Journal of Affective Disorders 188 (2015) 22–27

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research report

Transdiagnostic mechanisms in depression and anxiety: The role of rumination and attentional control

Kean J. Hsu [a,b,*], Courtney Beard [a,b], Lara Rifkin [a], Daniel G. Dillon [b,c], Diego A. Pizzagalli [b,c], Thröstur Björgvinsson [a,b]

[a] Behavioral Health Partial Program, McLean Hospital, Belmont, MA, United States
[b] Department of Psychiatry, Harvard Medical School, Boston, MA, United States
[c] Center for Depression, Anxiety and Stress Research, McLean Hospital, Belmont, MA, United States

ARTICLE INFO

Article history:
Received 24 April 2015
Received in revised form
3 June 2015
Accepted 4 August 2015
Available online 28 August 2015

Keywords:
Rumination
Attentional control
Depression
Anxiety
Mediation

ABSTRACT

Background: Deficits in attentional control have been hypothesized to cause rumination, suggesting that the relationships between attentional control and clinical symptoms may be mediated in part by rumination. However, to date, no clinical study has examined these constructs transdiagnostically in a path analysis model.

Methods: Fifty-one adults presenting for treatment completed measures of self-reported attentional control, rumination, and depression and anxiety symptoms. A bias-corrected path analysis-based approach was employed to test whether indirect (i.e., mediating) effects of rumination were significantly associated with the direct effects of attentional control on depression and anxiety symptoms. Separate models for depression and anxiety symptoms were tested along with reverse models using attentional control as a proposed mediator.

Results: The relationship between attentional control and clinical symptomatology (i.e., both depression and anxiety symptoms) was mediated by rumination. Poor attentional control was associated with more rumination and consequently more severe symptoms of depression and anxiety. The reverse relationship (i.e., attentional control mediating the relationship between rumination and depression or anxiety symptoms) was not significant.

Limitations: Study design did not allow testing of temporal precedence for the mediation models. All constructs were assessed via self-report.

Conclusions: Attentional control appears to impact depression and anxiety symptoms through rumination. The pathway between poor attentional control and emotion dysregulation via rumination suggests that interventions targeting attentional control may decrease maladaptive ruminative processes, leading to improved emotion regulation and reduced clinical symptomatology. Future studies should examine the stability of this mediational relationship over time (and in the face of targeted interventions).

1. Introduction

Rumination, broadly defined as repetitive thinking about self-relevant negative information or one's symptoms, has historically been associated with depression. However, more recent work has revealed that rumination is a transdiagnostic construct relevant across mood, anxiety, and psychotic disorders (Hartley et al., 2014; Just and Alloy, 1997; McLaughlin and Nolen-Hoeksema, 2011; Mellings and Alden, 2000; Muris et al., 2005; Roelofs et al., 2008; Spasojevic and Alloy, 2001; Surrence et al., 2009; Wolkenstein et al., 2014). It is important to identify mechanisms underlying rumination in order to more effectively target this maladaptive style of thinking in treatment. To this end, recent theoretical models (De Raedt and Koster, 2010; Koster et al., 2011) propose that deficits in attentional control may underlie rumination, and thus serve as important treatment targets themselves.

Attentional control refers to the ability to direct attention toward or away from stimuli depending on current goals or task demands. Attentional control affects a number of related cognitive processes, such as working memory and inhibition. Koster et al. (2011) proposed that the crucial cognitive vulnerability factor leading to excessive or persistent rumination is poor attentional control, and more specifically impaired ability to disengage attention from negative thoughts. This hypothesis contrasts with previous theories that characterized rumination primarily in terms

* Correspondence to: Behavioral Health Partial Program, McLean Hospital, Mail Stop 113, Belmont, MA 02478, United States.
E-mail address: khsu@mclean.harvard.edu (K.J. Hsu).

http://dx.doi.org/10.1016/j.jad.2015.08.008
0165-0327/

of increased attention and focus on one's symptoms and their implications (Nolen-Hoeksema, 2000; Nolen-Hoeksema and Davis, 1999; Nolen-Hoeksema and Morrow, 1991). Koster et al. (2011) suggest that individuals with good attentional control are able to disengage from negative thoughts, which allows for various emotion regulation strategies to be employed, ending the cycle of negative mood and rumination. Although Koster et al. (2011) focus exclusively on the relationship between rumination and depression, one could apply their model to explain relationships between rumination and anxiety (Mellings and Alden, 2000).

Empirical support for the theorized relationships between attentional control and rumination comes from a variety of tasks assumed to involve attentional control. For example, using a dot probe task, Donaldson et al. (2007) found that rumination was related to an attentional bias for negative words, even when controlling for depressive symptoms. Of note, several studies have shown that high trait ruminators perform more poorly than low ruminators on tasks requiring inhibition of non-valenced information (Daches et al., 2010; De Lissnyder et al., 2012; Whitmer and Banich, 2007), suggesting that the impairments in attentional control associated with rumination might not be valence-specific (however, see Tortella-Feliu et al., 2014). Self-reported attentional control has also been associated with rumination in an undergraduate sample (Fergus et al., 2012).

Although there is a wealth of data linking poor attentional control and rumination, and findings supporting their separate associations with psychopathology, there are very few studies examining these two constructs together as they relate to clinical symptoms. Furthermore, as noted in the review by Koster et al. (2011), most studies examining the relationship between rumination and attentional control have relied on undergraduate samples. To our knowledge, no study has employed mediational models to test the hypothesis that rumination mediates the relationship between attentional control and psychopathology in a clinical sample. Such data are needed as the field works to identify transdiagnostic mechanisms in order to develop more effective and targeted interventions for rumination and attentional control, as current interventions for attentional control (e.g., attention bias modification) and rumination (e.g., CBT) have room for more precision and increased efficacy in addressing these constructs.

The current study aimed to clarify the relationship between self-reported attentional control, rumination, and clinical symptomatology (e.g., anxiety and depression symptoms). Taking a transdiagnostic approach, we examined these constructs in a highly comorbid, heterogeneous, real-world patient population presenting for treatment at a partial hospital. We hypothesized that poor attentional control and higher levels of rumination would be associated with more severe depression and anxiety symptoms, and that rumination would mediate the relationship between poor attentional control and clinical symptomatology.

2. Materials and methods

2.1. Participants

Participants were patients receiving treatment at the Behavioral Health Partial Hospital Program at McLean Hospital. The partial hospital provides brief, intensive group, and individual evidenced-based psychotherapy (e.g., Cognitive Behavioral Therapy (CBT), Dialectical Behavior Therapy (DBT)) and pharmacological treatment to patients suffering from a wide range of psychiatric disorders (principally mood, anxiety, personality, and psychotic disorders; see Björgvinsson et al., 2014 for more detail regarding the treatment setting). Patients were either stepping down from an inpatient hospitalization or stepping up their level of care from the community. Patients were eligible for the study if they met criteria for a current depressive episode, as assessed by the Patient Health Questionnaire (PHQ-9), and were deemed stable enough to complete a research protocol (i.e., not actively psychotic). See Table 1 for demographic and clinical characteristics.

Table 1
Demographic and clinical characteristics ($n=51$).

Demographic characteristics	*N*	(%)
Female	33	(64.7%)
Male	18	(35.3%)
Age (M, SD)	32.78	(14.02)
Race		
White	40	(78.4%)
Multi-racial	6	(11.8%)
Did not specify	5	(9.8)
Ethnicity		
Non-Latino/a	48	(94.1%)
Latino/a	3	(5.9%)
Marital Status		
Single	32	(62.7%)
Married/Living with Partner	7	(13.7%)
Divorced/Separated/Widowed	12	(23.6%)
Highest Level of Education		
High School/GED	3	(5.9%)
Some college	23	(45.1%)
4-Year college graduate	12	(23.5%)
Post-college education	13	(25.5%)
Referral		
Stepping down from inpatient	14	(27.5%)
Stepping up from outpatient	37	(72.5%)
Co-morbid Anxiety Disorder	20	(43.5%)
Primary Diagnosis	*N*	(%)
MDD, recurrent, Severe w/o psychotic features	27	(52.9%)
MDD, recurrent, Severe with psychotic features	1	(2%)
Bipolar I Disorder, MRE depressed, Severe,without psychotic features	6	(11.8%)
Bipolar I Disorder, MRE mixed, Severe,without psychotic features	1	(2%)
Bipolar I Disorder, MRE mixed, Severe,with psychotic features	1	(2%)
Bipolar II Disorder	1	(2%)
Mood Disorder NOS	10	(19.6%)
Psychotic Disorder NOS	2	(3.9%)
Prolonged Posttraumatic stress disorder	1	(2%)

*Note.*ACS=Attentional Control Scale
RRS=Ruminative Responses Scale
CES-D-10=Center for the Epidemiological Studies of Depression-10
GAD-7=7-item Generalized Anxiety Disorder Scale.
MDD=Major Depressive Disorder
MRE=Most Recent Episode

2.2. Measures

2.2.1. Mini International Neuropsychiatric Interview (MINI; Sheehan et al., 1998)

The MINI is a structured interview assessing DSM-IV Axis I disorders (e.g., mood, anxiety, substance abuse, psychosis). Each MINI diagnostic module consists of a series of screening items followed by questions about specific symptomatology. The MINI has strong reliability and validity in relation to the Structured Clinical Interview for DSM-IV (SCID-IV), with inter-rater reliabilities ranging from kappas of .89–1.0 (Sheehan et al., 1998). For the partial hospital patients, inter-rater reliability between the MINI and program psychiatrists is .69 for MDD and .75 for Bipolar Disorder–Depressed (Kertz et al., 2012).

The MINI was administered by doctoral practicum students and interns in clinical psychology who received weekly supervision by a postdoctoral psychology fellow. Training included reviewing administration manuals and completing mock interviews. All

clinicians were required to pass a final training interview with their supervisor before administering MINIs for the program.

2.2.2. Ruminative Responses Scale (RRS; Treynor et al., 2003)

The RRS is a 22-item, self-report assessment of rumination with adequate psychometric properties (Treynor et al., 2003). Items are rated on a 4-point scale *(1=almost never, 2=sometimes, 3=often, 4=almost always)*, with higher scores indicating more rumination. In addition to a total score, Brooding and Self-Reflection subscales can be derived. However, we relied on total score given the overlap and reduced validity between subscales in clinical samples (Whitmer and Gotlib, 2011). The RRS had good internal consistency in this study (α=.85).

2.2.3. Attentional Control Scale (ACS; Derryberry and Reed, 2002)

The ACS is a 20-item self-report questionnaire that assesses an individual's perception of his/her ability to focus on relevant stimuli while ignoring distractors, as well as his/her ability to shift attention. Items are rated on a 4-point scale *(1=almost never, 2=sometimes, 3=often, 4=always)*. The total possible range of scores is from 20 to 80, where higher scores indicate greater attentional control. The ACS had good internal consistency in this study (α=.84).

2.2.4. Center for the Epidemiological Studies of Depression-10 (CESD-10; Andresen et al., 1994)

The CESD-10 is a widely used, brief instrument for measuring symptoms of depression. Items assess symptoms of depression and response anchors range from 0 (*rarely or none of the time/(less than 1 day)*) to 3 (*most or all of the time/(5-7 days)*). The total possible range of scores is from zero to 30, where higher scores indicate greater severity and/or duration of depressive symptoms. The CESD-10 had acceptable internal consistency in this study (α=.74).

2.2.5. The 7-item Generalized Anxiety Disorder Scale (GAD-7; Spitzer et al., 2006)

The GAD-7 is a self-report questionnaire that assesses general symptoms of anxiety (Spitzer et al., 2006). Although originally developed as a screening instrument for GAD, it is now widely used as a measure of global anxiety symptoms. Participants are asked how often in the past two weeks they have been bothered by anxiety symptoms (e.g., trouble relaxing). Participants respond according to a 4-point Likert type scale, from 0 (*not at all*), to 3 (*nearly every day*). The total possible range of scores is from zero to 21, where higher scores indicate greater severity and/or duration of anxiety symptoms. The GAD-7 has demonstrated good reliability and construct validity (Kroenke et al., 2007; Löwe et al., 2008; Spitzer et al., 2006) and is a valid measure of general anxiety in our partial hospital population (Beard and Björgvinsson, 2014). The GAD-7 had good internal consistency in this study (α=.86).

2.3. Procedures

The local Institutional Review Board approved all study procedures. Upon admission, patients were informed that they would complete daily, computerized questionnaires as part of standard clinical care, and they consented for their clinical data to be used for research purposes. The admission assessment included self-report measures (PHQ-9, CESD-10, GAD-7) collected and managed using REDCap (Research Electronic Data Capture) tools hosted at McLean Hospital. REDCap is a secure, web-based application designed to support data capture for research studies (Harris et al., 2009). Participants provided written consent to a separate study to examine cognitive control in depression, from which the RRS and ACS data were obtained. Participants completed the ACS, RRS, and a structured diagnostic interview in a separate assessment session upon admission to the program.

2.4. Data analyses

2.4.1. Regression analyses

In order to test the association between attentional control, rumination, and clinical symptoms (i.e., depression and anxiety, as measured by the CES-D and GAD-7, respectively), we utilized a hierarchical linear regression framework. We regressed outcomes (depression symptoms, anxiety symptoms) onto age and sex as the first step, followed by attentional control for the second step, with addition of rumination as the third and final step. Models were compared for statistically significant changes in explained variance (R^2) by computing a partial *F*-statistic. Linear regression models were conducted in SAS (SAS version 9.2, SAS Institute, Cary, NC).

2.4.2. Mediation analyses

Examination of possible mediators was done through an ordinary least squares (OLS) path analysis-based approach to examine direct and indirect effects of our proposed mediator (rumination) in our models (conducted through PROCESS, a computational and analytic modeling tool in SAS; Hayes, 2012). The PROCESS tool allows for explicit quantification of the indirect effect of a mediator on an outcome and tests the significance of this effect through asymmetric bootstrap confidence intervals (Hayes, 2009). Mediation analyses employed a 95% bias-corrected bootstrap confidence interval to test whether indirect effects were significantly associated with the outcome. Bootstrap confidence intervals for indirect effects entirely above or below zero are considered significant. We tested separate mediation models for CES-D and GAD-7 scores and examined a reverse mediation model, with attentional control as the proposed mediator. All listed *p*-values are two-tailed.

3. Results

3.1. Participant characteristics

Characteristics of the study sample are summarized in Table 1. Medication data were available from medical charts for 46 participants, and most of these individuals (n=42) were receiving pharmacological treatment upon admission to the partial hospital (antidepressant: n=34, antianxiety: n=18, mood stabilizer: n=16, antipsychotic: n=16, range=0–7 medications; M=2.70, S.D.=1.50). Correlations among questionnaires and associated subscales are presented in Table 2. Bivariate correlations indicated that depression symptoms were associated with attentional control and rumination, while anxiety symptoms were primarily associated with rumination. Attentional control and rumination

Table 2
Measure descriptive statistics and correlations.

Scale	1	2	3	4	5	*Mean*	*SD*	α
1. Age	–					32.78	14.02	–
2. CESD-10	.18	–				20.43	5.01	.738
3. GAD-7	.02	.49***	–			12.71	5.19	.857
4. RRS	−.00	.32*	.35*	–		62.88	9.52	.852
5. ACS	.09	−.30*	−.14	−.28*	–	41.49	8.86	.836

Note **p < .01. CESD-10=Center for Epidemiologic Studies of Depression – 10; PHQ-9=Patient Health Questionnaire – 9; GAD-7=the 7-item Generalized Anxiety Disorder Scale; RRS=Ruminative Responses Scale; ACS=Attentional Control Scale.

* p < .05

*** p < 0.001.

Table 3
Hierarchical linear regression analyses.

Outcome	Parameter estimate (SE)	R^2	ΔR^2	p-Value
CESD-10 (Step 1)		0.036	0.036	0.453
Age	0.06 (0.05)			0.239
Sex	0.67 (1.48)			0.652
CESD-10 (Step 2)		0.416	0.298	0.001
Age	0.07 (0.05)			0.164
Sex	1.13 (1.43)			0.433
Attentional control	−0.19 (0.08)			0.021
CESD-10 (Step 3)		0.479	0.063	0.130
Age	0.06 (0.05)			0.181
Sex	1.72 (1.41)			0.229
Attentional control	−0.14 (0.08)			0.087
Rumination	0.15 (0.08)			0.052
GAD-7 (Step 1)		0.001	0.001	0.998
Age	0.01 (0.05)			0.899
Sex	−0.17 (1.56)			0.914
GAD-7 (Step 2)		0.078	0.041	0.461
Age	0.01 (0.05)			0.844
Sex	0.04 (1.58)			0.980
Attentional control	−0.09 (0.09)			0.330
GAD-7 (Step 3)		0.224	0.146	0.018
Age	0.00 (0.05)			0.928
Sex	0.81 (1.54)			0.600
Attentional control	−0.02 (0.09)			0.788
Rumination	0.20 (0.08)			0.022

Note. Sex is coded as a dichotomous variable (0=female, 1=male). *N=51* CESD-10=Center for Epidemiologic Studies of Depression – 10; GAD-7=the 7-item Generalized Anxiety Disorder Scale; RRS=Ruminative Responses Scale; ACS=Attentional Control Scale.

were negatively correlated. While there was no significant relationship between attentional control and anxiety, contemporary methods of mediation model testing do not require a significant direct effect (Preacher and Hayes, 2008; i.e., between ACS and GAD-7 scores); thus we continued with the planned mediational analyses.

3.2. Association between attentional control and clinical symptoms

Results for the hierarchical linear regression analyses are presented in Table 3. Demographics were not associated with a significant increase in explained variance when added to regression models. The addition of the attentional control scale was associated with a significant increase in R^2 when predicting CESD-10 score ($F(1, 48)=5.48$, $p=0.023$), but not GAD-7 score ($F(1, 48)<1$, $p=0.330$). The significant increase in R^2 was reflected in the depression regression model, with better attentional control predicting lower depression symptoms even after controlling for age and sex. Addition of rumination resulted in a large increase in explained variance for the GAD-7 (F (1, 47=5.53, $p=0.023$), but only a marginal increase for the CESD-10 (F (1, 47)=2.40, $p=$ 0.072). The attentional control scale was only a moderate predictor of depression symptoms after including rumination in the model, as higher levels of rumination predicted more depression symptoms. Rumination was also a significant predictor of increased anxiety.

3.3. Mediation of the relationship between attentional control and depression/anxiety symptoms by rumination

Table 4 provides estimates of the total effect, direct effect, and indirect effect for attentional control on depression and anxiety symptoms through rumination.

For depression, rumination showed a significant indirect effect on the relation between attentional control (i.e., the ACS) and the CESD-10 (indirect effect 95% bootstrap CI: −0.148 to −0.003), indicating that rumination mediated the relationship between attentional control and depression symptoms. Rumination also showed a significant indirect effect on the relation between attentional control and anxiety symptoms (i.e., the GAD-7; indirect effect 95% bootstrap CI: −0.190 to −0.007), again indicating mediation by rumination. Both mediation models reveal that poor attentional control is associated with greater rumination, which in turn is associated with more severe depression and anxiety symptoms.

Table 4
Mediation analyses (Rumination versus Attentional Control).

	Total effect		Direct effect		Indirect effect (mediation)		
	Effect	SE	Effect	SE	Effect	SE	95% CI
Mediation model 1 (Rumination (RRS)=mediator; Attentional Control (ACS)=IV):							
Depression severity							
CESD-10	−0.18	0.08	−0.13	0.08	−0.05	0.04	(−0.148 to −0.003)
Anxiety symptoms							
GAD-7	−0.09	0.09	−0.02	0.09	−0.07	0.04	(−0.190 to −0.007)
Mediation model 2 (Attentional Control (ACS)=mediator; Rumination (RRS)=IV):							
Depression severity							
CESD-10	0.17	0.07	0.14	0.08	0.04	0.02	(−0.001 to 0.098)
Anxiety symptoms							
GAD-7	0.19	0.07	0.19	0.08	0.01	0.03	(−0.047 to 0.085)

Note: 95% Bias-corrected Bootstrap Confidence Intervals are based on a 5000-bootstrap sample. CESD-10=Center for Epidemiologic Studies of Depression – 10; GAD-7=the 7-item Generalized Anxiety Disorder Scale; RRS=Ruminative Responses Scale; ACS=Attentional Control Scale.

To test the directional specificity of these effects, we investigated whether or not attentional control mediated the relationship between rumination and clinical symptomatology. Table 4 also provides estimates for these total, direct, and indirect effects for rumination on depression and anxiety symptoms through attentional control. Attentional control did not show a significant indirect effect on the relationship between rumination and the CESD-10 (indirect effect 95% bootstrap CI: −0.001 to 0.098), or GAD-7 (indirect effect 95% bootstrap CI: −0.047 to 0.085). These findings support the role of rumination as a mediator in the association between attentional control and clinical symptomatology.

4. Discussion

Attentional control and rumination have received substantial theoretical and empirical interest as transdiagnostic mechanisms underlying psychopathology. While attentional dysfunction and rumination separately appear to serve as transdiagnostic mechanisms in both depression and anxiety, few studies have examined theorized relationships between attentional dysfunction and rumination across depression and anxiety symptoms in a patient population. This study tested the extent to which rumination mediated the association between attentional control and clinical symptoms in a clinical sample.

We hypothesized that poor attentional control and high rumination would be associated with more depression and anxiety symptoms. These hypotheses were partially supported. While rumination was significantly associated with depression and anxiety symptoms (even after accounting for demographics and

attentional control), attentional control was only associated with depression symptoms and no longer was a significant predictor after accounting for covariates. While power is often raised as a potential factor in these patterns of findings, it is worth noting that with 51 study participants, our study was sufficiently powered (i.e., 80% power) to detect the medium effect sizes previously found in the literature (f^2=.16; c.f., Moriya and Tanno, 2008; Ólafsson et al., 2011; Quigley, 2012). Future research ought to examine how the processes leading from rumination to depression symptoms versus anxiety symptoms compare and contrast, to further delineate the etiology of clinical symptomatology and highlight additional targets for intervention. For example, rumination may lead to increased anxiety symptoms through content related to perceived social failures, while ruminative content related to self-efficacy may lead to increased depression symptoms.

We also hypothesized that poor attentional control would be associated with greater rumination, which in turn would be associated with more symptoms of depression and anxiety. These hypothesized relationships were supported by our results, as rumination mediated the relationship between attentional control and both depression and anxiety symptoms. Although these effects were small, this pathway between poor attentional control and emotion dysregulation via rumination suggests that interventions targeting attentional control may decrease maladaptive ruminative processes, leading to improved emotion regulation and thus reduced clinical symptomatology. While there have been studies indicating that healthy and anxious individuals receiving such interventions do show decreased attentional biases (Beard et al., 2012) and better emotion regulation (Bomyea and Amir, 2011; Gyurak et al., 2010), reduction of ruminative processes has not been explored as a potential mediating pathway. Additionally, studies utilizing attentional control training with non-valenced stimuli do find reductions in depression symptomatology and rumination (Papageorgiou and Wells, 2000; Siegle et al., 2014, 2007), which are maintained even at 12-month follow-up (Papageorgiou and Wells, 2000). The findings from the current study suggest treatments targeting attentional control and rumination are worth further investigation.

Our findings provide support for existing theories which posit that attentional control deficits lead to stronger ruminative processes in clinical populations (De Raedt and Koster, 2010; Koster et al., 2011), resulting in greater severity of clinical symptoms. As most studies examining the overlap between attentional control and rumination have used subclinical and/or undergraduate samples, the use of a clinical sample and mediational models in this study represents an important extension of prior research. Although this study adds to the burgeoning literature on the relationship between attentional control, rumination, and psychopathology, a number of limitations merit consideration and suggest directions for future research. First, our study was cross-sectional in nature. While utilizing bias-corrected path analysis mediational models allowed us to test for statistical mediation between constructs, multiple assessment points are needed to be able to verify the time course and mechanistic pathways among attentional control, rumination, and clinical symptomatology. However, the precise timing of assessments necessary to appropriately capture these mechanistic processes is unclear. While some aspects of impaired attention appear to be stable over long periods of time, other aspects of attention dysfunction and rumination show variation on the order of weeks (c.f., Gruber et al., 2007; Hammar and Årdal, 2012; Hankin, 2008) or seconds (e.g., Zvielli et al., 2014). Consequently, depending on the interval between assessments, concurrent mediation models may hold more value than longitudinal mediation models due to the temporal dynamics of the phenomena being studied. That being said, prospective studies in at-risk individuals are still a valuable research direction and particularly needed, given that these constructs may have bi-directional relationships once a depressive or anxiety disorder has onset. Given the severity and comorbidity of our sample, it is noteworthy that the reverse mediational model was not significant. Second, we relied on a self-report measure of attentional control. Future studies that include both self-report and behavioral measures of attentional control would strengthen conclusions about their relationships. Third, we lacked a control group. Utilization of a healthy control sample in future research would allow us to consider possible relationships among ruminative processes, attentional control, and mood and anxiety symptoms in healthy individuals. While research has made consistent connections between attentional control and clinical symptomatology in non-clinical samples (Barry et al., 2013; Moriya and Tanno, 2008; Ólafsson et al., 2011; Quigley, 2012; Reinholdt-Dunne et al., 2009), rumination has not been tested as a potential mediator of that relationship. Relatedly, the lack of control group and high proportion of participants utilizing medication made it impossible to discern the impact of medication on the relationships between attentional control, rumination, and clinical symptomatology. Antidepressant medication has been found to impact indices of attentional control and cognitive processing of emotional information (e.g., Browning et al., 2010; Wagner et al., 2012; though see Douglas et al., 2011; Paradiso et al., 1997), suggesting these relationships may change in the face of existing, targeted interventions. Furthermore, inclusion of a control group and multiple assessment points would allow us to examine the impact of treatment response (and in particular, mindfulness-based interventions like DBT) on attentional control. As mindfulness-based interventions have been found to improve attentional control (e.g., Chambers et al., 2008; Malinowski, 2013; Moore et al., 2012), reduced ruminative processes may be one possible mechanism underlying treatment efficacy that deserves further attention.

In spite of these limitations, the current findings support recently proposed models of rumination (De Raedt and Koster, 2010; Koster et al., 2011) and have transdiagnostic clinical implications regarding the potential utility of interventions targeting attentional control in reducing rumination and clinical symptomatology. Future work using prospective and experimental designs, as well as behavioral and neural markers of attentional control, will further elucidate these mechanistic relationships.

References

Andresen, E.M., Malmgren, J.A., Carter, W.B., Patrick, D.L., 1994. Screening for depression in well older adults: evaluation of a short form of the CES-D. Am. J. Prev. Med. 10, 77–84.

Barry, T.J., Hermans, D., Lenaert, B., Debeer, E., Griffith, J.W., 2013. The eACS: attentional control in the presence of emotion. Personal. Individ. Differ. 55, 777–782. http://dx.doi.org/10.1016/j.paid.2013.06.014.

Beard, C., Björgvinsson, T., 2014. Beyond generalized anxiety disorder: psychometric properties of the GAD-7 in a heterogeneous psychiatric sample. J. Anxiety Disord. 28, 547–552. http://dx.doi.org/10.1016/j.janxdis.2014.06.002.

Beard, C., Sawyer, A.T., Hofmann, S.G., 2012. Efficacy of attention bias modification using threat and appetitive stimuli: a meta-analytic review. Behav. Ther. 43, 724–740.

Björgvinsson, T., Kertz, S.J., Bigda-Peyton, J.S., Rosmarin, D.H., Aderka, I.M., Neuhaus, E.C., 2014. Effectiveness of cognitive behavior therapy for severe mood disorders in an acute psychiatric naturalistic setting: a benchmarking study. Cogn. Behav. Ther. 43, 209–220. http://dx.doi.org/10.1080/16506073.2014.901988.

Bomyea, J., Amir, N., 2011. The effect of an executive functioning training program on working memory capacity and intrusive thoughts. Cogn. Ther. Res. 35, 529–535. http://dx.doi.org/10.1007/s10608-011-9369-8.

Browning, M., Holmes, E.A., Harmer, C.J., 2010. The modification of attentional bias to emotional information: a review of the techniques, mechanisms, and relevance to emotional disorders. Cogn. Affect. Behav. Neurosci. 10, 8–20. http://dx.doi.org/10.3758/CABN.10.1.8.

Chambers, R., Lo, B.C.Y., Allen, N.B., 2008. The impact of intensive mindfulness training on attentional control, cognitive style, and affect. Cogn. Ther. Res. 32,

303–322. http://dx.doi.org/10.1007/s10608-007-9119-0.
Daches, S., Mor, N., Winquist, J., Gilboa-Schechtman, E., 2010. Brooding and attentional control in processing self-encoded information: evidence from a modified Garner task. Cogn. Emot. 24, 876–885. http://dx.doi.org/10.1080/02699930902954825.
De Lissnyder, E., Koster, E.H.W., Everaert, J., Schacht, R., Van den Abeele, D., De Raedt, R., 2012. Internal cognitive control in clinical depression: general but no emotion-specific impairments. Psychiatry Res. 199, 124–130. http://dx.doi.org/10.1016/j.psychres.2012.04.019.
De Raedt, R., Koster, E.H.W., 2010. Understanding vulnerability for depression from a cognitive neuroscience perspective: a reappraisal of attentional factors and a new conceptual framework. Cogn. Affect. Behav. Neurosci. 10, 50–70. http://dx.doi.org/10.3758/CABN.10.1.50.
Derryberry, D., Reed, M.A., 2002. Anxiety-related attentional biases and their regulation by attentional control. J. Abnorm. Psychol. 111, 225–236. http://dx.doi.org/10.1037//0021-843X.111.2.225.
Donaldson, C., Lam, D., Mathews, A., 2007. Rumination and attention in major depression. Behav. Res. Ther. 45, 2664–2678. http://dx.doi.org/10.1016/j.brat.2007.07.002.
Douglas, K.M., Porter, R.J., Knight, R.G., Maruff, P., 2011. Neuropsychological changes and treatment response in severe depression. Br. J. Psychiatry 198, 115–122. http://dx.doi.org/10.1192/bjp.bp.110.080713.
Fergus, T.A., Bardeen, J.R., Orcutt, H.K., 2012. Attentional control moderates the relationship between activation of the cognitive attentional syndrome and symptoms of psychopathology. Personal. Individ. Differ. 53, 213–217. http://dx.doi.org/10.1016/j.paid.2012.03.017.
Gruber, S., Rathgeber, K., Bräunig, P., Gauggel, S., 2007. Stability and course of neuropsychological deficits in manic and depressed bipolar patients compared to patients with major depression. J. Affect. Disord. 104, 61–71. http://dx.doi.org/10.1016/j.jad.2007.02.011.
Gyurak, A., Ayduk, O., Gross, J.J., 2010. Training Executive Functions: Emotion Regulation and Affective Consequences.
Hammar, Å., Årdal, G., 2012. Effortful information processing in patients with major depression—a 10-year follow-up study. Psychiatry Res. 198, 420–423. http://dx.doi.org/10.1016/j.psychres.2011.11.020.
Hankin, B.L., 2008. Stability of cognitive vulnerabilities to depression: a short-term prospective multiwave study. J. Abnorm. Psychol. 117, 324–333. http://dx.doi.org/10.1037/0021-843X.117.2.324.
Harris, P.A., Taylor, R., Thielke, R., Payne, J., Gonzalez, N., Conde, J.G., 2009. Research electronic data capture (REDCap)—a metadata-driven methodology and workflow process for providing translational research informatics support. J. Biomed. Inform. 42, 377–381. http://dx.doi.org/10.1016/j.jbi.2008.08.010.
Hartley, S., Haddock, G., Vasconcelos e Sa, D., Emsley, R., Barrowclough, C., 2014. An experience sampling study of worry and rumination in psychosis. Psychol. Med. 44, 1605–1614. http://dx.doi.org/10.1017/S0033291713002080.
Hayes, A.F., 2012. PROCESS: A Versatile Computational Tool for Observed Variable Mediation, Moderation, and Conditional Process Modeling.
Hayes, A.F., 2009. Beyond baron and kenny: statistical mediation analysis in the new millennium. Commun. Monogr. 76, 408–420. http://dx.doi.org/10.1080/03637750903310360.
Just, N., Alloy, L.B., 1997. The response styles theory of depression: tests and an extension of the theory. J. Abnorm. Psychol. 106, 221.
Kertz, S.J., Bigda-Peyton, J.S., Rosmarin, D.H., Björgvinsson, T., 2012. The importance of worry across diagnostic presentations: prevalence, severity and associated symptoms in a partial hospital setting. J. Anxiety Disord. 26, 126–133. http://dx.doi.org/10.1016/j.janxdis.2011.10.005.
Koster, E.H.W., De Lissnyder, E., Derakshan, N., De Raedt, R., 2011. Understanding depressive rumination from a cognitive science perspective: the impaired disengagement hypothesis. Clin. Psychol. Rev. 31, 138–145. http://dx.doi.org/10.1016/j.cpr.2010.08.005.
Kroenke, K., Spitzer, R.L., Williams, J.B., Monahan, P.O., Löwe, B., 2007. Anxiety disorders in primary care: prevalence, impairment, comorbidity, and detection. Ann. Intern. Med. 146, 317–325.
Löwe, B., Decker, O., Müller, S., Brähler, E., Schellberg, D., Herzog, W., Herzberg, P.Y., 2008. Validation and standardization of the Generalized Anxiety Disorder Screener (GAD-7) in the general population. Med. Care 46, 266–274.
Malinowski, P., 2013. Neural mechanisms of attentional control in mindfulness meditation. Front. Neurosci. 7. http://dx.doi.org/10.3389/fnins.2013.00008.
McLaughlin, K.A., Nolen-Hoeksema, S., 2011. Rumination as a transdiagnostic factor in depression and anxiety. Behav. Res. Ther. 49, 186–193. http://dx.doi.org/10.1016/j.brat.2010.12.006.
Mellings, T., Alden, L.E., 2000. Cognitive processes in social anxiety: The effects of self-focus, rumination and anticipatory processing. Behav. Res. Ther. 38, 243–257.
Moore, A., Gruber, T., Derose, J., Malinowski, P., 2012. Regular, brief mindfulness meditation practice improves electrophysiological markers of attentional control. Front. Hum. Neurosci 6. http://dx.doi.org/10.3389/fnhum.2012.00018.
Moriya, J., Tanno, Y., 2008. Relationships between negative emotionality and attentional control in effortful control. Personal. Individ. Differ. 44, 1348–1355. http://dx.doi.org/10.1016/j.paid.2007.12.003.
Muris, P., Roelofs, J., Rassin, E., Franken, I., Mayer, B., 2005. Mediating effects of rumination and worry on the links between neuroticism, anxiety and depression. Personal. Individ. Differ. 39, 1105–1111. http://dx.doi.org/10.1016/j.paid.2005.04.005.
Nolen-Hoeksema, S., 2000. The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. J. Abnorm. Psychol. 109, 504–511. http://dx.doi.org/10.1037/0021-843X.109.3.504.
Nolen-Hoeksema, S., Davis, C.G., 1999. "Thanks for sharing that": ruminators and their social support networks. J. Personal. Soc. Psychol. 77, 801–814.
Nolen-Hoeksema, S., Morrow, J., 1991. A prospective study of depression and posttraumatic stress symptoms after a natural disaster: the 1989 Loma Prieta earthquake. J. Personal. Soc. Psychol. 61, 115–121.
Ólafsson, R.P., Smári, J., Guðmundsdóttir, F., Ólafsdóttir, G., Harðardóttir, H.L., Einarsson, S.M., 2011. Self reported attentional control with the attentional control scale: factor structure and relationship with symptoms of anxiety and depression. J. Anxiety Disord. 25, 777–782. http://dx.doi.org/10.1016/j.janxdis.2011.03.013.
Papageorgiou, C., Wells, A., 2000. Treatment of recurrent major depression with attention training. Cogn. Behav. Pract. 7, 407–413.
Paradiso, S., Lamberty, G.J., Garvey, M.J., Robinson, R.G., 1997. Cognitive impairment in the euthymic phase of chronic unipolar depression. J. Nerv. Ment. Dis. 185, 748–754.
Preacher, K.J., Hayes, A.F., 2008. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. Behav. Res. Methods 40, 879–891. http://dx.doi.org/10.3758/BRM.40.3.879.
Quigley, L., 2012. Cognitive Inhibition and its Relation to Emotion Regulation in Dysphoria (Master's Thesis). University of Calgary, Calgary, Alberta.
Reinholdt-Dunne, M.L., Mogg, K., Bradley, B.P., 2009. Effects of anxiety and attention control on processing pictorial and linguistic emotional information. Behav. Res. Ther. 47, 410–417. http://dx.doi.org/10.1016/j.brat.2009.01.012.
Roelofs, J., Huibers, M., Peeters, F., Arntz, A., van Os, J., 2008. Rumination and worrying as possible mediators in the relation between neuroticism and symptoms of depression and anxiety in clinically depressed individuals. Behav. Res. Ther. 46, 1283–1289. http://dx.doi.org/10.1016/j.brat.2008.10.002.
Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., Hergueta, T., Baker, R., Dunbar, G.C., 1998. The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. J. Clin. Psychiatry 59, 22–33.
Siegle, G.J., Ghinassi, F., Thase, M.E., 2007. Neurobehavioral therapies in the 21st century: summary of an emerging field and an extended example of cognitive control training for depression. Cogn. Ther. Res. 31, 235–262. http://dx.doi.org/10.1007/s10608-006-9118-6.
Siegle, G.J., Price, R.B., Jones, N.P., Ghinassi, F., Painter, T., Thase, M.E., 2014. You gotta work at it: pupillary indices of task focus are prognostic for response to a neurocognitive intervention for rumination in depression. Clin. Psychol. Sci. 2, 455–471. http://dx.doi.org/10.1177/2167702614536160.
Spasojevic, J., Alloy, L.B., 2001. Rumination as a common mechanism relating depressive risk factors to depression. Emotion 1, 25–37. http://dx.doi.org/10.1037//1528-3542.1.1.25.
Spitzer, R.L., Kroenke, K., Williams, J.B., Löwe, B., 2006. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch. Intern. Med. 166, 1092–1097.
Surrence, K., Miranda, R., Marroquín, B.M., Chan, S., 2009. Brooding and reflective rumination among suicide attempters: cognitive vulnerability to suicidal ideation. Behav. Res. Ther. 47, 803–808. http://dx.doi.org/10.1016/j.brat.2009.06.001.
Tortella-Feliu, M., Morillas-Romero, A., Balle, M., Bornas, X., Llabrés, J., Pacheco-Unguetti, A.P., 2014. Attentional control, attentional network functioning, and emotion regulation styles. Cogn. Emot. 28, 769–780. http://dx.doi.org/10.1080/02699931.2013.860889.
Treynor, W., Gonzalez, R., Nolen-Hoeksema, S., 2003. Rumination reconsidered: a psychometric analysis. Cogn. Ther. Res. 27, 247–259.
Wagner, S., Doering, B., Helmreich, I., Lieb, K., Tadić, A., 2012. A meta-analysis of executive dysfunctions in unipolar major depressive disorder without psychotic symptoms and their changes during antidepressant treatment: executive dysfunctions in unipolar MDD. Acta Psychiatr. Scand. 125, 281–292. http://dx.doi.org/10.1111/j.1600-0447.2011.01762.x.
Whitmer, A., Gotlib, I.H., 2011. Brooding and reflection reconsidered: a factor analytic examination of rumination in currently depressed, formerly depressed, and never depressed individuals. Cogn. Ther. Res. 35, 99–107. http://dx.doi.org/10.1007/s10608-011-9361-3.
Whitmer, A.J., Banich, M.T., 2007. Inhibition versus switching deficits in different forms of rumination. Psychol. Sci. 18, 546–553.
Wolkenstein, L., Zwick, J.C., Hautzinger, M., Joormann, J., 2014. Cognitive emotion regulation in euthymic bipolar disorder. J. Affect. Disord. 160, 92–97. http://dx.doi.org/10.1016/j.jad.2013.12.022.
Zvielli, A., Bernstein, A., Koster, E.H., 2014. Temporal dynamics of attentional bias. Clin. Psychol. Sci 2167702614551572.