Cognitive reactivity, rumination, stressful life events in childhood, and their relationship as vulnerability factors to depression: A cross-sectional retrospective study.

Gunnlaugur Már Pétursson

Ritgerð til Cand.Psych. gráðu
Háskóli Íslands
Sálfræðideild
Heilbrigðisvísindasvið

HÁSKÓLI ÍSLANDS

Hugnæmi, grufl, áföll í æsku, og tengsl þeirra sem næmisþátta í þróun þunglyndis: Baksýn þversniðsrannsókn.

Gunnlaugur Már Pétursson

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Umsjónarkennari: Ragnar P. Ólafsson

Sálfræðideild

Heilbrigðisvísindasvið Háskóla Íslands

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Gunnlaugur Már Pétursson

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Supervisor: Ragnar P. Ólafsson

Faculty of Psychology

School of Health Sciences

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Útdráttur

Hugnæmi, grufl og áföll í æsku eru þekktir næmisþættir fyrir þunglyndi. Markmið þessa verkefnis voru að kanna fyrrnefnda næmisþættir hjá fólki með og án sögu um þunglyndi, kanna tengsl hugnæmis og grufls við fjölda og lengd fyrri geðlægðarlota, og hvort tengslum áfalla í æsku við þunglyndi síðar á æfinni gæti verið miðlað af grufli eða hugnæmi. Fólk með sögu um þunglyndi (*N*= 62) og fólk án slíkrar sögu (*N*= 64) var metið með tilliti til hugnæmis, grufls og áfalla í æsku. Þá voru fjöldi geðlægðarlota og lengd alvarlegustu lotu metin hjá þátttakendum með sögu um þunglyndi. Samkvæmt sjálfsmatskvörðum greindust þátttakendur með sögu um þunglyndi með marktækt meira hugnæmi og tilhnegingu til grufls en þátttakendur án sögu um þunglyndi. Ekki kom fram munur milli hópanna á hugnæmi metnu í tilraunaaðstæðum. Kynferðisleg misnotkun í æsku og ótilgreind áföll í æsku voru marktækt algengari meðal þátttakenda með sögu um þunglyndi. Grufl í formi þunglyndisþanka (e. brooding) en ekki íhugunar (e. reflective pondering) spáði fyrir um tíðni fyrri geðlægðarlota í línulegri aðfallsgreinigu. Þá spáði samvirkni milli hugnæmis og grufls í formi íhugunar fyrir um lengd alvarlegustu geðlægðarlotu. Miðlunarbreytugreining gaf til kynna að tengslum þess að verða fyrir líkamlegu ofbeldi í æsku við þunglyndi síðar á æfinni gæti verið miðlað af grufli í formi þunglyndisþanka.

Abstract

Cognitive reactivity, rumination and stressful life events (SLE) in childhood have all been associated with increased risk for depression. This study aimed to investigate these vulnerability factors in people with and without a history of depression, how cognitive reactivity and rumination relate to number and duration of past depressive episodes, and if the relationship between SLEs in childhood and depression later in life is mediated by rumination or cognitive reactivity. Formerly depressed (*N*= 62) and never depressed (*N*= 64) participants were assessed for cognitive reactivity as well as rumination and SLEs in childhood. Number of past depressive episodes and duration of index episodes were assessed within the formerly depressed group. The formerly depressed group reported significantly greater cognitive reactivity and rumination with self-report questionnaires compared to the never depressed group. There was no difference between the groups in mood-induced cognitive reactivity. Sexual abuse and unspecified SLEs were significantly more prevalent among formerly depressed participants. Regression analysis revealed that brooding but not reflective pondering predicted the frequency of past depressive episodes. Reflective pondering and cognitive reactivity interacted to predict duration of index episodes. Mediation analysis indicated that rumination may be a mediator between physical abuse in childhood and depression.

Table of contents

Wealth of evidence suggests that stressful life events (SLE) in childhood, including physical and sexual abuse (Lindert et al., 2014), emotional abuse (Liu, Alloy, Abramson, Iacoviello & Whitehouse, 2009), and parental loss due to death or separation (Agid et al., 1999; Weller, Weller, Fristad, & Bowes, 1991) are associated with increased risk for depressive disorders later in life. Research has also indicated a dose response relationship between the number of types of SLEs experienced in childhood and risk for depression later in life (e.g. Chapman et al., 2004; Felitti et al., 1998). Onset of first episode of depression is more often related to proximal stressors (e.g. marital divorce or loss of a job) than onset of later episodes, indicating that the role of stress in depression onset changes over the course of the disorder (for reviews see Monroe & Harkness, 2005; Stroud, Davila & Moyer, 2008). Although depression is related to stressful life events in childhood, such experiences are very common (Briere & Elliott 2003; Edwards, Holden, Felitti & Ansa, 2003) and clearly not all incidences of such events lead to depression later in life. The causal mechanisms linking SLEs to depression still remain poorly understood (Heim & Binder, 2012).

Traditional cognitive models of depression propose our interpretations, attitudes and other cognitive processes as moderating variables between life events and depression (e.g. Abramson, Seligman, & Teasdale, 1978; Beck, 1967). According to Beck's cognitive model (1967, 2008), people are vulnerable to depression if dysfunctional attitudes become embedded within cognitive schemas. Beck hypothesized that traumatic experiences in childhood and adolescence can lead to the development of such maladaptive schemas that may later emerge under circumstances sufficiently reminiscent of the original trauma. The model contends that, when activated, maladaptive schemas skew information processing and can lead to negative beliefs and thinking about oneself, the world and the future (i.e. the negative cognitive triad) and thus depression (Beck, 1967, 2008).

Although research has found evidence for increased dysfunctional attitudes in depressed people compared to control participants, such difference seems to become undetectable as depression remits (e.g. Eaves & Rush, 1984; Hamilton & Abramson, 1983). While this is in accordance with the diathesis-stress nature of schemas in Beck's theory, it leaves it open to question whether dysfunctional attitudes are mere symptoms of depression (e.g. Silverman, Silverman & Eardley, 1984). More recent research has indicated that dysfunctional attitudes increase to a greater extent in formerly depressed people compared to control participants following an experimental manipulation to induce sad mood (Lau, Haigh, Christensen, Segal, & Taube-Schiff, 2012; Miranda, Gross, Persons, & Hahn, 1998; Segal, Gemar, & Williams, 1999). This finding has been explained with Teasdale's (1988) differential activation

hypothesis, which contends that during depressive episodes an association is formed between depressed mood and negative thinking patterns, such that low mood will reactivate negative thinking in the remitted state, constituting a vulnerability to relapses in depression. The degree to which dysfunctional attitudes and negative thinking are activated in response to sad mood has been termed cognitive reactivity (Lau, Segal & Williams, 2004). Higher levels of cognitive reactivity are associated with greater risk of subsequent depressive relapses in some prospective studies (e.g. Segal et al., 2006; Segal et al., 1999) but not in others (e.g. Lethbridge & Allen, 2008; van Rijsbergen, et al., 2013)

The response styles theory of depression (Nolen-Hoeksema, 1991) proposes rumination as an important vulnerability factor to depression. The theory defines rumination as a mode of thinking that involves "repetitively focusing on the fact that one is depressed; on one's symptoms of depression; and on the causes, meanings, and consequences of depressive symptoms" (Nolen-Hoeksema, 1991, p. 569). The response styles theory contends that rumination prolongs the duration of depressed mood making individuals with a ruminative response style prone to longer episodes of depression. Nolen-Hoeksema (1998) theorized that those who perceive little control over their environment in childhood and those deprived of the opportunity to learn more active coping strategies may be especially prone to rumination. Although empirical research on the developmental antecedents of rumination has been relatively scarce, studies show that rumination is linked to over-controlled parenting (Nolen-Hoeksema, Wolfson, Mumme & Guskin, 1995; Spasojevic & Alloy, 2002) and SLEs in childhood, such as sexual- and emotional abuse (e.g. Conway, Mendelson, Giannopoulos, Csank, & Holm, 2004; Sarin & Nolen-Hoeksema, 2010). Some of these studies support rumination as a mediator in the relationship between sexual- and emotional abuse and depression (Paredes & Calvete, 2014; Raes & Hermans, 2008; Spasojevic & Alloy, 2002). Rumination may also mediate the relationship between depression and more proximal significant stressors such as bereavement (e.g. Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Parker & Larson, 1994). For example, Michl, McLaughlin, Shepherd and Nolen-Hoeksema (2013) found that rumination mediated the relationship between recent stressors (e.g. divorce and serious illness or injury of a family member) and symptoms of depression assessed one year later.

While rumination consistently predicts onset of depressive episodes (Nolen-Hoeksema, 2000; Just & Alloy, 1997; Spasojevic & Alloy, 2001) it may not predict their duration (Just &Alloy, 1997; Lara, Klein & Kasch, 2000; Nolen-Hoeksema, 2000; but see Kuehner & Weber, 1999). Several possible explanations have been proposed for this (Nolen-Hoeksema,

Wisco & Lyubomirsky, 2008). First, recent evidence indicates that rumination may be constituted of two distinct subtypes termed brooding, which involves dwelling on the negative consequences of low mood, and reflective pondering, which involves attempting to understand the reasons for low mood (Treynor, Gonzales & Nolen-Hoeksema, 2003) which may have differential effects in depression (e.g. Burwell & Shirk, 2007; Surrence, Miranda, Marroquín & Chan, 2009). Brooding involves a tendency to dwell Thus, measurements of rumination in previous research may have confounded potentially adaptive (reflective pondering) with a more maladaptive (brooding) cognitive process. Also, the effect of rumination may be moderated by other factors. Rumination may for example contribute to clinical depression by amplifying a bidirectional circuit between depressed mood and negative cognition such as dysfunctional attitudes (Lyubomirsky, Layous, Chancellor & Nelson, 2015; Nolen-Hoeksema, 1991; Teasdale,1999). In line with this hypothesis two prospective studies have found that the interaction of rumination and negative cognitive styles predicts both the number and duration of depressive episodes (Ciesla & Roberts, 2002; Robinson & Alloy, 2003), suggesting that the combination of negative cognition and rumination may constitute a maintenance factor in major depression. Other studies support the interaction between rumination and negative cognitive styles in the prediction of depressive symptoms (Ciesla & Roberts, 2007; Ciesla et al., 2011). Of note, in the studies that have found rumination and negative cognitive styles to interact in predicting relapse and duration of depressive episodes (Ciesla & Roberts, 2002; Robinson & Alloy, 2003) negative cognitions, including dysfunctional attitudes, have been measured in the absence of a mood induction procedure.

Aims of the present study

Even though cognitive reactivity and rumination are among the most researched cognitive vulnerability factors to depression, they have seldom been subject to the same study. While they may conceptually overlap, cognitive reactivity and rumination are also distinct constructs, and their comparison and potential interaction as vulnerability factors to depression remain largely unexplored. The first aim of this study is to replicate the results of previous research (e.g. Lau et al., 2012; Moulds, 2008) indicating that euthymic people with a history of depression have more cognitive reactivity and more rumination than people with no such history. Second, based on theory and previous research findings, we may expect cognitive reactivity and rumination to relate to the number and duration of past depressive episodes in a certain way. First, if cognitive reactivity develops as described by Teasdale's (1988) differential activation hypothesis, we would expect cognitive reactivity to correlate

both with number and duration of past depressive episodes. To date, published empirical evidence that cognitive reactivity develops as described by Teasdale (1988) is scarce. Secondly, based on the response styles theory (Nolen-Hoeksema, 1991) and previous research (e.g. Nolen-Hoeksema, 2000), formerly depressed people that have a greater tendency to ruminate in response to low mood, should have a history of longer and more frequent episodes of depression, although brooding and reflective pondering might have a differential effect in this respect (e.g. Burwell & Shirk, 2007; Surrence et al., 2009). The effect of rumination on the frequency and duration of depressive episodes may also be moderated by cognitive reactivity or dysfunctional attitudes (e.g. Robinson & Alloy, 2003). Third, as indicated by previous research we may expect SLEs to be more common among people with a history of depression than people with no such history (e.g. Agid et al., 1999; Lindert et al., 2014; Liu et al., 1999) and that the relationship between SLEs and depression is mediated by rumination (e.g. Paredes & Calvete, 2014; Spasojevic & Alloy, 2002). Cognitive reactivity or dysfunctional attitudes may also mediate the relationship between SLEs and depression as implied by Beck's cognitive model (Beck, 1967).

Method

Participants

In total, 126 paricipants (81% female) were recruited from two populations. First, a student sample was recruited via e-mail that was sent to all listed students in the University of Iceland. Second, a sample from the general population was recruited via advertisements in media and social media. The e-mail and the advertisements directed participants to a dedicated website, containing information about the research along with a link to a short internet survey assessing general exclusion and inclusion criteria in the study. Potential participants were contacted via telephone. 185 individuals participated in an assessment interview, of which 59 were excluded from further participation. General exclusion criteria included current major depressive episode and moderate or severe depression symptoms (score of 14 or greater on the BDI-II). Participants had to be between 18 and 65 years of age. This study is a part of a larger research project which included the following exclusion criteria not directly relevant to the present study: Pregnancy, cardiovascular diseases, diabetes, tachyarrhythmia and hypertension.

Formerly-depressed participants. 62 formerly depressed (FD) individuals participated, of which 37 (89% female) where university students (age *M*= 29.5, *SD*= 8.25) and 25 (76% female) from the general population (age *M*= 37.4, *SD*= 13.49). Inclusion criteria in the FD group included a history of at least one major depressive episode and little or no symptoms of depression present during the past two months. Exclusion criteria in the FD group included any of the following diagnoses as evaluated in the MINI diagnostic interview; current major depressive episode, current or past manic or hypomanic episodes, current or past psychotic disorders and alcohol or substance dependency within the last 12 months.

Never-depressed control participants. 64 individuals with no history of depression participated of which 48 (81% female) where university students (age *M*= 26.5, *SD*= 8.31) and 16 (69% female) from the general population (age *M*= 38.6, *SD*= 10.94). Exclusion criteria in the never-depressed (ND) group included the precence or history of any mental disorders as evaluaded in the MINI diagnostic interview.

Measures

Demographic information. Participants filled out a simple questionnaire on demographic variables, including age, gender, marital status, level of education and employment status.

Mini-International Neuropsychiatric Interview (MINI; Sheehan et al., 1998). MINI is a semi structured interview for the most common Axis I disorders of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2000). Good interrater reliability and good convergent validity with lengthier diagnostic interviews such as the Composite International Diagnostic Interview (CIDI) have been reported for MINI (Sheehan et al., 1997). An Icelandic version of MINI was administered for which adequate convergent validity with CIDI has been measured (Sigurðsson, 2008). The present study utilized a composite version of MINI with the depression module from MINI-Plus but other modules from the standard MINI. Questions were added to assess number of past depressive episodes, duration of the index episode, as well as age of first episode onset. The reliability of information on the number of depressive episodes was assessed on a four point rating scale designed by the researchers of which the values were: 0 (unreliable: number of episodes unclear), 1 (possibly unreliable: Participant had great difficulty reporting the number of episodes), 2 (fairly reliable: Participant had some difficulty reporting the number of episodes), 3 (reliable: participant had no problems reporting information on past depressive episodes).

The Beck Depression Inventory—2nd edition (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a widely used 21-item self-report measure of the presence and severity of depressive

symptoms over the past two weeks. An Icelandic version of BDI-II was administered for which good psychometric properties have been measured (Arnarson, Ólason, Sári & Sigurðsson, 2007).

The Dysfunctional Attitudes scale (DAS; Weissman & Beck, 1978). The DAS is a 40-item self-report measure of dysfunctional attitudes, reflective of a rigid world view, perfectionistic performance standards and over-concern with judgement of others. Respondents rate their agreement with each statement on a scale from 1 (totally disagree) to 7 (totally agree). Possible total scores range from 40 to 280 with higher scores indicating more dysfunctional attitudes. Good internal consistence and test-retest reliability have been reported for the DAS (Hammilton & Abramson, 1983; Weissman, 1979). The DAS exists in two equivalent forms, DAS-A and DAS-B, which have generally been used in research on mood induced cognitive reactivity (e.g. Segal et al., 2006). DAS-A and DAS-B were administered in Icelandic translations that show good internal consistency estimates and a strong inter-correlation (Guðmundsdóttir & Rögnvaldsdóttir, 2015).

The Leiden Index of Depression Sensitivity Revised (LEIDS-R; Van der Does, 2002; Van der Dos & Williams, 2003). LEIDS-R is a 34-item self-report measure of cognitive reactivity to sad mood. The items describe particular forms of thinking in response to sad mood and are categorized into six subscales (aggression, hopelessness/suicidality, acceptance/coping, control/perfectionism, risk aversion and rumination). Respondents rate how well each item applies to them on a scale from 0 (not at all) to 4 (very strongly). Total scores range from 0 to 136 with higher scores indicading more cognitive reactivity. Adequate validity and reliability has been reported for the LEDS-R (Williams, Van der Does, Barnhofer, Crane & Segal, 2008) although there are no published studies validating the subscales. An Icelandic version of LEIDS-R was administered which has been measured with similar internal consistency to the original version (Ellertsdóttir, 2015).

The Ruminative Response Scale of the *Response Styles Questionnaire* (RRS; Nolen-Hoeksema & Morrow, 1991). The RRS is 22-item self-report measure of the tendency to ruminate in response to dysphoric mood. The items describe responses to dysphoric mood that are focused on the self, the symptoms of the depressed mood and the the possible causes and of consequences of the depressed mood. Respondents rate each item on a scale from 1 (almost never) to 4 (almost always). Possible total scores range from 22 to 88 with higher scores indicating a stronger tendency to ruminate in response to dysphoric mood. Treynor, Gonzales and Nolen-Hoeksema (2003) found that 10 of the 22 items comprise two 5 item factors termed “brooding” and “reflective pondering”. Total scores for each of these two subscales

range from 5 to 20. An Icelandic version of the RRS was administered which has been measured with comparable psychometric properties to the original version (Pálsdóttir & Pálsdóttir, 2008).

The Childhood Traumatic Events Scale (CTES; Pennebaker & Susman, 1988). The CTES is a 6-item self-report measure of the experience of traumatic life events in childhood and adolescence. The CTES allows respondents to report 6 types of traumatic events (death of a friend or family member, divorce or other major upheaval between parents, sexual abuse, physical abuse or assault, major illness or injury, any other major upheaval). For each type of traumatic events, respondents report whether or not they experienced it before the age of 17 years, their age at the time of the event, and how traumatic the event was on a scale from 1 (not at all) to 7 (extremely traumatic). Good reliability and validity has been reported for the original version of the CTES (Pennebaker & Susman, 1988). The CTES was translated to Icelandic by Ragnar P. Ólafsson especially for this research. The psychometric properties of the Icelandic version are unknown.

Mood induction

The mood induction procedure (MIP) involved a combination of music and autobiographical recall. The orchestral introduction by Prokofiev entitled "Russia under the Mongolian Yoke", from the film *Alexander Nevsky*, was played for participants at half speed trough headphones. During the approximately 8 minutes they were asked to recall a time in their lives when they felt sad. Both before and after the MIP participants rated their current mood with a horizontal *Visual Analogue Scale* (VAS) measuring 75 mm from the center to each endpoint. The left end of the scale was labeled "sad" and the right end was labeled "happy". This method for induction and measurement of mild and transient sad mood has proved effective in previous research (e.g. Martin, 1990; Segal et al., 1999; Segal et al., 2006).

Procedure

This study is a part of a larger research project. Some of the measures mentioned in the procedure are irrelevant to the present study and are therefore not discussed in detail.

Assessment session. Upon arrival to the assessment session, informed consent was obtained from participants, before they provided demographic information about themselves and completed number of self-report measures that were administered in a counterbalanced order, including BDI-II and CTES as well as the *Beck Anxiety Inventory* (BAI), *Difficulties in Emotion Regulation Scale* (DERS), *Emotion Reactivity Scale* (ERS) and the *Recent Traumatic*

Events Scale (RTES). Then, a short semi structured interview for participant's treatment history was administered. Finally, a modified version of MINI was administered. Participants that did not meet any of the exclusion criteria were booked for a second session within a week. Participants that were excluded at this stage were compensated for their participation with 2000 ISK. The duration of the assessment session was approximately 1.5 hours.

Experimental testing session. In the beginning of the second session, a number of self-report measures were administered in a counterbalanced order, including LEIDS-R and RRS as well as the *Penn State Worry Questionnaire* (PSWQ), *Positive and Negative Affect Schedule* (PANAS), *Self-Report Habit Index* (SHRI) and the *Distress Scale* (DS14). Then, participants completed a series of computerized tasks designed to measure the tendency for habitual responding (for a detailed description see Gillan et al., 2011). Following that, participants completed the DAS (form A or B) and rated their current mood with the VAS scale. During the MIP heartrate variability was measured which involved placing electrodes on participants after the first administration of VAS. Then participants watched a wildlife documentary for 10 minutes during a baseline measurement of heart rate variability. After that the MIP was conducted as described above. Immediately following the MIP participants again rated their mood with the VAS scale and completed the other form of DAS. The administration of the two forms of DAS was counterbalanced. Before participants left they were compensated for their participation with 5000 ISK. The duration of the experimental testing session was approximately 2 hours.

Statistical analysis

Mediation analysis was carried out in SPSS with the PROCESS macro written by Andrew F. Hayes. Multiple mediation models were tested for each type of SLEs assessed by the CTES as an independent variable, group (ND, FD) as the independent variable in all models and cognitive vulnerability factors entered simultaneously as moderators in each model. As recommended (Cerin & MacKinnon, 2009; Hays, 2009) mediation analysis was carried out for all types SLEs regardless of wither they significantly predicted history of depression or not. Indirect effects were tested with a bootstrapping approach generating bias-corrected 95% confidence intervals based on 5000 samples. Effect sizes are not reported for indirect effects since the mediation models are based on both simple linear regression and binary logistic regression.

Results

Sample characteristics

Table 1 displays demographic variables by group. Of note is the high proportion of females and students in the sample. The groups are similar on demographic variables although they differ significantly in level of education, $\chi^2(2, N=126) = 6.2$, $p < .05$.

Table 1. Sample characteristics by group.

Variable	ND group (n= 64)	FD group (n= 62)
Gender, women: *n* (%)	50 (78)	52 (84)
Age (in years): *M* (*SD*)	29.5 (10.4)	32.7 (11.3)
Marital status: *n* (%)		
Single	27 (42)	25 (40)
In a relationship	19 (30)	19 (31)
Married or cohabiting	18 (28)	18 (29)
Level of education: *n* (%)		
Secondary school	1 (1.5)	4 (6.5)
Upper secondary school level	40 (62.5)	26 (42)
University degree	23 (40)	32 (51.5)
Employment status: *n* (%)		
Student	50 (78)	38 (61)
Full time job	13 (20.5)	18 (29)
Part time job	0 (0)	3 (5)
Unemployed	1 (1.5)	3 (5)
History of depressive episodes: *M* (*SD*)		
Age of onset (in years)	—	19.7 (9.2)
Number of episodes[a]	—	2.5 (1.6)
Duration of index episode (in weeks)	—	31.5 (32.2)
Other psychiatric disorders: *n* (%)		
Aniety disorders	—	12 (19)
Other disorders	—	1 (1.6)

Notes. ND = never depressed, FD = formerly depressed.

[a]*M* and *SD* for number and duration of depressive episodes is based on 47 participants.

Cognitive vulnerability factors

BDI-II scores were significantly higher in the FD group compared to the ND group, $t(124) = 5.26$, $p < .001$. Potential influence of current depressive symptomology on the measurement of cognitive vulnerability factors will thus be controlled for in the statistical analysis. Means and standard deviations for all questionnaire measures are displayed by group in table 2.

Table 2. Means and standard deviations for all questionnaire measures by group.

Measure: *M* (*SD*)	ND group	FD group
BDI-II	3.5 (3.3)	6.8 (3.9)
Mood (VAS rating)		
baseline	11.6 (2.19)	11.46 (2.52)
post mood induction	9.3 (3.0)	8.8 (3.0)
DAS		
baseline	98.4 (21.7)	113.8 (26.1)
post mood induction	98.8 (22.2)	111.6 (26.3)
LEIDS-R	29.7 (14.4)	48.9 (14.9)
RRS		
Total (10 items)	14.7 (3.4)	21.6 (5.5)
brooding	7.0 (1.5)	10.4 (3.1)
reflection	7.7 (2.7)	11.1 (3.5)

Note. ND = never depressed, FD = formerly depressed, BDI-II = Beck Depression Inventory–2nd edition, VAS = Visual Analogue Scale, DAS = Dysfunctional Attitudes Scale, LEIDS-R = Leiden Index of Depression Sensitivity–Revised, RRS = Ruminative Response Scale.

Dysfunctional attitudes at baseline

Analysis of covariance (ANCOVA) revealed that, controlling for current BDI-II scores, the FD group had significantly higher DAS scores at baseline compared to the ND group, $F(1, 123)= 7.13$, $p< 0.01$, $\eta_p^2= 0.055$, 95% CI [3.28; 22.05]. This implies that euthymic people with a history of depression have more dysfunctional attitudes than people with no history of depression.

Mood manipulation check

A mixed 2 (group: ND, FD) x 2 (time: baseline, post mood induction) repeated measures analysis of variance (ANOVA) revealed a significant main effect of time, $F(1, 123)= 109.13$, $p< 0.001$, $\eta_p^2= 0.47$, no significant main effect of group, $F(1, 123)= 0.61$, $p= 0.436$, and no significant interaction between group and time, $F(1, 123)= 0.60$, $p= 0.424$. This implies that the mood induction procedure was successful in producing changes in self-rated mood, and that it was similarly effective among people with a history of depression as people with no such history.

Cognitive reactivity following mood induction

A mixed design 2 (time: baseline DAS, post DAS) x 2 (group: ND, FD) repeated measures ANCOVA, controlling for BDI-II scores, revealed no significant effect of time, $F(1, 122)= 3.30$, $p= 0.072$, and no interaction between group and time, $F(1, 122)= 2.72$, $p= 0.102$. Thus the data does not indicate more cognitive reactivity following induction of sad mood, among euthymic people with a history of depression than among people with no such history.

Cognitive reactivity measured with the LEIDS-R

An ANCOVA, controlling for current BDI-II scores, revealed that the FD group had significantly higher LEIDS-R scores compared to the ND group, $F(1, 123)= 30.94$, $p< 0.001$, $\eta_p^2= 0.201$, 95% CI [10.02; 21.10]. This indicates that euthymic people with a history of depression have greater self-reported cognitive reactivity than people with no history of depression.

Rumination

An ANCOVA, controlling for current BDI-II scores, revealed that the FD group scored significantly higher on both the RRS-brooding scale, $F(1, 123)= 44.55$, $p< 0.001$, $\eta_p^2= 0.266$, 95% CI [2.23; 4.11], and RRS-reflection scale, $F(1, 123)= 31.11$, $p< 0.001$, $\eta_p^2= 0.202$, 95% CI [2.21; 4.64]. This indicates that, compared to people with no history of depression, formerly depressed euthymic people have a greater tendency to engage in both brooding and reflective pondering in response to sad mood.

Number and duration of depressive episodes

Participants that had major difficulties in providing information on number of past depressive episodes in the assessment interview ($n= 13$), as well as two extreme outliners in duration of index episode (10 and 5 years respectively), were excluded from data analysis on the number and duration of depressive episodes. Since the groups differed significantly on DAS baseline scores, the relationship between dysfunctional attitudes to number and duration of depressive episodes was examined. Means and standard deviations for the number and duration of depressive episodes among the remaining 47 participants are displayed in table 1. Table 3 displays a correlation matrix of the number of depressive episodes, duration of the index episode and the cognitive risk factors among the 47 participants. Of note is that LEIDS-R scores do not significantly correlate with number or duration of depressive episodes. Neither did LEIDS-R scores differ significantly between participants with a history of one depressive

episode compared to those with a history of more than one episode, $t(45) = .14$, $p = .89$. Another unexpected finding is the negative correlation between baseline DAS scores and number of episodes. Also of note is that all the cognitive vulnerability factors are correlated except RRS reflection and DAS baseline scores.

Table 3. Correlation matrix of number and duration of major depressive episodes and cognitive risk factors within the FD group.

	1	2	3	4	5	6
1. RRS reflection	—					
2. RRS brooding	.542**	—				
3. LEDS-R	.401**	.586**	—			
4. DAS baseline	.059	.297**	.444**	—		
5. Duration of index episode	-.038	-.038	.128	-.043	—	
6. Number of episodes	.105	.005	.113	-.324*	-.061	—

Notes. FD = formerly depressed, RRS = Ruminative Response Scale, LEIDS-R = Leiden Index of Depression Sensitivity–Revised, DAS = Dysfunctional Attitudes Scale.

*$p < 0.05$, ** $p < 0.01$.

Linear regression analysis was conducted to test the hypothesis that cognitive risk factors predict the frequency and duration of depressive episodes. A summary of the results is shown in table 4. In the analysis of frequency of depressive episodes, we controlled for time from first onset of depression along with BDI-II scores. When the cognitive vulnerability factors were regressed simultaneously on the number of episodes, brooding had a significant effect but not reflection (Model 1). DAS baseline scores had the strongest effect in predicting the number of episodes – but in the unexpected direction. Simpler models were also fitted and the effect of brooding became insignificant ($p = .18$) when DAS baseline scores were omitted, but the pattern of brooding having a stronger relation with the frequency of depressive episodes ($\beta = .212$) than reflection ($\beta = .082$) was still evident. No interaction terms were significant when individually added to model 1.

None of the cognitive risk factors had a significant main effect when regressed simultaneously on the duration of index episodes (model 2). When interaction terms were individually added to model 2, RRS reflection interacted with LEIDS-R such that increased reflective pondering predicted longer episodes when cognitive reactivity was high but shorter episodes when cognitive reactivity was low. No other interaction effects were significant when individually added to model 2. A simplified model containing the interaction effect is displayed as model 3 in table 4 and a plot of the interaction effect is depicted in figure 1.

Table 4. Multiple linear regression models with cognitive vulnerability factors as predictors of the number and duration of past depressive episodes.

	b	*SE*	*β*	*t*	*p*	ΔR^2
Number of episodes						
Model 1 (R^2= .49)						
(intercept)	2.30	1.22				
RRS reflection	-.040	.057	-.094	1.28	.49	.01
RRS brooding	.204	.083	.364	2.45	.02	.08
LEIDS-R	-.003	.014	-.034	.25	.81	.00
DAS baseline	-.026	.008	-.425	3.11	<.001	.13
TFO	.121	.025	.568	4.89	<.001	.31
BDI-II	.058	.047	.147	1.23	.22	.02
Duration of index episode						
Model 2 (R^2= .085)						
(intercept)	58.76	31.73				
RRS reflection	-.625	1.55	-.071	.40	.69	.01
RRS brooding	-.118	2.25	-.010	.05	.96	.00
LEIDS-R	.403	.38	.193	1.06	.30	.03
DAS baseline	-.195	.22	-.155	.86	.40	.02
BDI-II	-2.113	1.29	.258	1.64	.11	.07
Model 3 (R^2= .17)						
intercept	162.70	57.29				
RRS reflection	-11.21	4.97	-1.276	-2.25	.03	.10
RRS brooding	-.942	2.01	-.081	.47	.64	.05
LEIDS-R	-2.35	1.19	-1.137	1.97	.05	.08
BDI-II	-1.59	1.19	-.199	1.34	.18	.04
RRS reflection *LEIDS-R	.24	.10	.027	2.32	.03	.11

Note. RRS = Ruminative Response Scale, LEIDS-R = Leiden Index of Depression Sensitivity–Revised, DAS = Dysfunctional Attitudes Scale, TFO = Time from first depression onset, BDI-II = Beck Depression Inventory–2nd edition.

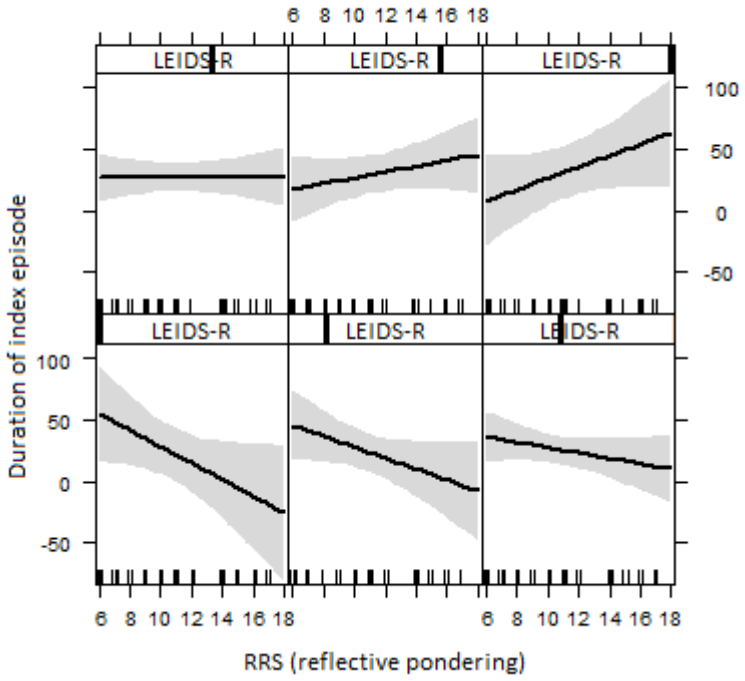

Figure 1. Interaction in linear regression analysis between reflective pondering and cognitive reactivity predicting duration of depressive episodes. The plot shows the estimated effect of reflective pondering on duration of index episode at 6 different points on the LEIDS-R scale. Lower points on LEIDS-R are depicted in the bottom row and higher points in the top row, in ascending order from left to right in each row.

Stressful life events in childhood and mediation analysis

The number and proportion of participants that experienced each type of stressful life events included in CTES is displayed by group in table 5. Although all types of stressful life events, except death of a friend or family member, were more prevalent in the FD group, significant group difference was only observed for sexual abuse, $t(124)= 2.80$, $p< .01$, and unspecified SLEs, $t(124)= 3.06$, $p< .01$.

Table 5. Stressful life events experienced before the age of 17 by group.

Type of SLE: *n* (%)	ND group	FD group
Death of a friend or family member	36 (56)	35 (56)
Divorce or major upheaval between parents	14 (22)	20 (32)
Sexual abuse	7 (11)	19 (31)
Physical abuse or assault	7 (11)	11 (18)
Major illness or injury	8 (13)	10 (16)
Unspecified SLE	13 (20)	28 (45)

Note. SLE = Stressfull life event, ND = Never depressed, FD = Formerly depressed.

Regression analysis was used to test the hypotheses that the connection between SLE in childhood and depression later in life is mediated by cognitive risk factors. Multiple mediator analysis was conducted for each type of SLEs with RRS scores, DAS baseline scores, and LEIDS-R scores as mediating variables, and group (ND, FD) as dependent variable. BDI-II scores were used as a covariate in all regressions to control for current depressive symptoms. A summary of the significant mediator models, containing effect-coefficients and 95% confidence intervals for the indirect effects, is displayed in table 6. Physical abuse predicted

Table 6. Summary of multiple mediator analyses.

Indipendent variable	Moderator variable	Dependent variable	Control variable	Effect of IV on M	Effect of M on DV	Direct Effect	Indirect effect	
(IV)	(M)	(DV)	(CV)	(a)	(b)	(c')	(axb)	95% CI
Physical abuse	RRS ref.	Group (ND or FD)	BDI-II	2,05*	.23**	.00	.48*	[.03; 1.48]
	RRS bro.			1,77*	.50**		.88	[-.02; 3.15]
	LEIDS-R			5,70	.02		.14	[-.12; .83]
	DAS			-7,24	.02		-.13	[-.11; .86]
Unspecified SLE	RRS ref.	Group (ND or FD)	BDI-II	1,70*	.21*	.91	.36*	[.04; 1.07]
	RRS bro.			1,40**	.51**		.71*	[.06; 2.13]
	LEIDS-R			6,45*	.02		.13	[-.18; .83]
	DAS			3,33	.02		.06	[-.11; .47]

Note. RRS ref. = Ruminative Response Scale – reflective pondering, RRS bro. = Ruminative Response Scale – brooding, LEIDS-R = Leiden Index of Depression Sensitivity–Revised, DAS = Dysfunctional Attitudes Scale, ND = never depressed, FD = formerly depressed, BDI-II = Beck Depression Inventory 2nd edition.
*p< 0.05, ** p< 0.01.

both RRS reflection (p= .02) and brooding scores (p= .01) but not LEIDS-R (p= .15) or DAS baseline scores (p= .24). Physical abuse had no direct effect when it was regressed on the dependent variable along with the proposed mediating variables indicating full mediation by the model. The indirect effect was only significant in the case of RRS reflection scores (p< .05) which implies that the relationship between physical abuse and depression may be mediated by reflective pondering. Unspecified SLEs predicted RRS reflection (p= .01) and brooding scores (p<.01), as well as LEDS-R scores (p= .032), but not DAS baseline scores (p= .48). The direct effect of unspecified SLE was insignificant (p= .15) in the mediator model indicating full mediation. The indirect effect was significant for both RRS reflection

($p < .05$) and brooding ($p < .05$) but not LEIDS-R or DAS baseline scores. Thus, the data implies that the effect of unspecified SLEs on depression may be fully mediated by reflective pondering and brooding. None of the other types of SLEs, including sexual abuse, predicted any of the cognitive vulnerability factors.

Discussion

The first aim of this study was to replicate previous research findings that formerly depressed euthymic people have more cognitive reactivity and more rumination compared to never depressed controls. Formerly depressed participants reported more rumination, even when current depressive symptomology was controlled for, and this was the case for both the brooding and reflective pondering factors of the RRS. In line with prior research (e.g. Van der Does, 2005; Moulds, 2008) cognitive reactivity was greater among formerly depressed participants than never depressed controls as measured by the LEIDS-R. On the other hand, there was no difference between the groups in cognitive reactivity as measured by changes in DAS scores following a mood induction procedure, which was unexpected in light of previous research (e.g. Lau et al., 2012; Miranda et al., 1998) but not unprecedented (Brosse, Craighead & Craighead, 1999; Van der Does, 2005). Potential explanations for such null results have previously been proposed, including differences in sample composition between studies (Fresco, Heimberg, Abramowitz & Bertram, 2006) and the potential unreliability of DAS as a measure for mood-induced cognitive reactivity (Van der Does, 2005). Another explanation that has been discussed (Figueroa, et al., 2015) is that in some studies dysfunctional attitudes may get primed during the research procedure before the mood induction begins. All of these explanations might apply to the present study. First, the majority of the sample was composed of university students and student samples (e.g. Brosse, et al., 1999; Freco et al., 2006; Van der Does, 2005) seem to be more associated with null results in cognitive reactivity studies with a MIP than outpatient or community samples (e.g. Lau et al., 2012; Miranda et al., 1998). Second, the formerly depressed group had significantly higher DAS scores at baseline compared to the control group, even when controlling for current depressive symptomology. Such has usually not been the case in prior research and may indicate that participants were already primed before the mood induction procedure. However, these explanations are speculative and it is unclear why cognitive

reactivity as measured by changes in DAS scores following a mood induction procedure was not observed in this study.

The second aim was to investigate how the cognitive vulnerability factors examined in this study relate to the number and duration of past depressive episodes within the formerly depressed group. First, in light of Teasdale's differential activation hypotheses (1988) it was expected that cognitive reactivity would correlate with number and duration of past depressive episodes. This was not observed since LEIDS-R scores correlation with number of past depressive episodes, as well as duration of index episode, where weak and insignificant. Previous research results indicating increased cognitive reactivity among those that have experienced more than one depressive episode compared to those that have experienced only one episode (Elgersma et al., 2015) was not replicated in this study. Published empirical evidence that cognitive reactivity develops as described by Teasdale (1988) is scarce and research indicating that cognitive reactivity predicts first onset of depression (Kruijt et al., 2013) implies that cognitive reactivity (at least as measured by the LEIDS-R) may develop in other ways. Secondly, cognitive vulnerability factors were expected to be associated with the frequency of past depressive episodes. Results of multiple linear regression analysis implied that rumination in the form of brooding but not reflective pondering was associated with the frequency of depressive episodes. This is in line with research indicating that brooding is more maladaptive than reflective pondering (e.g. Burwell & Shirk, 2007; Surrence et al., 2009). The relationship between rumination and frequency of depressive episodes was not moderated by dysfunctional attitudes or cognitive reactivity as might have been expected in light of prior research (Ciesla & Roberts, 2002; Robinson & Alloy, 2003). LEIDS-R scores where not associated with the frequency of depressive episodes which was unexpected since cognitive reactivity has primarily been discussed as a risk-factor for relapse in depression (e.g. Segal et al., 1999; Kuyken et al., 2010). Another unexpected result was a rather strong negative association between dysfunctional attitudes and frequency of depressive episodes. This finding is difficult to put into context but it may potentially indicate that dysfunctional attitudes tend to decrease over the course of the disorder. Thirdly, although none of the cognitive vulnerability factors had a significant main effect in predicting duration of index episode, reflective pondering and cognitive reactivity interacted to predict duration of index episodes. Reflective pondering was found to predict longer episodes when cognitive reactivity is low but shorter episodes when cognitive reactivity is high. This result is in line with prior research (Ciesla & Roberts, 2002; Robinson & Alloy, 2003) although those studies have used other measures of rumination and negative cognition. Thus, reflective pondering may be an

adaptive problem-solving process – but only when it is not dominated by negative cognitive content.

The third aim of this study was to investigate whether cognitive vulnerability factors may be mediators in the relationship between significant SLEs in childhood and depression later in life. As expected (Chapman et al., 2004; Felitti et al., 1998), stressful life events as assessed by the CTES were generally more prevalent among the formerly depressed group compared to the never depressed control participants. Group difference reached significance only in sexual abuse and unspecified SLEs and the biggest non-significant group difference was observed in the case of physical abuse. This pattern fits previous research results in that physical- and sexual abuse, along with emotional abuse, are the types of SLEs that have most consistently been connected with increased risk for depression (Chapman et al., 2004; Felitti et al., 1998; Lindert et al. 2014; Liu et al., 2009). The finding that unspecified SLEs are more prevalent among the formerly depressed group is more difficult to interpret. Since emotional abuse has been strongly related to depression onset later in life, and the CTES does not assess emotional abuse specifically, it is possible that some of the group difference in unspecified SLEs is explained by emotional abuse. However, it cannot be ruled out that the group difference in unspecified SLEs is to some extent explained by memory biases, or differences in interpretation of life events between the formerly depressed and never depressed participants. The mediation analysis indicated that the connection between physical abuse in childhood and depression later in life may be mediated by reflective pondering – but not brooding, dysfunctional attitudes or cognitive reactivity. This implies that children that experience physical abuse are more likely to develop a ruminative response style which in turn makes them more likely to experience depression later in life. There is no theory or research results explaining separate developmental paths for brooding and reflective pondering and thus it is difficult to speculate on why the connection between physical abuse in childhood and depression later in life might be mediated by reflective pondering rather than brooding. Results also indicated that the relationship between unspecified SLEs in childhood and depression later in life is mediated by both brooding and reflective pondering – but not dysfunctional attitudes or cognitive reactivity. This finding is hard to interpret and does not support any conclusions, but to the extent that indices of emotional abuse in childhood may be represented by unspecified SLEs in this study, mediation by rumination would be in line with prior research (Paredes & Calvete, 2014; Raes & Hermans, 2008; Spasojevic & Alloy, 2002). Results of research indicating that rumination may mediate the relationship between

sexual abuse in childhood and depression later in life (e.g. Spasojevic & Alloy, 2002) were not replicated.

Limitations

A major limitation of this study is the cross-sectional retrospective research design. This most notably limits conclusions on the causal mechanisms behind the observed associations between cognitive vulnerability factors and number and duration of depressive episodes. The same applies to the mediation analysis. Another noteworthy limitation is that females and students are overrepresented in the sample which possibly limits the generalizability of results.

Conclusions

Euthymic people with a history of depression have more cognitive reactivity (at least as measured by the LEIDS-R) and a greater tendency to ruminate in response to low mood compared to people with no history of depression. Brooding may be more associated with depressive relapses than reflective pondering. Reflective pondering and cognitive reactivity may interact to predict duration of depressive episodes, indicating that reflective pondering may be an adaptive response to depressed mood when cognitive reactivity is low but maladaptive when cognitive reactivity is high. Rumination may be a mediator in the relationship between physical abuse in childhood and depression later in life.

Future Directions

It remains unclear whether cognitive reactivity develops as sequelae of experiencing depression as described by Teasdale (1988) or if it develops in other ways and may thus constitute a vulnerability factor to first depression onset. More prospective studies in high-risk samples of never-depressed people are needed to answer this outstanding question. The finding that cognitive reactivity and reflective pondering may interact to predict duration of depressive episodes needs to be replicated in prospective studies.

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