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BRIEF ARTICLE



## Cognitive reactivity as outcome and working mechanism of mindfulness-based cognitive therapy for recurrently depressed patients in remission

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### ABSTRACT

Major depressive disorder is a prevalent condition with high relapse rates. There is evidence that cognitive reactivity is an important vulnerability factor for the recurrence of depression. Mindfulness-based interventions are designed to reduce relapse rates, with cognitive reactivity as one of the proposed working mechanisms. In a randomised controlled trial we compared the effect of mindfulness-based cognitive therapy (MBCT) with treatment-as-usual (TAU) on cognitive reactivity in recurrently depressed patients ( $N = 115$ ). Depressive symptoms, cognitive reactivity, and mindfulness skills were assessed pre and post treatment. Patients in the MBCT group reported a significantly greater reduction in cognitive reactivity than those in the TAU group ( $d = .51$ ). The reduction of cognitive reactivity appeared to mediate the association between MBCT/TAU and decrease of depressive symptoms, using pre and post scores. The current study provides evidence that MBCT reduces cognitive reactivity and preliminary evidence that cognitive reactivity is a working mechanism of MBCT.

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Despite evidence-based treatments for major depression, residual depressive symptoms are very common and relapse rates are high (Zajecka, Kornstein, & Blier, 2013). This means that not only recovery from an initial depressive episode, but also reducing the vulnerability to relapse is very important. According to the cognitive model of depression (Beck, 2008), one of the core factors that characterises depressed individuals is the underlying negative beliefs about themselves. These negative beliefs, for example “If I make a mistake, this means I am a failure”, are called dysfunctional attitudes. Importantly, dysfunctional attitudes tend to persist latently, even after remission from a depressive episode, and can be easily activated by a sad mood (Scher, Ingram, & Segal, 2005). The ease with which these dysfunctional attitudes are activated by sad mood states is defined as *cognitive reactivity* (Van der Does, 2002).

Cognitive reactivity is not only an important factor in the onset (Kruijt et al., 2013) and maintenance of depressive symptoms (Struijs, Groenewold, Voshaar, & de Jonge, 2013), but especially important for depressive relapse/recurrence. Patients who experienced several depressive episodes reported higher cognitive reactivity compared with patients with just one previous episode (Elgersma et al., 2015). In addition, in prospective studies, those with higher post-treatment levels of cognitive reactivity appeared to have an earlier relapse/recurrence (Figuroa et al., 2015; Segal et al., 2006). However, non-replications of the effect of cognitive reactivity on relapse rates also exist (e.g. van Rijsbergen et al., 2013).

There are two common methods for measuring cognitive reactivity: the first consists of a mood induction accompanied by a scale to measure

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dysfunctional beliefs (Segal et al., 2006); the second consists of a self-report measure, the Leiden Index of Depression Sensitivity Revised [LEIDS-R] (Van der Does, 2002), asking the participant to indicate typical reactions to a sad mood. The LEIDS-R addresses cognitive reactions to a negative swing in mood and has been shown to differentiate between previously depressed and never-depressed individuals (Van der Does, 2002).

As cognitive reactivity seems to be related to the onset, maintenance, and recurrence of major depression, it is worth investigating whether cognitive reactivity can be reduced by psychological interventions. Previous studies have shown that cognitive behavioural therapy, but not pharmacological treatment, results in a reduction in cognitive reactivity, indicating that a reduction in cognitive reactivity is not a sheer effect of a reduction of depressive symptoms (Segal et al., 2006).

Another promising candidate to reduce cognitive reactivity in recurrent depression is Mindfulness-Based Cognitive Therapy (MBCT). MBCT was designed to prevent relapse in remitted depressed patients, and combines elements of cognitive therapy and mindfulness practices. The aim of MBCT is to teach participants to experience emotions, thoughts, and bodily sensations in a non-judgmental and compassionate way. Participants learn to become more aware of dysfunctional automatic patterns, such as depressive rumination and cognitive avoidance, and thereby to disengage from them (Segal, Williams, & Teasdale, 2002). Thereby MBCT teaches the ability to decentre from negative cognitive thoughts and disengage from dysfunctional cognitive processes, rather than to try to avoid them. MBCT has been shown to reduce relapse rates for depression and to effectively reduce residual depressive symptoms as well as symptom severity in current depression (Kuyken et al., 2016; Strauss, Cavanagh, Oliver, & Pettman, 2014). Only recently, research has focused more on the working mechanisms of MBCT (Gu, Strauss, Bond, & Cavanagh, 2015; van der Velden et al., 2015).

There is preliminary evidence that mindfulness-based interventions might result in a reduction in cognitive reactivity. Trait mindfulness skills seem to be negatively correlated with cognitive reactivity in university students (Raes, Dewulf, Van Heeringen, & Williams, 2009, Study 1). In a non-randomised controlled study, participants receiving MBCT showed a greater reduction in cognitive reactivity than those

on a MBCT waiting list acting as controls (Raes et al., 2009, Study 2). This finding was replicated in a study offering a mindfulness-based intervention to economically disadvantaged individuals with depressive symptoms. Cognitive reactivity decreased more over the course of the mindfulness-based intervention than during their own control period on the waiting list (Van der Gucht, Takano, Van Broeck, & Raes, 2015). Furthermore, Raes et al. (2009) also found a mediating effect of mindfulness skills on the relationship between MBCT and cognitive reactivity, indicating that it is an increase in mindfulness skills, rather than another non-specific therapy effect, that influences cognitive reactivity. Somewhat conflicting results of the effect of MBCT on cognitive reactivity have been found by Kuyken et al. (2010) in a sample of remitted depressed patients. Participants receiving MBCT showed increased cognitive reactivity post treatment compared with those on maintenance antidepressant medication. It was, however, found that although cognitive reactivity was predictive of relapse/recurrence in the antidepressant medication group, it no longer appeared to be predictive of relapse/recurrence in the MBCT group, possibly indicating a protective effect of MBCT. These somewhat conflicting results could also be partly explained by the fact that no baseline measure of cognitive reactivity was included and that the mood-induction method was used, whereas the other two studies made use of the LEIDS-R. In a systematic review on working mechanisms of mindfulness-based interventions Gu et al. (2015) reported preliminary evidence of cognitive reactivity as a possible working mechanism of the effect of mindfulness-based interventions on symptoms, but also mentioned that more evidence is needed.

In summary, there is ample evidence that MBCT reduces relapse/recurrence rates in recurrent depression, and some evidence that it might also reduce residual depressive symptoms. One of the possible working mechanisms is cognitive reactivity. However, randomised controlled studies are needed to further investigate this relationship. The aim of this study is to fill this gap by investigating the effect of MBCT on cognitive reactivity in remitted depressed patients participating in a randomised controlled trial comparing MBCT with treatment-as-usual (TAU). It is hypothesised that MBCT will result in a reduction in cognitive reactivity. Additionally, the mediating effect of cognitive reactivity on depressive symptoms will be investigated.

## Methods

### Study population and procedure

This study is part of a randomised controlled trial comparing MBCT with TAU in patients with three or more previous depressive episodes ( $N=205$ ) who were either in remission or currently depressed (van Aalderen et al., 2011). Measurements of cognitive reactivity were available for 174 participants, which had not been presented previously. In line with previous studies (Kuyken et al., 2010), we only selected those participants who were in full or partial remission at baseline according to DSM-IV criteria ( $n=115$ ) and excluded currently depressed participants ( $n=59$ ). This decision was based on the fact that the LEIDS-R was developed to be used in remitted depressed patients. The instruction of the LEIDS-R to imagine a slightly sad mood (see also measurement description) probably leads to different effects in remitted versus currently depressed patients. Exclusion criteria were: (1) one or more previous (hypo)manic episodes according to DSM-IV criteria; (2) current alcohol or drug abuse; (3) urgent need for psychiatric treatment; (4) problems hampering participation in a group; and (5) problems hampering completion of the questionnaires. Participants taking antidepressant medication were required to be on a stable dose for at least six weeks prior to inclusion and were asked to maintain this dose during the study period. After baseline measurements were taken, participants were randomised to either the MBCT or the TAU group. Post-measurements were taken after finishing MBCT (MBCT group) or after a 3-month waitlist period (TAU group).

The trial was approved by the local Medical Ethical Committee. All participants gave written informed consent. For a detailed description of all measurements and a CONSORT flowchart see van Aalderen et al. (2011).

### Measures

#### Cognitive reactivity

The LEIDS-R (Van der Does, 2002) is a 34-item questionnaire measuring cognitive reactivity. It consists of six subscales: hopelessness/suicidality; acceptance/coping; aggression; control/perfectionism; harm avoidance; and rumination. Participants are instructed to imagine being in a sad or down but not seriously depressed mood. An example item is "When I feel sad, I spend more time thinking about what my

moods reveal about me as a person." Participants are asked to indicate on a 5-point scale (from "not at all" to "very strongly") whether the items reflect their thoughts when experiencing a sad mood. Adequate validity and reliability (Cronbach's  $\alpha = .92$ ) are reported (Solis, Antypa, Conijn, Kelderman, & Van der Does, 2016).

#### Depressive symptoms

The Hamilton Rating Scale for Depression (HAM-D; Hamilton, 1960) is a 17-item standardised interview to measure the number and severity of depressive symptoms on a scale of 0–52. The HAM-D has good psychometric properties with good internal consistency (Cronbach's  $\alpha = .789$ ) and high inter-rater reliability ( $ICC = .937$ ) (Trajković et al., 2011). Additionally, self-reported levels of depression were investigated with the Beck Depression Inventory (BDI). The BDI is a 21-item self-report questionnaire to measure depressive symptoms. The scores indicate the following: 0–9 minimal depression; 10–18 mild depression; 19–29 moderate depression; 30–63 severe depression. The BDI has good psychometric properties with a high internal consistency (Cronbach's  $\alpha = .86$ ) in psychiatric patients (Beck, Steer, & Carbin, 1988).

#### Mindfulness skills

The Kentucky Inventory of Mindfulness (KIMS), is a self-report questionnaire measuring mindfulness skills with 39 items. The KIMS consists of four subscales: observe; describe; act with awareness; and accept without judgment. The KIMS has good psychometric properties in recurrent depression (Baum et al., 2010) with high internal consistency (subscales ranging from Cronbach's  $\alpha = .72$ –.88).

### Interventions

#### MBCT

Participants followed a standardised 8-week MBCT training based on the manual of Segal et al. (2002). The course consisted of weekly 2.5-hour group sessions and 1 day (6 hours) of silent meditation, and took place at the Radboudumc Centre for Mindfulness located at the Radboud University Medical Center in Nijmegen, the Netherlands. Treatment groups consisted of 8–12 participants. Mindfulness trainers were all experienced in working with groups of psychiatric patients, had received at least 1.5 years of mindfulness training and were experienced meditators with experience ranging from 2 to 20+ years.

## TAU

TAU consisted of a waitlist-control group receiving mental health care from general practitioners or specialised mental health care providers. Therefore, TAU was a naturalistic condition consisting of (in general high-quality) mental health care in the Netherlands, as for example, antidepressant medication or individual psychotherapy. Participants were asked not to change their medication during the study period.

## Statistical analyses

As the goal of the current study was to investigate specific effects of MBCT on cognitive reactivity and cognitive reactivity as a working mechanism, we based our analysis on the per-protocol sample, meaning that all participants attended at least four sessions of MBCT. A linear mixed model was used to compare post-measurement scores of cognitive reactivity between the groups (MBCT vs. TAU), controlling for baseline cognitive reactivity scores and baseline depressive symptoms. To take clustering of the data into account, we added a random group effect. Cohen's *d* effect size was computed. The same model was used to verify previously examined (van Aalderen et al., 2011) effects of condition on depressive symptoms and mindfulness skills in this sample. To explore whether reductions in cognitive reactivity mediate the effect of condition (MBCT vs. TAU) on depressive symptoms, a mediation analysis was conducted with condition (MBCT vs. TAU) as independent variable, change in depressive symptoms (from pre to post MBCT/post TAU) as dependent variable, and change in cognitive reactivity (from pre to post

MBCT/post TAU) as the hypothesised mediator. For all change scores, standardised residualised change scores were used (Kuyken et al., 2010). A nonparametric bootstrapping method was used to assess the indirect effect based on 1000 bootstrapped samples using bias corrected and accelerated 95% confidence intervals (BCa CI) as provided by Hayes [(2013) SPSS PROCESS macro version 2.13]. To estimate the effect size of the indirect effect,  $\kappa^2$  was computed which represents a "ratio to the maximum possible indirect effect" (Preacher & Kelley, 2011). We also tested the reverse model, using cognitive reactivity as the outcome variable, and change in depressive symptoms as mediator (*not* predicted by the theoretical model) to gain indications about causality in the absence of temporal order of variables.

## Results

### Participant characteristics

The demographic and baseline characteristics of participants are presented in Table 1. The groups (MBCT/TAU) did not differ on most demographic characteristics (gender, age, tertiary education, number of previous depressive episodes), cognitive reactivity, mindfulness skills, and depressive symptoms assessed with clinical interview (HAMD) at baseline. However, participants in the MBCT group reported a higher age of onset and somewhat lower baseline levels of depressive symptoms on the self-report questionnaire (BDI) than those in the TAU group. No follow-up assessments were available for two participants, so they were excluded from the analyses. They had significantly lower levels of cognitive

**Table 1.** Participant characteristics at baseline.

	Total sample ( <i>N</i> = 115) Mean (SD)	MBCT ( <i>n</i> = 63) Mean (SD)	TAU ( <i>n</i> = 52) Mean (SD)	MBCT vs. TAU <sup>a</sup>
<i>Demographic characteristics</i>				
Female	70.4%	68.3%	73%	$\chi^2(1) = .318, p = .573$
Age (years)	47.53 (11.67)	46.84 (11.58)	48.37 (11.83)	$t(113) = .696, p = .488$
Tertiary education	56%	58%	52%	$\chi^2(1) = .535, p = .465$
Age of onset (years)	22.93 (10.70)	25.32 (11.44)	20.12 (9.09)	$t(109) = -2.67, p = .010^b$
Number of depressive episodes	6.86 (6.46)	7.18 (7.52)	6.47 (4.95)	$t(110) = -.577, p = .565$
<i>Symptoms at baseline</i>				
Cognitive reactivity (LEIDS-R)	79.46 (21.64)	81.48 (22.09)	77.02 (21.04)	$t(113) = -1.1, p = .274$
Depressive symptoms-clinician (HAMD)	7.75 (5.08)	7.73 (5.47)	7.77 (7.17)	$t(113) = 0.41, p = .967$
Depressive symptoms-self report (BDI)	13.12 (7.43)	11.76 (7.18)	14.77 (7.47)	$t(113) = 2.195, p = .030^b$
Mindfulness skills (KIMS)	74.13 (15.96)	75.24 (15.63)	72.76 (15.79)	$t(112) = -.837, p = .404$

<sup>a</sup>Due to missing values the degrees of freedom differ between the analyses.

<sup>b</sup>Significant at  $p < .05$  level.

reactivity on baseline ( $p = .02$ ), but did not differ on other measures.

### Effect of MBCT vs. TAU on cognitive reactivity

Controlling for cognitive reactivity at baseline, depressive symptoms at baseline (HAMD), and age of onset, patients in the MBCT group showed significantly less cognitive reactivity compared with the TAU group at the end of treatment (group difference:  $-11.02$ , 95% CI  $[-18.29, -3.75]$ ,  $F(1, 56.99) = 9.22$ ,  $p = .004$ ). This effect represents a medium effect size, Cohen's  $d = .51$ . Excluding age of onset showed similar results with a slightly larger effect size (group difference:  $-13.08$ , 95% CI  $[-20.27, -5.88]$ ,  $F(1, 53.93) = 13.29$ ,  $p = .001$ , Cohen's  $d = .60$ ). Controlling for self-reported levels of depressive symptoms (BDI) instead of clinical rated symptoms (HAMD) led to similar effects. Paired sample  $t$ -tests revealed that the MBCT condition showed a significant decrease in cognitive reactivity over time (baseline  $M = 82.67$ , post  $M = 72.47$ ;  $t(60) = 4.52$ ,  $p < .001$ ), whereas the TAU condition showed a significant increase in cognitive reactivity (baseline  $M = 77.02$ , post  $M = 82.92$ ;  $t(51) = -2.23$ ,  $p = .03$ ).

### Effect of MBCT vs. TAU on depressive symptoms and mindfulness skills

Controlling for baseline scores and age of onset, patients in the MBCT group showed significantly less depressive symptoms (HAMD) (group difference:  $-2.88$ , 95% CI  $[-4.76, -1.01]$ ,  $F(1, 107) = 9.26$ ,  $p = .003$ , Cohen's  $d = .57$ ) and more mindfulness skills (group difference:  $+13.96$ , 95% CI  $[9.331, 18.60]$ ,  $F(1, 105) = 35.702$ ,  $p < .001$ , Cohen's  $d = .87$ ) compared with the TAU group at the end of treatment. Similar effects on depressive symptoms were observed when using

self-reported symptoms (BDI) instead of clinician-rated symptoms (HAMD). Paired sample  $t$ -tests revealed that the MBCT group showed a significant decrease in depressive symptoms (baseline HAMD  $M = 7.73$ , post HAMD  $M = 5.95$ ,  $t(62) = 2.60$ ,  $p = .012$ ) and significant increase in mindfulness skills (baseline KIMS  $M = 75.36$ , post KIMS  $M = 86.68$ ;  $t(60) = -5.87$ ,  $p < .001$ ) whereas no changes in the TAU group were observed (depressive symptoms: baseline HAMD  $M = 7.77$ , post HAMD  $M = 8.83$ ;  $p = .21$ ; mindfulness skills: baseline KIMS  $M = 72.76$ , post KIMS  $M = 70.77$ ,  $p = .16$ ).

### Mediation of depressive symptoms through cognitive reactivity

Figure 1 depicts that condition had a significant effect on change in cognitive reactivity,  $b = -.785$ , BCa CI  $[-1.129, -.4418]$ , change in cognitive reactivity in turn had a significant effect on change in depressive symptoms,  $b = .2294$ , BCa CI  $[.0358, .4231]$ . A significant indirect effect (because zero is not included in the 95% bias corrected confidence intervals) of condition on change in depressive symptoms through change in cognitive reactivity was observed,  $b = -.1802$ , BootBCa CI  $[-.3705, -.0501]$ . This mediation effect represents a medium effect size,  $\kappa^2 = .09$ , BCa CI  $[.0245, .1672]$ . Adding age of onset as a covariate in the model, led to similar results with a significant indirect effect,  $b = -.152$  BCa CI  $[-.3221, -.0294]$ . When testing the reversed model, using change in depressive symptoms as mediator and change in cognitive reactivity as outcome, a significant but slightly smaller indirect effect  $b = .124$  BootBCa CI  $[-.2461, -.0357]$ ,  $\kappa^2 = .07$ , BCa CI  $[.0165, .1203]$  was observed. Comparable effects in all mediation analyses were observed when using self-reported depressive

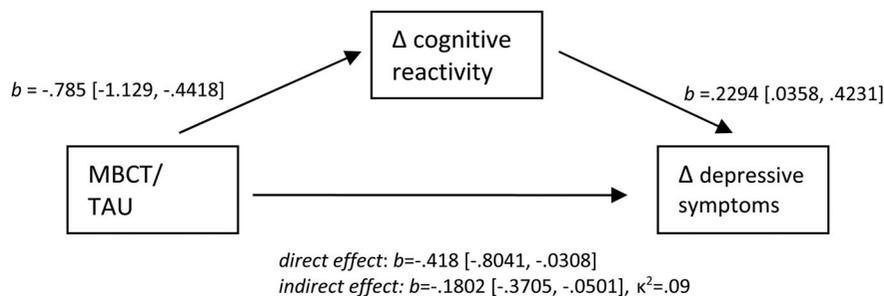


Figure 1. Mediation model.

symptoms (BDI) instead of symptoms rated by the clinician (HAMD).

## Discussion

The aim of this study was to investigate the effect of MBCT on cognitive reactivity in recurrently depressed patients in remission by using a randomised controlled design. Our results show that participants of the MBCT course demonstrated a significant reduction in cognitive reactivity. In contrast, the participants of the TAU group showed a significant increase in cognitive reactivity, possibly indicating that the MBCT even inhibited a natural increase in cognitive reactivity over time. Furthermore, by using baseline and post-treatment scores, we found that cognitive reactivity partially mediated the effect on depressive symptoms.

Cognitive reactivity is an important factor in the relapse/recurrence of depressive episodes (Beck, 2008; Figueroa et al., 2015). Therefore, it is important to investigate which treatments can reduce cognitive reactivity. The current results indicate that MBCT might be more effective than TAU in reducing cognitive reactivity. These results are in line with previous non-randomised studies on the effect of mindfulness-based interventions on cognitive reactivity (Raes et al., 2009; Van der Gucht et al., 2015). Our findings are partly in contrast with results by Kuyken et al. (2010) who found *increased* cognitive reactivity in participants receiving MBCT. However, in their study, cognitive reactivity was only related to relapse in the TAU group, indicating that MBCT diminished the toxic effect of the relationship between cognitive reactivity and outcome. Importantly, in contrast to Kuyken et al. (2010), we used the LEIDS-R instead of a mood-induction method. In a recent comment, Raes (2015) argued that both methods possibly tap into different stages of cognitive reactivity: the mood-induction method would then measure dysfunctional thoughts more as products of the mind, whereas the LEIDS-R specifically investigates dysfunctional thinking as a mental process in reaction to sad mood. This could partly explain the different results, as MBCT is not aiming at changing negative thoughts, but instead teaches participants to react differently on negative mood or initial dysfunctional thoughts.

Next to the effect of MBCT on cognitive reactivity as an outcome, we found a mediating effect of cognitive reactivity on the reduction in depressive symptoms indicating that cognitive reactivity is a working mechanism of MBCT. This is in line with the theoretical

rationale of MBCT (Segal et al., 2002) and previous findings (Gu et al., 2015). However, because we also found a significant effect when testing the “reversed model” (change in depressive symptoms mediating change in cognitive reactivity), which would *not* be predicted by the theoretical model of MBCT, conclusions should to be drawn with caution. Changes in cognitive reactivity and depressive symptoms seem to be interrelated when measured simultaneously. Longitudinal studies taking the temporal order of variables into account are needed to investigate the mediating effect of cognitive reactivity on depressive symptoms.

Our results suggest that we might consider how to strengthen the effect of MBCT on the reduction in cognitive reactivity. We might, for example, emphasise this mechanism even more in the psycho-educational part of the MBCT course. Or we might increase the practice of recognising negative mood and accompanying thoughts during sitting meditations in the second half of the course.

It is important to keep in mind, however, that cognitive reactivity is not the only proposed working mechanism of MBCT. Other assumed mechanisms of change in MBCT include for example mindfulness skills, self-compassion, and psychological flexibility (Gu et al., 2015). Kuyken et al. (2010) reported an interaction between cognitive reactivity and self-compassion skills, showing that in remitted depressed patients participating in the MBCT course the negative prognostic value of cognitive reactivity was mitigated by an increase in self-compassion. The interaction between different working mechanisms goes beyond the scope of this study, but future research should focus on disentangling the mechanisms of change of MBCT and their interconnectedness by combining the results of clinical randomised controlled trials with other designs, such as dismantling designs, individual difference designs, and experimental manipulations (van der Velden et al., 2015). Thereby we could learn more about the exact working mechanisms of MBCT and could use this knowledge to improve the curriculum and to be better able to predict which patients would benefit most from MBCT.

Several strengths and limitations of this study should be noted. First of all, the current study is one of the first to specifically investigate the effects of MBCT on cognitive reactivity in recurrently depressed patients by using a randomised controlled design with pre and post measures of cognitive reactivity, adding important knowledge to the existing literature.

Previous research has shown that levels of cognitive reactivity predict relapse rates (Figuroa et al., 2015; Kuyken et al., 2010). Figuroa et al. (2015) calculated that a 20-point increase on the LEIDS-R resulted in a 10–15% higher chance of relapse. Relating their figures to the current study would indicate that a decrease in LEIDS-R of 11 points could have a substantial effect on relapse rates, which should be further investigated.

The limitations of this study give several directions for future research. First, it should be noted that we compared MBCT with a waitlist-control group receiving treatment as usual. Therefore, we do not know how much of the effect is based on specific aspects of mindfulness training or non-specific therapy effects as peer support, hope or activation. In addition, the proposed mediator and outcome were measured simultaneously. Finding a variable statistically mediating an effect does not prove that it is a working mechanism, however, it is an important step in narrowing down the possible variables of influence (Kazdin, 2007). Therefore, further longitudinal research is needed to draw conclusions about causality. Whereas the focus of the current study was on investigating cognitive reactivity as an outcome and mediator of MBCT in a pre-post design, it would be important to also investigate the predictive effect of cognitive reactivity on relapse rates compared with a control group in longitudinal designs. Finally, as already noted, we used the LEIDS-R questionnaire (Van der Does, 2002) to measure cognitive reactivity rather than a mood-induction method (e.g. Kuyken et al., 2010). The LEIDS-R investigates the ease in which the participant reacts to a sad mood with dysfunctional beliefs. This is based on the assumption that dysfunctional beliefs tend to persist in remitted depressed patients (Scher et al., 2005) and can be activated by low mood states, which forms a vulnerability factor for relapse. However, it is therefore not possible with the current design to differentiate whether the overall level of dysfunctional beliefs was reduced after the MBCT or whether, in particular, the *reaction* to a sad mood with dysfunctional beliefs decreased. It would be valuable if future research investigated the effect of MBCT on cognitive reactivity as well as on overall levels of dysfunctional beliefs, independent of mood or stress. In addition, future research directly comparing the differences and similarities of the LEIDS-R and the mood-induction method to measure cognitive reactivity would be valuable. The significant advantages of the LEIDS-R are its applicability in

clinical practice and that it excludes the possibility of a failing mood induction; however, it requires insight in one's own cognitive processes. It would also be useful if future research explored whether the LEIDS-R successfully excludes social desirable answers and answers merely based on knowledge obtained during MBCT, rather than actual behaviour.

Taken together, the present study provides evidence that MBCT reduces cognitive reactivity based on a randomised controlled design in recurrently depressed patients in remission. Furthermore, the results add preliminary evidence to the existing literature about the theoretical assumption that the reduction in cognitive reactivity decreases depressive symptoms and is therefore a working mechanism of MBCT.

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No potential conflict of interest was reported by the authors.

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