Psychological Medicine (2009), **39**, 1226–1228.

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Psychological Medicine, **39** (2009).
doi:10.1017/S0033291709005704

Research Letter

Emotional reactivity to induced rumination predicts 1-year levels of depressive symptoms

Ruminative coping in response to negative mood has been proposed as a vulnerability factor for depression. Habitual rumination negatively affects the course of dysphoric or depressive episodes, and an experimentally induced ruminative self-focus reliably impairs short-term emotional and cognitive processing in dysphoric individuals (e.g. Joorman & Siemer, 2004; Rimes & Watkins, 2005; Donaldson *et al*. 2007; Kuehner *et al*. 2007, 2009; Nolen-Hoeksema *et al*. 2008). However, the predictive value of individual reactivity to induced rumination, i.e. to an induced state of dysfunctional cognitive processing, has so far not been studied, although high reactivity towards a rumination priming procedure in dysphoric subjects could indicate a vulnerability factor for depression.

In a recent experimental investigation (Kuehner *et al*. 2009), we randomized a non-clinical sample of 60 young adults after negative mood induction into a rumination, distraction, and mindful self-focus induction condition. In this study, induced rumination prolonged dysphoric mood and was linked to an increase in dysfunctional attitudes. In a subsample of participants scoring high on the Beck Depression Inventory (BDI-II), induced rumination also affected the cortisol stress response during the experimental session.

The aim of the present study was to expand previous research by exploring the role of individual emotional reactivity to induced rumination for the natural longitudinal course of depressive symptoms. For this purpose, we conducted a 1-year follow-up of the Kuehner *et al*. (2009) cohort. Our hypothesis was that higher individual levels of emotional reactivity to an induced ruminative self-focus at baseline would predict deterioration of depressive symptoms over time. In contrast, we did not expect respective longitudinal associations for the remaining induction conditions (distraction, mindful self-focus). Accordingly, the proposed effect difference across conditions should be demonstrated by a significant statistical interaction of emotional reactivity by induction group in the prediction of follow-up symptoms.

Follow-up participants were 56 undergraduate students (93.5% of the original cohort) from different faculties at the University of Mannheim, Germany. The follow-up examination took place after the experimental data of our study had been submitted. Full description of the experimental study is given by Kuehner *et al*. (2009). Briefly, participants were subjected to a negative mood induction, consisting of sad music and recall of negative life events for 6 min, and were subsequently randomly assigned to a rumination, distraction, or mindful self-focus induction for 8 min, with respective statements presented on cards (adapted paradigm from Lyubomirsky *et al*. 2003).

Depressive symptoms at baseline (before negative mood induction) and after 12 months were assessed with the BDI-II. The Positive and Negative Affect Schedule (PANAS) was employed during the experimental session to assess mood change as a consequence of negative mood induction (T1–T2) and of response induction (rumination, distraction, mindful self focus, T2–T3). For the present analysis, we chose the PANAS negative affect (NA) difference score from T2 to T3 (PANAS-NA-T2T3) to assess individual emotional reactivity to response induction. The NA subscale of the PANAS was preferred over the positive affect (PA) subscale due to its broader empirical evaluation within the context of self-focused attention paradigms (e.g. Mor & Winquist, 2002)†.

We performed a multiple regression analysis with BDI-II scores at follow-up as the dependent variable, and BDI-II scores at baseline, group status (rumination *versus* other conditions‡), PANAS-NA-T1T2, PANAS-NA-T2T3, and interaction of group status by PANAS-NA-T2T3 as independent variables. In addition, partial correlations between PANAS-NA-T2T3 and BDI-II scores at follow-up, controlled for BDI-II scores at baseline, were calculated separately for the rumination and the remaining condition.

The follow-up sample consisted of 29 women and 27 men aged 19–28 years (mean $\pm$ S.D. $= 22.1 \pm 2.6$). Baseline BDI-II scores did not differ between response

† Importantly, we did not expect predictive effects of individual reactivity to the negative mood induction itself, since mood priming studies have shown that vulnerable and non-vulnerable individuals experience similar mood deterioration (cf. Scher *et al*. 2005). However, following the suggestion of an anonymous reviewer, we included this variable as a covariate in the regression analysis shown in Table 1.

‡ Preliminary analyses identified no differential effects of individual reactivity on the 1-year course of depressive symptoms between the distraction and mindful self-focus conditions [interaction group (distraction, mindfulness) $\times$ PANAS-NA-T2T3: $B = -0.258$, S.E. $= 0.452$, $t = -0.571$, N.S.], therefore we collapsed the longitudinal data across these groups.

Table 1. *Predictive effect of emotional reactivity to response induction on depressive symptoms at 1-year follow-up*

	BDI-II scores at 1-year follow-up			
Predictors	*B*	S.E.	*t*	*p*
Constant	2.212	1.296	1.707	0.094
BDI-II score at baseline	0.607	0.105	5.776	0.001
PANAS-NA-T1T2	0.083	0.260	0.320	0.751
PANAS-NA-T2T3	−0.144	0.257	−0.0561	0.577
Group: rumination (1), other (0)	0.876	1.779	0.492	0.625
Interaction PANAS-NA-T2T3 × group	1.031	0.460	2.242	0.029

BDI-II, Beck Depression Inventory, 2nd revision; PANAS-NA-T1T2, negative affect difference score (Positive and Negative Affect Schedule, PANAS) from pre- to post-negative mood induction; PANAS-NA-T2T3, negative affect difference score (PANAS) from pre- to post-response induction.

induction groups [rumination ($n=20$): mean ± S.D. = 7.60 ± 5.81; other ($n=36$): mean ± S.D. = 7.53 ± 7.34, $t(55)=-0.038$, N.S.]. Participants' BDI-II scores at follow-up (mean ± S.D. = 7.34 ± 6.79) were similar to those at baseline [mean ± S.D. = 7.55 ± 6.78; $t(55)=-0.287$, N.S.], and relative stability over 12 months amounted to $r=0.662$ ($p<0.001$).

Table 1 shows the results from the multiple regression analysis. BDI-II scores at 1-year follow-up were significantly predicted by BDI-II scores at baseline and by the interaction term of group status by PANAS-NA-T2T3. The significant interaction term indicates a different closeness of relationship between PANAS-NA-T2T3 at baseline and BDI-II outcome at 12 months across groups. Separate analyses showed a significant effect of PANAS-NA-T2T3 on BDI-II scores at follow-up in the rumination group [$B=0.722$, S.E. = 0.332, $t(19)=2.177$, $p=0.044$], while this effect was not significant in the comparison group [$B=-0.232$, S.E. = 0.197, $t(35)=-1.177$, N.S.]. Partial correlations between PANAS-NA-T2T3 and BDI-II scores at follow-up amounted to $r=0.467$ ($p=0.044$) in the rumination sample, and to $r=-0.201$ (N.S.) in the comparison sample. The positive sign of this coefficient in the rumination group indicates that a stronger increase in negative affect during induced rumination at baseline was linked to higher BDI-II scores at 1-year follow-up.

To the best of our knowledge, this is the first study to assess predictive effects of emotional reactivity to induced rumination for the longitudinal course of depressive symptoms. Our hypothesis of a specific detrimental effect of heightened reactivity towards rumination was confirmed. The finding that participants who displayed high emotional reactivity to the rumination induction showed higher levels of depressive symptoms over time gives a first hint that individual reactivity towards induced dysfunctional information processing may indicate a vulnerability factor for depression. Within this context, it is important to note that the observed association between emotional reactivity and symptom outcome at 1 year was not due to potential confound with baseline depression levels, since the latter were statistically controlled. While baseline depressive symptoms were highly predictive of future depressive symptoms, emotional reactivity to induced rumination contributed independently to the prediction of outcome.

Previous experimental research on cognitive vulnerability in depression has mainly focused on cognitive reactivity to negative mood priming procedures. Somewhat surprisingly, most of these studies have used remitted depression designs (cf. Lau *et al*. 2004; Scher *et al*. 2005), which have been criticized due to their post-morbid approach and backward logic (Just *et al*. 2001). In contrast, research connecting experimentally identified vulnerability – operationalized as an individual difference variable – with a longitudinal design to explore its predictive value in non-clinical samples is clearly lacking. Our study is a first attempt to do so, and we were able to show that high emotional reactivity towards an induced ruminative self-focus during negative mood predicted an impaired long-term course of psychological wellbeing. More longitudinal research is now needed using larger, more heterogeneous samples and variations of experimental procedures to explore predictive effects of individual reactivity to induced dysfunctional information processing in a broader context, to assess its temporal stability, and to explore its role for predicting the onset of clinical episodes of depression.

Acknowledgements

This work was supported by research grant DFG KU1464/1-4 to C.K.

Declaration of Interest

None.

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https://doi.org/10.1017/S0033291709005704 Published online by Cambridge University Press