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Research report

Cognitive reactivity, self-depressed associations, and the recurrence of depression

Hermien J. Elgersma [a,*], Peter J. de Jong [a], Gerard D. van Rijsbergen [a], Gemma D. Kok [a], Huibert Burger [b], Willem van der Does [c,d], Brenda W.J.H. Penninx [e], Claudi L.H. Bockting [a,f]

[a] Department of Clinical Psychology, University of Groningen, The Netherlands
[b] Department of General Practice, University of Groningen, University Medical Centre Groningen, Groningen, The Netherlands
[c] Institute of Psychology, Leiden University, Leiden, The Netherlands
[d] Department of Psychiatry, Leiden University Medical Centre, Leiden, The Netherlands
[e] Department of Psychiatry/EMGO Institute for Health and Care Research, VU University Medical Centre, Amsterdam, The Netherlands
[f] Utrecht University, Department of Clinical and Health Psychology, Utrecht, The Netherlands

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ABSTRACT

Background: Mixed evidence exists regarding the role of cognitive reactivity (CR; cognitive responsivity to a negative mood) as a risk factor for recurrences of depression. One explanation for the mixed evidence may lie in the number of previous depressive episodes. Heightened CR may be especially relevant as a risk factor for the development of multiple depressive episodes and less so for a single depressive episode. In addition, it is theoretically plausible but not yet tested that the relationship between CR and number of episodes is moderated by the strength of automatic depression-related self-associations.

Aim: To investigate (i) the strength of CR in remitted depressed individuals with a history of a single vs. multiple episodes, and (ii) the potentially moderating role of automatic negative self-associations in the relationship between the number of episodes and CR.

Method: Cross-sectional analysis of data obtained in a cohort study (Study 1) and during baseline assessments in two clinical trials (Study 2). Study 1 used data from the Netherlands Study of Depression and Anxiety (NESDA) and compared never-depressed participants ($n=901$) with remitted participants with either a single ($n=336$) or at least 2 previous episodes ($n=273$). Study 2 included only remitted participants with at least two previous episodes ($n=273$). The Leiden Index of Depression Sensitivity Revised (LEIDS-R) was used to index CR and an Implicit Association Test (IAT) to measure implicit self-associations.

Results: In Study 1, remitted depressed participants with multiple episodes had significantly higher CR than those with a single or no previous episode. The remitted individuals with multiple episodes of Study 2 had even higher CR scores than those of Study 1. Within the group of individuals with multiple episodes, CR was not heightened as a function of the number of episodes, even if individual differences in automatic negative self-associations were taken into account.

Limitations: The study employed a cross-sectional design, which precludes a firm conclusion with regard to the direction of this relationship.

Conclusions: The findings are consistent with the view that high CR puts people at risk for recurrent depression and is less relevant for the development of an incidental depressive episode. This suggests that CR is an important target for interventions that aim to prevent the recurrence of depression.

1. Introduction

Major depressive disorder (MDD) causes suffering in affected individuals and those around them (Ormel et al., 2008). Furthermore, the recurrent nature of MDD not only leads to suffering but contributes to high societal and health care costs (Mathers and

* Correspondence to: Department of Clinical Psychology, University of Groningen, Grote Kruisstraat 2/1, 9712 TS Groningen,The Netherlands.
E-mail address: h.j.elgersma@rug.nl (H.J. Elgersma).

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Loncar, 2006; Smit et al., 2006). It is therefore crucial to improve our understanding of the mechanisms that underlie vulnerability to recurrence in depression.[1]

In cognitive models, so-called dysfunctional attitudes are considered to be the key vulnerability factor, both for the first onset and for recurrences of depression (Beck, 1967; Teasdale, 1988; Clark et al., 1999). 'Dual process' models (e.g., Beevers, 2005) distinguish between relatively automatic (reflexive) and more deliberate (reflective) attitudes. In individuals vulnerable to depression, automatic (reflexive) associations are predominantly negative and trigger further negative cognitive and affective responses (Beevers, 2005). The reflective processing system may then adjust or affirm this initial response. The reflective system is concerned with the validation of cognitions and requires time and resources. The activation of these cognitions occurs non-intentionally, and may even occur when a person (deliberately) considers these associations to be false/invalid. A negative feedback loop may develop between associative processing and negative mood if people fail to correct their dysfunctional automatic associations (e.g., because of a lack of available resources, and/or dysfunctional reflective strategies).

Both reflexive and reflective attitudes may be involved in triggering the recurrence of depression. It has been argued that explicit (reflective) dysfunctional attitudes may remain undetected ('latent') in depression-vulnerable individuals unless they are primed or activated by an event or a negative mood state (Beck, 1967; Scher et al., 2005). Consequently, these latent attitudes may remain undetected if assessment is by questionnaire. Since the priming may occur during relatively mild, non-pathological negative mood states (Miranda et al., 1998), the traditional way to solve this problem is to measure dysfunctional attitudes both in euthymic and dysphoric mood states (either naturally occurring or induced). The difference score is called cognitive reactivity to sad mood (CR). An alternative and relatively easily-administered measure of CR is the self-report Leiden Index of Depression Sensitivity (LEIDS, and its revised version the LEIDS-R) (van der Does, 2002). The LEIDS aims to assess the extent to which negative beliefs increase when individuals experience mild dysphoria. A sample item is "When in a sad mood, I more often think about how my life could have been different". Supporting its validity as a measure of CR, the LEIDS had strong predictive value for CR, as measured with a mood induction procedure (van der Does, 2002). CR as indexed by the LEIDS-R was found to be associated with an increase in depressive symptoms over a one year period (Struijs et al., 2013) and emphasizing its relevance as a premorbid risk factor it was also found to be predictive for the first onset of depression (Kruijt et al., 2013).

Automatic (implicit) dysfunctional attitudes are assessed by means of reaction time measures such as the Implicit Associations Test (IAT) (Greenwald et al., 1998). Previous research using this type of performance measure has shown that people with a depressive disorder have relatively strong negative self-associations (e.g., between 'I' and 'worthless'; Glashouwer and de Jong, 2010), and the strength of these associations is related to suicidal ideation (Glashouwer et al., 2010). Strong automatic associations also predict an unfavorable course of depressive disorders (Glashouwer et al., 2012).

The mechanisms involved in a first onset of depression may differ from those involved in recurrences (Just et al., 2001; Monroe and Harkness, 2011; Lewinsohn et al., 1981). CR (as indexed by the LEIDS-R) has been shown to predict first onsets of depression, whereas self-depressed associations (IAT scores) did not predict first onsets after statistically partialling out the level of depressive symptoms and negative life events (Kruijt et al., 2013). High CR may also increase the risk of recurrence of depression and two studies using mood induction procedures have shown CR to be a significant predictor of recurrence over periods of 15 and 18 months after remission (Segal et al., 2006; Kuyken et al., 2010). However, two other studies, failed to find a similar relationship between CR and recurrence/return of symptoms (Lethbridge and Allen, 2008; van Rijsbergen et al., 2013). Unfortunately, the number of previous episodes was not taken into consideration in any of these studies.

Yet, different factors may be involved in the pathway to a single/incidental vs. multiple depressive episodes. Thus, also the risk for recurrence might differ between individuals who experienced a single episode vs. individuals who experienced multiple episodes. In line with this reasoning, there is strong evidence that recurrence risk is higher in individuals with multiple episodes than in individuals with a single depressive episode (Judd et al., 1998a, 1998b; Bockting et al., 2006; ten Doesschate et al., 2010; Hardeveld et al., 2013). A relatively strong habitual cognitive responsivity to a negative mood (high CR) may be especially relevant as a premorbid risk factor for recurrent depression and less so for incidental/single depressive episodes. If so, as a group, individuals with multiple episodes should show higher CR than those with only a single episode. Therefore, the first aim of this cross-sectional study was to test the hypothesis that people with multiple depressive episodes have higher CR scores than those with a single past episode.

Although automatic self-depressed associations have not shown to predict first onset of depression and can thus not be considered as a premorbid risk factor (Kruijt et al., 2013), they may still be involved in its recurrence. The repeated activation of negative associations during depressive episodes may result in an associative memory network in which the self becomes increasingly linked to negative attributes (Risch et al., 2010). A feedback loop between self-negative associative processing and depressive symptoms may occur. Dysfunctional self-associations may become increasingly easy to activate, even during mild stress or mild negative mood states, lowering the threshold for a depressive episode. In line with the idea that repeated episodes may give rise to such a 'hidden scar', we recently found that individuals with a relatively high number of previous episodes also had relatively strong self-depressed associations (Elgersma et al., 2013). Moreover, the duration of depressive symptoms also predicted the increase in strength of self-depressed associations (Elgersma et al., 2013).

Surprisingly, the interaction between automatic (reflexive) associations and CR has not received much attention. From a dual process perspective, CR can be conceptualized as a more reflective (propositional) cognitive process, one that will act in a way to further affirm reflexively triggered dysfunctional automatic self-associations. These dysfunctional automatic self-associations may thus moderate the relationship between CR and depression recurrence. In other words, individuals with relatively strong and easily elicited dysfunctional automatic self-associations and high CR may be especially prone to a course of illness with multiple depressive episodes. Therefore, the second aim of this study was to test whether the relationship between CR and the number of depressive episodes is moderated by the strength of automatic self-depressed associations.

[1] In this paper we use the term recurrence. Recurrence is proposed to represent a new episode after recovery (a period of at least two months of no longer meeting criteria for depression after the acute phase of depression). In the literature the terms 'relapse', and 'recovery' are used; not always based on clear-cut definitions of each and sometimes not even distinguishing between the two. Hollon et al. (2006) proposed a more stringent use of the terms relapse (reemergence of an episode of depression within the first 6–12 months after initial remission, whereas recurrence was proposed to represent a new episode after recovery, occurring after 12 months of initial remission. In this study it was not always possible to make this distinction. For the sake of clarity, we made the decision to use the term recurrence.

In summary, in the present investigation we tested the following hypotheses: (i) remitted MDD participants with a history of multiple episodes have higher CR scores than those with a single or no episode; and (ii) the relationship between the number of previous depressive episodes and CR is moderated by automatic self-depressed associations. We tested our hypotheses using a cross-sectional analysis of baseline data from participants of the NESDA study ($N=2981$), a large longitudinal study on the long-term course of anxiety and depressive disorders. Next, we examined the robustness of the NESDA findings (Study 1) using the baseline data from a group of remitted recurrently depressed participants ($N=309$) of two clinical trials (Study 2).

2. Study 1

2.1. Method

We used data from a large multicentre longitudinal cohort study, the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008). NESDA is an ongoing multi-center, longitudinal cohort study, designed to examine the long-term course and consequences of anxiety and depressive disorders (see also www.nesda.nl). The NESDA study protocol was approved by the Ethical Review Board of VU University Medical Centre Amsterdam and by local review boards of each participating center. All participants provided written informed consent. Cognitive reactivity and automatic self-depressed associations were measured in individuals who were remitted from a single or from multiple depressive episodes as well as in individuals who had never experienced a depressive episode.

2.1.1. Participants

Participants of the NESDA study were recruited from the general population, general practices, and from mental health care institutions. Participants ranging from no (healthy controls) to varying degrees of psychopathology were included: healthy controls, individuals at risk because of previous episodes, subthreshold symptoms or family history and individuals with a first or recurrent depressive or anxiety disorder. Uniform inclusion and exclusion criteria were used across all recruitment settings. A general inclusion criterion was an age of 18–65 years. The two exclusion criteria were as follows: (1) a primary diagnosis of a psychotic disorder, obsessive compulsive disorder, bipolar disorder or substance use disorder; and (2) not being fluent in Dutch. For a more detailed description of the study see Penninx et al. (2008).

The present study concerned an analysis of data from the baseline assessments conducted from September 2004 until February 2007. We selected participants with (1) no depressive diagnoses (MDD, Dysthymia) at baseline nor in their history, participants with (2) one or more depressive diagnoses (MDD, Dysthymia) in the past but who were remitted at baseline; and participants (3) in remission at baseline with multiple previous depressive diagnoses in their history.

2.1.2. Instruments

Psychiatric diagnosis Psychiatric disorders were determined by means of the lifetime Composite International Diagnostic Interviews (CIDI) (WHO version 2.1, Robins et al., 1988; Wacker et al., 2006). The CIDI classifies diagnoses according to DSM-IV-TR criteria (APA, 2001).

Depression history The number of previous depressive episodes at baseline was established using the CIDI interview.

Depressive symptoms Depressive symptoms were assessed using the 30-item Inventory of Depressive Symptoms Self-Report version (IDS-SR) (Rush et al., 1996). The total score of the IDS-SR was used as an index for the severity of depression.

Cognitive reactivity The Leiden Index of Depression Sensitivity-Revised (LEIDS-R) is a 34-item self-report measure of cognitive reactivity to sad mood (van der Does, 2002). Participants are asked to imagine feeling a mild state of dysphoria, and then to fill out 34 items on a 5-point Likert scale. A sample item would be “When I feel down, I lose my temper more easily” or “In a sad mood I do more things that I will later regret”.

Automatic self-associations The IAT is a computerized reaction time task originally designed by Greenwald et al. (1998) to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts. Words from all four concept categories appear in mixed order in the middle of a computer screen and participants are instructed to sort them with a left (Q) or right (P) response key. The premise here is that sorting becomes easier when a target and attribute that share the same response key are strongly associated than when they are weakly associated. The category labels are visible in the upper left and right-hand corners of the screen during the whole task (for an example see https://implicit.harvard.edu/implicit). The target labels were ‘me’ and ‘other’. The attribute labels were ‘depressed’ and ‘elated’. Each category consisted of five stimuli (Appendix A). The IAT consists of two critical test blocks that were preceded by practice blocks (Table 1). In one test block ‘me’ and ‘depressed’ (and ‘other’ ‘elated’) share the same response key, whereas in the other test block ‘me’ and ‘elated’ (and ‘other’ and ‘depressed’) shared the response key. Before the start of a new sorting task, written instructions were presented on screen. After a correct response, the next stimulus was presented after 500 ms. Following an incorrect response, the word WRONG! appeared briefly above the stimulus. Meanwhile, the stimulus remained on the screen until the correct response was given. The order of the category combinations was fixed across participants to reduce method variance.

2.1.3. Procedure

The complete baseline assessment of the NESDA study lasted between 3 and 5 h and was conducted on one day. During the assessment, first the CIDI, then LEIDS-R and the Implicit Associations Test (IAT) were administered. Respondents were compensated with a euro 15, – gift certificate and travel expenses.

2.1.3.1. Data reduction. LEIDS-R In line with previous research, the total scores of the LEIDS-R (34 items) were used to index CR. For each item the scores ranged from 0 to 4 and for statistical analyses we used the mean total score.

IAT IAT performance was indexed by the widely used D-measure proposed by Greenwald et al. (2003) on the basis of internet-studies. The D-measure also performed best in a laboratory setting such as used in NESDA (Glashouwer et al., 2013). Here we report the D4-measure. Following the guidelines, reaction times above 10,000 ms were discarded and error trials were replaced with the mean reaction times of the correct responses in the block in which the error occurred, plus a ‘penalty’ of 600 ms. The IAT effect was calculated by subtracting the mean reaction times of block 3 from block 6 (practice) and block

Table 1
Arrangement of Implicit Association Test blocks.

Block		Left Label(s)	Right Label(s)	Number of trials
1	Practice	Depressed	Elated	20
2	Practice	Me/depressed	Other/elated	20
3	Test	Me/depressed	Other/elated	60
4	Practice	Elated	Depressed	20
5	Practice	Me/elated	Other/depressed	20
6	Test	Me/elated	Other/depressed	60

4 from block 7 (test). The means of these two effects were divided by their pooled standard deviation based on all responses in blocks 3, 4, 6 and 7. A negative IAT score indicates a relatively fast response to trials in which 'me' shared the response key with 'depressed'. Thus the stronger the negative self-associations the more negative the IAT score.

2.1.3.2. Statistical analyses. We first compared mean CR scores between participants with none vs. a single depressive episode. To that end, group (none vs. one episode) was entered as an independent variable into a univariable regression model with the LEIDS-R scores as the dependent variable. We subsequently added current depression severity as an independent variable to statistically partial out its potential association with CR. We used the same statistical procedure for the most critical comparison between the groups with one vs. multiple depressive episodes.

To examine the relationship between number of previous episodes and CR we selected participants with two or more episodes. We again used the LEIDS-R score as the dependent variable, and the number of previous episodes as a continuous independent variable. Next, depression severity as indexed by IDS score was added to the model.

We used multiple regression analyses to test whether the relationship between CR and the number of previous episodes is moderated by the strength of automatic self-depressed associations. In this analysis we used the LEIDS-R score as the dependent variable, and group (one vs. two or more episodes), the IAT (D-4), and their interaction term (group × IAT) as independent variables. Finally, we added depression severity to the model. For each analysis we tested the assumptions of linearity, normality and homoscedasticity by inspecting residual plots. Regression coefficients were supplied with a 95% confidence interval. Statistical significance was conventionally defined as a two-sided *p*-value less than .05.

2.2. Results

2.2.1. Descriptives

Demographics, CR, IAT, and IDS-SR scores are reported in Table 2.

2.2.2. CR in participants with a history of none vs. a single depressive episode

Participants who were in remission from a single depressive episode had a statistically significantly higher mean total score on the LEIDS-R than never-depressed participants (β=.27, t=9.89, $p<.01$). This difference remained statistically significant when the IDS-SR was included in the model (β=.14, t=5.96, $p<.01$).

2.2.3. CR in remitted participants with a history of single vs. multiple depressive episodes

As predicted, MDD participants with a history of multiple depressive episodes had statistically higher total scores on the LEIDS-R than those with only one previous episode (β=.15, t=3.86, $p<.01$). This difference remained statistically significant when the IDS-SR was included in the model (β=.12, t=3.37, $p<.01$).

2.2.4. CR and number of multiple previous depressive episodes

In participants with two or more previous episodes we found no significant relationship between the number of episodes and CR (β=.10, t=1.74, p=.08) although there was a non-significant trend in the predicted direction (the higher CR, the higher the number of episodes).

2.2.5. Is the relationship between single vs. multiple previous depressive episodes and cognitive reactivity moderated by automatic self-depressed associations?

Multiple regression analyses showed that the relationship between group membership (one vs. two or more previous depressive episodes) and the total score on the LEIDS-R was not moderated by the strength of automatic self-depressed associations. As can be seen in Table 3, the interaction variable (IAT × group) did not show an independent relationship with the LEIDS-R mean total score.

3. Study 2

Consistent with the view that high CR may be especially relevant as a risk factor for recurrent depression and less so for incidental/single depressive episodes, the results of study 1 showed that remitted participants with multiple depressive episodes have higher cognitive reactivity scores than participants with a single depressive episode. This difference could not be (fully) attributed to differences in residual depressive symptoms, and was found to be independent of the strength of negative automatic self-associations.

Thus the current pattern of results provided no convincing support for the view that the lower threshold for subsequent episodes in individuals with increasing episode numbers is reflected in higher CR scores. One explanation could be that the NESDA sample was not specifically selected on the basis of being remitted of (recurrent) depression and might thus not optimally

Table 2
Descriptives of participants of Study 1 and Study 2; means and standard deviations.

Study	NESDA 0 episodes (n=901)	NESDA 1 episode (n=336)	NESDA 2 or more episodes (n=273)	NESDA at least 1 episode (n=609) (1 and 2 or more episodes together)	Study 2 2 or more episodes (n=273)
Gender (%female)	64.3	71.7	73.6	72.6	69.6
Age at baseline	41.59 (14.44)	43.30 (13.64)	43.14 (12.04)	43.23 (12.94)	47.08 (10.53)
Number of depressive episodes (median)	0	1	3	1	4
LEIDS mean score	.62 (.45)	.91 (.49)	1.07 (.47)	.98 (.49)	1.51 (.48)
LEIDS mean shared total score			1.11 (.46)		1.27 (.43)
IAT D-measure[a]	.37 (.37)	.27 (.40)	.25 (.40)	.26 (.40)	.15 (Pooled)
IDS total score	11.66 (9.54)	17.14 (10.06)	18.67 (9.89)	17.82 (10.01)	17.39 (10.59)
Use of AD (% yes)	6.0	23.5	24.5	23.9	68.1

Note: IAT=Implicit Association Test, LEIDS=Leiden Index of Depression Sensitivity-R, IDS=Inventory of Depressive Symptoms-SR, AD=antidepressants.

[a] Positive effects indicate a relatively stronger automatic association between me and elated. The stronger the negative self-associations the more negative the D-measure. Please note that the D-measure can take negative as well as positive values.

Table 3
Single and multiple regression models for predicting cognitive reactivity with the number of depressive episodes and implicit self-depressed associations in Study 1.

Dependent variable LEIDS-R total score	β	t	p
Model 1			
Group	.15	3.86	< .01
Model 2			
Group	.15	3.83	< .01
IAT	− .06	− 1.50	.13
Model 3			
Group	.15	3.83	< .01
IAT	− .06	− 1.50	.13
Group*IAT	.01	.25	.80
Model 4			
Group	.12	3.36	< .01
IAT	.001	.03	.97
Group*IAT	− .009	− .24	.80
IDS-SR	.44	12.21	< .01

Note: IAT = Implicit Association Test, IDS = Inventory of Depressive Symptoms-SR, LEIDS-R = Leiden Index of Depression Sensitivity-R, group: the group with one or the group with 2 or more depressive episodes in their history.
Adjusted R^2:
Model 1: .02
Model 2: .02
Model 3: .02
Model 4: .21.

reflect the CR of individuals who suffer from recurrent depression but are currently in remission.

We therefore decided to examine whether these findings of Study 1 could be corroborated in a group of individuals specifically selected on the basis of being remitted with a history of two or more depressive episodes. These participants, who were in remission for at least two months but not longer than two years, represent a more chronic range of the spectrum than the participants of Study 1. The aims of Study 2 were to examine (i) the robustness of the main finding of study 1 (heightened CR in individuals with two or more depressive episodes), (ii) the relationship between CR and number of depressive episodes in this group at high risk for recurrence. We also examined (iii) whether the absence of a moderating influence of automatic self-depressed associations on the relationship between the number of previous depressive episodes and CR (as found in Study 1) is actually replicated among a more specific group characterized by highly-recurrent depression.

3.1. Method

3.1.1. Participants

Participants were remitted from two or more depressive episodes and participated in one of two multi-center randomized controlled trials (RCTs). The RCTs were designed to examine the (cost-)effectiveness of preventive cognitive therapy for remitted participants. For the current study, we used the baseline data of these RCTs which were assessed from July 2009 until October, 2012. The studies were approved by the Medical Ethical Committee of the Dutch mental health care institutions (METIGG), and registered at the Dutch Trial Register (NTR 1907 and NTR 2503). The design and protocol of both RCTs are described elsewhere (Bockting et al., 2011a, 2011b). Participants in both RCTs had been recruited via media and via referrals of general practitioners, pharmacists, company doctors, and mental health care institutions (Bockting et al., 2011a, 2011b). Inclusion criteria were as follows: two or more episodes of major depressive disorder (DSM-IV criteria); currently in remission for longer than eight weeks but no longer than two years; current score on the 17-item Hamilton Rating Scale for Depression (HRSD) below or equal to 10; age between 18 and 65 years. For one of the RCTs there was the additional inclusion criterion that the participants had to use antidepressant medication for at least 6 months at study entry (for details of the design, see Bockting et al. (2011a)). Exclusion criteria were as follows: current mania or hypomania, a history of bipolar illness, any psychotic disorder (current and/or previous), organic brain damage, alcohol or drug misuse, primary diagnosis of anxiety disorder. As an additional exclusion criterion for one of the RCTs (Bockting et al., 2011b), participants without Internet access were excluded. The initial sample consisted of 309 (197 from one RCT + 112 from the other RCT) participants. CR and/or IDS-SR scores were missing for 36 participants, leaving a final sample of 273 participants.

3.1.2. Instruments

Psychiatric diagnosis and depression history Psychiatric disorders at baseline and the number of previous depressive episodes were determined using the SCID I interview (Spitzer et al., 1990).

Depressive symptoms Depressive symptoms were assessed using the 30-item Inventory of Depressive Symptoms Self-Report version (IDS-SR) (Rush et al., 1996). The total score of the IDS-SR was used as an index for the severity of depression.

Cognitive reactivity CR was indexed with the Leiden Index of Depression Sensitivity (LEIDS). To be consistent with a previous multi-center RCT (the Delta study; International Standard Controlled Trial Register Identifier ISRCTN68246470) we used the original LEIDS in these RCTs (van der Does, 2002). This version, referred to here as LEIDS2002, was revised in 2003 (LEIDS-R; http://www.dousa.nl/publications_depression.htm). The two versions share 21 identical items and identical instructions (see Appendix for details).[2]

Automatic associations Automatic associations were assessed using the same IAT (a computerized reaction time task to measure the relative strengths of automatic associations between two contrasted target concepts and two attribute concepts) as was used in Study 1.

3.1.3. Procedure

All consenting participants were asked to provide information about their socio-demographic background. In addition, they participated in a semi-structured clinical interview (SCID-I) and the LCI. If participants met all inclusion and none of the exclusion criteria, they entered the study and were randomly assigned to a treatment condition. Immediately after randomization they were invited to complete the IDS and the LEIDS through a personalized hyperlink. Other data were collected, but these were not of interest in the present study. Two days later participants received a personalized hyperlink to complete the IAT. Respondents were compensated with a € 25, – gift certificate.

3.1.3.1. Data reduction. We calculated mean total scores of the LEIDS2002 and – to facilitate comparison between Study 1 and Study 2 – the mean total of the overlapping items (see Table 2). The items are shown in Appendix B. The number of previous depressive episodes was expressed in a continuous variable.

3.1.3.2. Statistical analysis. We first compared CR scores between participants of Study 2 and participants of the NESDA study with two or more previous depressive episodes. To do this, group (Study 2 vs. Study 1) was entered as an independent variable into a

[2] When we repeated the regression analysis for the NESDA study post-hoc (i.e. Study 1) only with the shared items of the LEIDS-R and the LEIDS, we found the same pattern as with the total LEIDS-R.

regression model. We used the mean total scores of the shared items of both LEIDS versions as the dependent variable. Next, we included depressive symptom severity in the analyses as an independent variable to statistically partial out its potential association with CR.

To examine the relationship between the number of previous depressive episodes and CR in Study 2 we used the mean total score of the LEIDS2002 as the dependent variable, and the previous number of depressive episodes as a continuous independent variable in a regression analysis. Next, depression severity as indexed by the IDS score was added to the model.

We used multiple regression analyses to test whether the relationship between CR and the number of previous episodes is moderated by the strength of implicit self-depressed associations in Study 2. In this analysis we used LEIDS2002 mean total scores as the dependent variable, and the number of previous depressive episodes, the IAT (D-4), and their interaction term (number of depressive episodes × IAT) as independent variables. Finally, we added depression severity to the model. For each analysis we tested the assumptions of linearity, normality and homoscedasticity by inspecting residual plots. Regression coefficients were supplied with a 95% confidence interval. Statistical significance was conventionally defined as a two-sided p-value less than .05.

3.1.3.3. Multiple imputations. We had 54.57% missing data on the IAT measure, mostly due to technical problems. For instance, the software program we used for the IAT was not compatible for a specific type of computer and the personalized hyperlink which was sent to the participants to complete the IAT appeared less robust than was anticipated on the basis of our testing. To optimally account for the unfavorable effects of missing data on the regression analyses, i.e. the restriction to subjects with complete data only, we conducted multiple imputation under the assumption that data were missing at random, which cannot be proved (Sterne et al., 2009). The imputation was done by chained equations and involved 40 iterations as recommended by Bodner (2008). The imputation model included the following baseline data: kind of RCT trial, number of previous depressive episodes, IDS, IAT, gender. Results from 40 identical analyses of each imputed data set were pooled using Rubin's (1987) rules; results can therefore be considered averages across the 40 imputed datasets.

3.2. Results

3.2.1. Descriptives

Demographics, CR, IAT and self-report IDS-SR scores are reported in Table 2.

3.2.2. Cognitive reactivity in remitted patients with two or more episodes: Study 1 vs. Study 2 participants

Group (included as dummy variable: Study 1 = 0, Study 2 = 1, showed a statistically significant association with the scores on the shared items of the LEIDS-R and the LEIDS2002 (β = .18, t = −4.30, $p < .01$); meaning that Study 2 participants had higher CR scores than participants of Study 1. This relationship remained statistically significant after adding depressive symptom severity to the model.

3.2.3. Association between number of previous depressive episodes and cognitive reactivity

Regression analyses with the score on the LEIDS2002 as the dependent variable and the number of previous episodes and IDS-SR score as the independent variables showed that there was no relationship between CR and the number of episodes in this high risk group (β = .02, t = .38, p = .70).

3.2.4. Automatic associations, number of previous episodes, and cognitive reactivity

Univariable regression analysis showed no statistically significant relationship between the number of previous depressive episodes and the total score on the LEIDS2002 (B = −.002, t = .06, p = .70. Multiple regression analyses showed that this relationship was not moderated by the strength of automatic self-depressed associations: the interaction variable (number of depressive episodes x IAT) did not have an independent relationship with the LEIDS-total score (B = −.08; S.E. = .07, t = −1.04, p = .29).

4. General discussion

The major findings can be summarized as follows: First, remitted depressed participants with two or more previous episodes have higher cognitive reactivity scores than those with one or no previous episode, whereas the group with a single previous episode had higher CR scores than never-depressed individuals. Second, within the group of individuals with multiple episodes, CR is not heightened as a function of the number of episodes. Thus the critical differences are among those who experienced none vs. a single vs. multiple episodes. Finally, the relationship between CR and number of depressive episodes was found to be independent of the strength of automatic self-depressed associations.

The heightened CR scores in remitted patients with multiple episodes are consistent with the view that CR might be especially relevant as a mechanism involved the group with multiple depressive episodes and less so for incidental/single depressive episodes. The present pattern of findings may also help explain the mixed findings of previous studies between CR and the recurrence of depression (Segal et al., 2006; Kuyken et al., 2010; Lethbridge and Allen, 2008; van Rijsbergen et al., 2013). Since the number of previous episodes was not taken into account in these studies, patients with only one depressive episode and/or patients with many (more than two) episodes may have been overrepresented.

Another explanation for the mixed findings may lie in the instrument used to assess CR. Previous studies have typically used a mood induction procedure in the lab (Segal et al., 2006; Kuyken et al., 2010; Lethbridge and Allen, 2008; van Rijsbergen et al., 2013), whereas the current study relied on the LEIDS scales. The LEIDS asks people to rate their habitual tendency to make more dysfunctional inferences when their mood deteriorates. It may be that the LEIDS is more sensitive than a laboratory procedure to cover the type of inferences people make on the basis of their negative mood in daily life. Consistent with this, more than 10 studies have now shown that the LEIDS-R is sensitive to depression history with no known replication failures, whereas studies using mood inductions regularly fail to distinguish between recovered depressed and never-depressed individuals (e.g., Brosse et al., 1999; van der Does, 2005).

Previous prognostic findings have already shown that high CR as indexed by the LEIDS-R can be seen as a premorbid characteristic that lowers the threshold for the first onset of depression (Kruijt et al., 2013). The present (cross-sectional) findings are consistent with the view that high CR may be especially important as a risk factor for the first few episodes of depression. The relevance of CR in the context of recurrent depression is further supported by the finding that CR scores within a highly recurrent sample (study 2 participants with two or more previous episodes) were even higher than those of the remitted individuals in the NESDA cohort.

A factor that might have contributed to the relatively high CR scores in Study 2 is that most of these individuals used antidepressant medication (68.1%; see Table 2). Although the use of AD may have immediate (i.e., within two hours) positive effects on information processing (Harmer et al., 2009), this effect may not extent to the reactivity of deliberate, higher order cognitive processes. Pharmacological interventions do not invite individuals to challenge their cognitions and information processing style (e.g., their tendency to use their negative mood as information) as is typically the case in cognitive therapy (e.g., Beck, 1967). However, in post-hoc analysis within this group, we did not find differences in CR between participants who did and those who did not use medication.[3] Another explanation for the difference between the Study 1 and Study 2 participants could be that the latter group had more previous depressed episodes (median 3 vs. 4 episodes). However, within both groups of participants with multiple episodes, the relationship between CR and the number of episodes was non-significant. Most likely, then, Study 2 participants' CR scores were higher than participants' scores in Study 1 because they were selected on a history of recurrent depressive episodes, whereas participants of Study 1 were not.

The relationship between CR and the number of depressive episodes was not moderated by the strength of automatic self-depressed associations. In other words, in neither study did we find any evidence for the hypothesis that high CR would be especially important as a risk factor for those with relatively strong automatic self-depressed associations. Consequently, the mixed findings in previous studies cannot be explained by differences in negative automatic self-associations. Combined with previous findings indicating that negative self-associations predict an unfavorable course of depressive disorders (Glashouwer et al., 2012), this suggests that the reflective attitude (CR) and the reflexive attitude (automatic self-depressed associations) are independent risk factors for recurrent depression. The importance of the different risk factors may change during the course of recurrent depression.

5. Limitations

The present studies have a number of limitations. Most importantly, the cross-sectional designs preclude firm conclusions with regard to the direction of the relationship between CR and the number of episodes. Although previous research has shown that high CR is a premorbid characteristic that predicts the first onset of depression (Kruijt et al., 2013), the present findings are also consistent with a scarring hypothesis: CR may also be increased as a consequence of having experienced one or multiple depressive episodes. However, the absence of a relationship between the number of episodes and the strength of CR in individuals with multiple depressive episodes, casts doubt on the view that CR might be seen as a consequence (scar) of a depressive episode. Yet, to reach more firm conclusions in this respect it would be critical to conduct a longitudinal investigation.

To test the possible causal status of heightened CR more rigorously, it would be crucial to examine whether reducing CR lowers the chance of recurrence. Such approach would not only be of theoretical importance but would also provide starting points for clinical interventions that may help disrupt the invalidating rhythm of depression. It has recently been demonstrated that a computerized experimental training was effective in decreasing anxiety-based cognitive reactivity (Lommen et al., 2013). A similar strategy may be employed to teach remitted participants not to rely on dysphoric mood as information (e.g., "when I feel sad, I think I am worthless") which could reduce negative mood based cognitive reactivity. If indeed the tendency to make dysfunctional inferences on the basis of a depressed mood is causally involved in the recurrence of depression, this would suggest that current effective preventive treatments might be effective because they reduce cognitive reactivity while being dysphoric (Bockting et al., In press).

6. Conclusions

To conclude, people with multiple depressive episodes have higher CR than individuals with a single (or no) previous episode, and individuals with a single episode have higher CR than never-depressed individuals. This pattern of findings is consistent with the view that high CR puts people at risk for recurrent depression. This relationship appears to be independent of the strength of negative automatic self-associations. Together these findings suggest that CR is an important target for interventions that aim to prevent the recurrence of depression.

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Conflict of interest

None of the authors declares any conflict of interest.

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[3] Post-hoc we used an univariable regression analysis for Study 2, using the mean total score of the LEIDS2002 as the dependent variable, and group (using antidepressants yes/no) as the independent variable. We found no statistically significant relationship between CR and group ($\beta = -.01$, $t = -.17$, $p = .86$).

Appendix A. Implicit Association Test stimulus words

Me: I, myself, self, my, own.
Other: other, you, they, them, themselves.
Depressed: useless, pessimistic, inadequate, negative, meaningless.
Elated: positive, optimistic, active, valuable, cheerful.
Words are translated from Dutch.

Appendix B. LEIDS-R and LEIDS 2000; both versions compared

LEIDS-R

Item	Shared or not
1. I can only think positive when I am in a good mood.	S
2. When in a low mood, I take fewer risks.	S
3. When I feel sad, I spend more time thinking about what my moods reveal about me as a person.	S
4. When I am in a sad mood, I am more creative than usual.	S
5. When I feel down, I more often feel hopeless about everything.	
6. When I feel down, I am more busy trying to keep images an thoughts away.	S
7. In a sad mood, I do more things that I will later regret.	
8. When I feel sad, I go out and do more pleasurable activities.	S
9. When I feel sad, I feel as if I care less if I lived or died.	
10. When I feel sad, I am more helpful.	S
11. When I feel sad, I am less inclined to express disagreement with someone else.	S
12. When I feel somewhat depressed, I think I can permit myself fewer mistakes.	S
13. When I feel down, I more often feel overwhelmed by things.	
14. When in a low mood, I am more inclined to avoid difficulties or conflicts.	S
15. When I feel down, I have a better intuitive feeling for what people really mean.	S
16. When in a sad mood, I become more bothered by perfectionism.	S
17. When I feel sad, I more often think that I can make no one happy.	
18. When I feel bad, I feel more like breaking things.	
19. I work harder when I feel down.	S
20. When I feel sad, I feel less able to cope with everyday tasks and interests.	
21. In a sad mood, I am bothered more by aggressive thoughts.	
22. When I feel down, I more easily become cynical (blunt) or sarcastic.	S
23. When I feel down, I feel more like escaping everything.	
24. When in a sad mood, I feel more like myself.	S
25. When I feel down, I more often neglect things.	S
26. When I feel sad, I do more risky things.	
27. When I am sad, I have more problems concentrating.	S
28. When in a low mood, I am nicer than usual.	S
29. When I feel down, I lose my temper more easily.	
30. When I feel sad, I feel more that people would be better off if I were dead.	
31. When I feel down, I am more inclined to want to keep everything under control.	S
32. When I feel sad, I spend more time thinking about the possible causes of my moods.	S
33. When in a sad mood, I more often think about how my life could have been different.	S
34. When I feel sad, more thoughts of dying or harming myself go through my mind.	

LEIDS-R shared total: 1, 2, 3, 4, 6, 8, 9, 10, 11, 12, 14, 15, 16, 22, 24, 25, 27, 28, 31, 32, 33.
LEIDS-R: not shared: 5, 7, 9, 13, 17, 18, 20, 21, 23, 26, 29, 30, 34.

LEIDS 2000

Item	Shared or not
1. When I wake up, feeling down, it is usely nothing that day.	
2. I can only think positive when I am in a good mood.	S
3. When in a low mood, I take fewer risks.	S
4. When I feel sad, I spend more time thinking about what my moods reveal about me as a person.	S
5. When I am in a sad mood, I am more creative than usual.	S
6. When I feel down, I am more busy trying to keep images an thoughts away.	S
7. When I feel sad, I go out and do more pleasurable activities.	S
8. When I feel sad, I have less confidence in my future.	
9. When I feel sad, I am more helpful.	S
10. When I feel sad, I become more indifferent.	
11. When I feel sad, I am less inclined to express disagreement with someone else.	S
12. When in a sad mood, my thoughts always come back to the same subjects.	
13. When I feel somewhat depressed, I think I can permit myself fewer mistakes.	S
14. When in a low mood, I am more inclined to avoid difficulties or conflicts.	S
15. When I feel down, I have a better intuitive feeling for what people really mean.	S
16. When in a sad mood, I become more bothered by perfectionism.	S
17. When in a low mood I think less people will appreciate me.	
18. When in a sad mood, I more often think I can make no one happy.	
19. When I feel sad, I think more negative about things I achieved.	
20. I work harder when I feel down.	S
21. When I feel down, I often think how bad my life is in general.	
22. When I feel sad, it is of less interest what people think of me.	
23. When I feel sad, I find myself uglier.	

24. When I feel down, I more easily become cynical (blunt) or sarcastic.	S
25. When in a sad mood, I feel more like myself.	S
26. When I feel down, I more often neglect things.	S
27. When I am sad, I have more problems concentrating.	S
28. When in a sad mood, I am less creative.	
29. When in a low mood, I am nicer than usual.	S
30. When in a sad mood, I give myself more things to blame.	
31. When in a sad mood, I often think how terrible it is to have such moods.	
32. When I feel down, I am more inclined to want to keep everything under control.	S
33. When I feel sad, I spend more time thinking about the possible causes of my moods.	S
34. When in a sad mood, I more often think about how my life could have been different.	S

LEIDS 2000 shared total items: 2, 3, 4, 5, 6, 7, 9, 11, 13, 14, 15, 16, 20, 24, 25, 26, 27, 29, 32, 33, 34.
LEIDS 2000 not shared items: 1, 8, 10, 12, 17, 18, 19, 21, 22, 23, 28, 30, 31.

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