< .001	
CM vs. SR	13.250	4.226	.271	.002	
ND vs. SR	15.813	3.574	.347	< .001	
Negative memory bias	7.262	4.554	.117	.112	
IDS-SR	.402	.133	.255	.003**	
Model 4a (ASI)				.002**	.327
Constant	21.991	3.222		< .001	
CM vs. SR	-4.893	1.739	-.220	.005**	
ND vs. SR	-1.600	1.632	-.077	.328	
Negative memory	3.168	2.013	.112	.117	
Model 4b (ASI)				< .001	.538
Constant	6.712	3.628		.066	
CM vs. SR	.713	1.753	.032	.685	
ND vs. SR	.205	1.482	.010	.890	
Negative memory bias	-.791	1.889	-.028	.676	
IDS-SR	.382	.055	.532	< .001	

Note: gender identification, age, and education level were also added to each model. Education level was only significantly associated with ASI ($p < .01$).
*$p < .05$, ** $p < .01$.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

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Declarations of Competing Interest

None.

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References

Allan, Nicholas, Capron, Daniel, Raines, Amanda, Schmidt, Norman, 2014. Unique relations among anxiety sensitivity factors and anxiety, depression, and suicidal ideation. Journal of Anxiety Disorders 28 (2), 266–275.
Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., Clubley, E., 2001. The Autism-Spectrum Quotient (AQ): evidence from Asperger syndrome/high-functioning autism, males and females, scientists and mathematicians. Journal of Autism and Developmental Disorders 31, 5–17.
Beck, A.T., 1974. The development of depression: a cognitive model. In: Friedman, R.J., Katz, M.M. (Eds.), The psychology of depression: contemporary theory and research. John Wiley & Sons, Oxford, England.
Beck, A.T., 1987. Cognitive models of depression. Journal of Cognitive Psychotherapy 1, 5–37.
Beck, A.T., 2008. The evolution of the cognitive model of depression and its neurobiological correlates. American Journal of Psychiatry 165, 969–977.
Beck, A.T., Bredemeier, K., 2016. A unified model of depression: integrating clinical, cognitive, biological, and evolutionary perspectives. Clinical Psychological Science 4, 596–619.
Beevers, C.G., Mullarkey, M.C., Dainer-Best, J., Stewart, R.A., Labrada, J., Allen, J.J.B., …, Shumake, J., 2019. Association between negative cognitive bias and depression: a symptom-level approach. Journal of Abnormal Psychology 128, 212–227.
Benau, E.M., Hill, K.E., Atchley, R.A., O'Hare, A.J., Gibson, L.J., Hajcak, G., Ilardi, S.S., Foti, D., 2019. Increased neural sensitivity to self-relevant stimuli in major depressive disorder. Psychophysiology. https://doi.org/10.1111/psyp.13345.
Bradley, B.P, Mathews, A., 1983. Negative self-schemata in clinical depression. The British Journal of Clinical Psychology/The British Psychological Society 22, 173–181.
Bradley, B.P., Mogg, K., Williams, R., 1995. Implicit and explicit memory for emotion-congruent information in clinical depression and anxiety. Behaviour Research and Therapy 33, 755–770.
Bradley, M. M., & Lang, P. J. (1999). *Affective norms for English words (ANEW): Instruction manual and affective ratings*. Technical report C-1, The Center for Research in Psychophysiology, University of Florida.
Chandrasekhar, T., Sikich, L., 2015. Challenges in the diagnosis and treatment of depression in autism spectrum disorders across the lifespan. Dialogues in Clinical Neuroscience 17, 219–227.
Clerkin, Elise, Beard, Courtney, Fisher, Christopher, Schofield, Casey, 2015. An attempt to target anxiety sensitivity via cognitive bias modification. PLoS One 10 (2). https://doi.org/10.1371/journal.pone.0114578.
Coles, M.E., Heimberg, R.G., 2002. Memory biases in the anxiety disorders: current status. Clinical Psychology Review 22, 587–627.
Conners, C.K., Erhardt, D., Epstein, J.N., Parker, D.A., Sitarenios, G., Sparrow, E., 1999. Self-ratings of ADHD symptoms in adults I: factor structure and normative data. Journal of Attention Disorders 3, 141–151.
Cuthbert, B.N., Insel, T.R., 2013. Toward the future of psychiatric diagnosis: the seven pillars of RDoC. BMC Medicine 11, 126.
Dalgleish, Tim, Watts, Fraser, 1990. Biases of attention and memory in disorders of anxiety and depression. Clinical Psychology Review 10 (5). https://doi.org/10.1016/0272-7358(90)90098-U.
De Graaf, R., Bijl, R.V., Spijker, J., Beekman, A.T.F., Vollebergh, W.A.M., 2003. Temporal sequencing of lifetime mood disorders in relation to comorbid anxiety and substance use disorders. Social Psychiatry and Psychiatric Epidemiology 38, 1–11.
Deacon, B., Abramowitz, J., 2006. Anxiety sensitivity and its dimensions across the anxiety disorders. Journal of Anxiety Disorders 20, 837–857.
Del Valle, C.H.C., Mateos, P.M., 2018. Implicit Mood Congruent Memory Bias in Subclinical Depression. International Journal of Cognitive Therapy 11, 287–298.
Derry, P.A., Kuiper, N.A., 1981. Schematic processing and self-reference in clinical depression. Journal of Abnormal Psychology 90, 286–297.
Dobson, K.S., Shaw, B.F., 1987. Specificity and stability of self-referent encoding in clinical depression. Journal of Abnormal Psychology 96, 34–40.
First, M.B., Spitzer, R.L., Gibbon, M., Williams, J.B.W., 2002. Structured Clinical Interview for DSM-IV Axis I Disorders, Research Version, Patient Edition. Biometrics Research, New York State Psychiatric Institute, New York.
Freeth, M., Ropar, F., Chapman, P., Mitchell, P., 2010. The eye gaze direction of an observed person can bias perception, memory and attention in adolescents with and without autism spectrum disorder. Journal of Experimental Child Psychology 105, 20–37.
Gargaro, B.A., Rinehart, N.J., Bradshaw, J.L., Tonge, B.J., Sheppard, D.M., 2011. Autism and ADHD: how far have we come in the comorbidity debate. Neuroscience & Biobehavioural Reviews 35, 1081–1088.
Gerritsen, L., Rijpkema, M., Van Oostrom, I., Buitelaar, J., Franke, B., Fernández, G., Tendolkar, I., 2012. Amygdala to hippocampal volume ratio is associated with negative memory bias in healthy subjects. Psychological Medicine 42, 335–343.
Gotlib, I.H., Joormann, J., 2010. Cognition and depression: current status and future directions. Annual Reviews in Clinical Psychology 6, 285–312.
Gotlib, I.H., Kasch, K.L., Traill, S., Joormann, J., Arnow, B.A., Johnson, S.L., 2004.

Coherence and specificity of information-processing biases in depression and social phobia. Journal of Abnormal Psychology 113, 386–398.
Gotlib, I.H., Krasnoperova, E., 1998. Biased information processing as a vulnerability factor for depression. Behavior Therapy 29, 603–617.
Gupta, R., Kar, B.R., 2012. Attention and memory biases as stable abnormalities among currently depressed and currently remitted individuals with unipolar depression. Frontiers in Psychiatry. https://doi.org/10.3389/fpsyt.2012.00099.
Hamilton, J.P., Gotlib, I.H., 2008. Neural substrates of increased memory sensitivity for negative stimuli in major depression. Biological Psychiatry 63, 1155–1162.
Harkin, B., Snyder, H.R., Gulley, L.D., Schweizer, T.H., Bijttebier, P., Nelis, S., Toh, G., Vasey, M.W., 2016. Understanding comorbidity among internalizing problems: Integrating latent structural models of psychopathology and risk mechanisms. Development and Psychopathology 28, 987–1012.
Harmer, C.J., Duman, R.S., Cowen, P.J., 2017. How do antidepressants work? New perspectives for refining future treatment approaches. Lancet Psychiatry 4, 409–418.
Harmer, C.J., Goodwin, G.M., Cowen, P.J., 2009. Why do antidepressants take so long to work? A cognitive neuropsychological model of antidepressant drug action. British Journal of Psychiatry 195, 102–108.
Harmer, C.J., Hill, S.A., Taylor, M.J., Cowen, P.J., Goodwin, G.M., 2003. Toward a neuropsychological theory of antidepressant drug action: increase in positive emotional bias after potentiation of norepinephrine activity. The American Journal of Psychiatry 160, 990–992.
Henderson, H.A., Zahka, N.E., Kojkowski, N.M., Inge, A.P., Schwartz, C.B., …, Mundy, P.C., 2009. Self-referenced memory, social cognition, and symptom presentation in autism. The Journal of Child Psychology and Psychiatry 50, 853–861.
Hertel, P.T., Mathews, A., 2011. Cognitive bias modification: past perspectives, current findings, and future applications. Perspectives on Psychological Science 6, 521–536.
Hofvander, B., Delorme, R., Chaste, P., Nydén, A., Wentz, E., Stahlberg, O., Herbrecht, E., …, Leboyer, M., 2009. Psychiatric and psychosocial problems in adults with normal-intelligence autism spectrum disorders. BMC Psychiatry 9. https://doi.org/10.1186/1471-244X-9-35.
Insel, T.R., 2014. The NIMH Research Domain Criteria (RDoC) project: precision medicine for psychiatry. American Journal of Psychiatry 171, 395–397.
Johnson, S.L., Joormann, J., Gotlib, I.H., 2007. Does processing of emotional stimuli predict symptomatic improvement and diagnostic recovery from major depression? Emotion 7, 201–206.
Joormann, J., Stanton, C.H., 2016. Examining emotion regulation in depression: a review and future directions. Behaviour Research and Therapy 86, 35–49.
Joshi, G., Wozniak, J., Petty, C., Martelon, M., Fried, R., Bolfek, A., Kotte, A., …, Biederman, J., 2013. Psychiatric comorbidity and functioning in a clinically referred population of adults with autism spectrum disorders: a comparative study. Journal of Autism and Developmental Disorders 43, 1314–1325.
Kalenzaga, S., Jouhaud, V., 2018. The self-reference effect in memory: an implicit way to assess affective self-representations in social anxiety. Memory 26, 894–903.
Kessler, R.C., Berglund, P.B., Demler, O., Jin, R., Merikangas, K.R., Walters, E.E., 2005. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry 62, 593–602.
Kooij, J.J.S., 2010. Diagnostic Interview for ADHD in Adults 2.0 (DIVA 2.0). Adult ADHD. Diagnostic assessment and treatment. Pearson Assessment and Information BC, Amsterdam.
Lang, T.J., Moulds, M.L., Holmes, E.A., 2009. Reducing depressive intrusions via a computerizes cognitive bias modification of appraisal task: developing a cognitive vaccine. Behaviour Research and Therapy 47, 139–145.
LeMoult, J., Gotlib, I.H., 2019. Depression: a cognitive perspective. Clinical Psychology Review 69, 51–66.
LeMoult, J., Kircanski, K., Prasad, G., Gotlib, I.H., 2016. Negative self-referential processing predicts the recurrence of major depressive episodes. Clinical Psychological Science 5, 174–181.
Marchetti, I., Everaert, J., Dainer-Best, J., Loeys, T., Beevers, C.G., Koster, E.H.W., 2018. Specificity and overlap of attention and memory biases in depression. Journal of Affective Disorders 225, 404–412.
Matt, G.E., Vàzquez, C., Campbell, W.K., 1992. Mood-congruent recall of affectively toned stimuli: a meta-analytic review. Clinical Psychology Review 12, 227–255.
Miskowaik, K.W., Carvalho, A.F., 2014. 'Hot' cognition in major depressive disorder: a systematic review. CNS & Neurological Disorders - Drug Targets 13, 1787–1803.
Mogg, Karin, Mathews, Andrew, Weinman, John, 1987. Memory bias in clinical anxiety. Journal of Abnormal Psychology 96 (2). https://doi.org/10.1037/0021-843X.96.2.94.
Nikendei, C., Weisbrod, M., Schild, S., Bender, S., Walther, S., Herzog, W., Friederich, H.C., 2008. Anorexia nervosa: selective processing of food-related word and pictorial stimuli in recognition and free recall tests. International Journal of Eating Disorders 41, 439–447.
Powers, M., Davis, M., Kauffman, B., Baird, S., Zvolensky, M., Marcus, B., Smits, J.A., 2016. Anxiety sensitivity and smoking variability among treatment seeking smokers. Addictive Disorders & Their Treatment 15, 136–142.
Rodriguez, B.F., Bruce, S.E., Pagano, M.E., Spencer, M.A., Keller, M.B., 2004. Factor structure and stability of the Anxiety Sensitivity Index in a longitudinal study of anxiety disorder patients. Behaviour Research and Therapy 42, 79–91.
Royer, F.L., 1971. Information processing of visual figures in the digit symbol substitution task. Journal of Experimental Psychology 87, 335–342.
Rush, A.J., Guillion, C.M., Basco, M.R., Jarrett, R.N., Trivedi, M.H., 1996. The Inventory of Depressive Symptomatology (IDS): psychometric properties. Psychological Medicine 26, 477–486.
Schippers, G.M., Broekman, T.G., Buchholz, A., 2011. Manual and protocol, English Edition. Bèta Boeken, Nijmegen W.M. Cox.
Sharma, S., Powers, A., Bradley, B., Ressler, K.J., 2016. Gene × environment determinants of stress- and anxiety-related disorders. Annual Review of Psychology 67, 239–261.
Stronks, K., Snijder, M.B., Peters, R.J., Prins, M., Schene, A.H., Zwinderman, A.H., 2013. Unravelling the impact of ethnicity on health in Europe: the HELIUS study. BMC Public Health. https://doi.org/10.1186/1471-2458-13-402.
Symons, C.S., Johnson, B.T., 1997. The self-reference effect in memory: a meta-analysis. Psychological Bulletin 121, 37–394.
Thapar, A., Cooper, M., Rutter, M., 2017. Neurodevelopmental disorders. The Lancet Psychiatry 4, 339–346.
Van Oostrom, I., Franke, B., Rijpkema, M., Gerritsen, L., Arias-Vásques, A., Fernández, G., Tendolkar, I., 2012. Interaction between BDNF Val66Met and childhood stressful life events is associated to affective memory bias in men but not women. Biological Psychology 89 (1), 214–219.
Vigo, D., Thornicroft, G., Atun, R., 2016. Estimating the true global burden of mental illness. Lancet Psychiatry 3, 171–178.
Vogel, S., Gerritsen, L., Van Oostrom, I., Arias-Vásquez, A., Rijpkema, M., Joëls, M., …, Fernández, G., 2014. Linking genetic variants of the mineralcorticoid receptor and negative memory bias: Interaction with prior life adversity. Psychoneuroendocrinology 40, 181–190.
Vrijsen, J.N., Van Amen, C.T., Koekkoek, B., Van Oostrom, I., Schene, A.H., Tendolkar, I., 2017. Childhood trauma and negative memory bias as shared risk factors for psychopathology and comorbidity in a naturalistic psychiatric patient sample. Brain and Behaviour. https://doi.org/10.1002/brb3.693.
Vrijsen, J.N., Becker, E.S., Arias-Vásquez, A., Van Dijk, M.K., Speckens, A., Van Oostrom, I., 2014. What is the contribution of different cognitive biases and stressful childhood events to the presence and number of previous depressive episodes? Psychiatry Research 217, 134–142.
Vrijsen, J.N., Dainer-Best, J., Witcraft, S.M., Papini, S., Hertel, P., Beevers, C.G., …, Smits, J.A.J., 2018a. Effect of cognitive bias modification-memory on depressive symptoms and autobiographical memory bias: two independent studies in high ruminating and dysphoric samples. Cognition and Emotion 33, 288–304.
Vrijsen, J.N., Tendolkar, I., Onnink, M., Hoogman, M., Schene, A.H., Fernández, G., Van Oostrom, I., Franke, B., 2018b. ADHD symptoms in healthy adults are associated with stressful life events and negative memory bias. ADHD Attention Deficit and Hyperactivity Disorders 10, 151–160.
Vuijk, R., 2016. Nederlands Interview ten behoeven van Diagnostiek Autismespectrumstoornis bij volwassenen (NIDA). Sarr Expertisecentrum Autisme/Dare to Design, Rotterdam.
Watkins, E., 2015. An alternative transdiagnostic mechanistic approach to affective disorders illustrated with research from clinical psychology. Emotion Review 7, 250–255.
Watkins, P.C., Vache, K., Verney, S.P., Muller, S., Mathews, A., 1996. Unconscious mood-congruent memory bias in depression. Journal of Abnormal Psychology 105, 34–41.
Wiers, R.W., Boelema, S.R., Nikolaou, K., Gladwin, T.E., 2015. On the development of implicit and control processes in relation to substance use in adolescence. Current Addiction Reports 2, 141–155.
Young, J.E., 1990. Cognitive therapy for personality disorders: a schema-focused approach. Professional Resource Exchange, Sarasota.