Cognitive Therapy and Research (2019) 43:603–610
https://doi.org/10.1007/s10608-018-9972-z

ORIGINAL ARTICLE

A Prospective Test of the Metacognitive Model of Depression in Previously Depressed Individuals

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Published online: 13 October 2018

Abstract

Metacognitive theory proposes that depression is caused by excessive rumination, which is in turn maintained by maladaptive positive and negative beliefs about rumination ("metacognitions") and reduced executive control. Moreover, the metacognitive model asserts that metacognitions are maintained by prolonged depression symptoms. However, no studies have tested the metacognitive model of depression prospectively in a clinical population. Currently remitted adults with recurrent depressive disorder ($N = 105$) reported depression symptoms at five time points over a 12-month period. Based on this data, we used latent growth modelling to estimate depression levels and symptom trajectories. Positive metacognitions were associated with rumination, while negative metacognitions and rumination predicted higher depression levels, but not symptom recurrence. Moreover, depression levels and symptom recurrence predicted positive and negative metacognitions, as well as rumination. There was no association between metacognitions and reduced executive control. The present study lends partial support for the metacognitive model, but raises questions of the relevance of metacognitions as a proximal vulnerability marker for symptom recurrence.

Keywords Metacognitions · Metacognitive beliefs · Depression · Executive control

Metacognitive theory proposes that depression is caused by excessive rumination, which is in turn maintained by certain maladaptive beliefs about rumination, or "metacognitions" (Wells 2011; Wells and Matthews 1996). Rumination is a repetitive and passive focus on one's depressive symptoms and its consequences (Nolen-Hoeksema 1991). Depressed individuals who habitually engage in rumination tend to believe that this helps them to recover from the depression (positive metacognitions; Wells 2011). This was first demonstrated by Papageorgiou and Wells (2001a), who showed that all patients with recurrent depression that was interviewed reported that there were advantages of rumination (reflecting themes concerning rumination as a viable coping strategy). Subsequent studies have replicated this finding in a large, non-clinical sample (Solem et al. 2016) and in a prospective study (Hjemdal et al. 2016). As rumination leads to reduced problem solving, reduced social support and increased depression (Nolen-Hoeksema et al. 2008), depressed individuals also hold negative beliefs about rumination, that is, thinking that rumination is uncontrollable, and harms mental health and social relationships (negative metacognitions). Thus, depressed individuals assume that rumination is helpful, but also believe it is harmful and not under control. This effectively gridlocks the individual in a vicious cycle (Papageorgiou and Wells 2003).

The metacognitive model of depression (Fig. 1) has mainly been examined using cross-sectional studies. Path analyses have shown support for the metacognitive model in depressed and non-depressed individuals (Papageorgiou and Wells 2003). Similar findings have been found in diverse cultural contexts, and most recently in a large, non-clinical sample in Norway (Solem et al. 2016). To our knowledge, only one study has used a prospective design to investigate the metacognitive model, although only in a non-clinical sample (Papageorgiou and Wells 2009). To establish the

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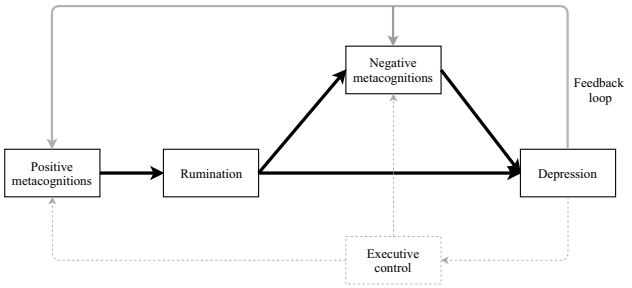

Fig. 1 The metacognitive model of depression

causal role of metacognitions in depression, there is a need for studies testing the metacognitive model using longitudinal data in clinical populations.

Previously depressed individuals are often burdened by residual depression symptoms, which is among the most important predictors for relapse (Paykel 2008). These individuals also show increased levels of negative metacognitions, which have therefore been proposed as a vulnerability factor for depressive relapse (Halvorsen et al. 2015). Hence, according to the metacognitive model, residual symptoms are maintained because patients still hold positive metacognitions that both motivates for more rumination and negative metacognitions that demotivates the stopping of rumination. Residual symptoms in the presence of metacognitions is therefore likely to increase the probability for symptom recurrence in previously depressed individuals.

The metacognitive model emphasizes that metacognitions are reinforced by prolonged experiences of depression symptoms, through what Wells and Matthews (1996) has termed "belief elaboration". That is, that prolonged rumination inhibits adaptive coping (e.g., social isolation, reducing activity levels), and leads to performance deficits and inflexible attentional control. Together, this reduces the processing of information that is incompatible with their beliefs about rumination as harmful and out of control. The model posits that worsening in depression symptoms should reinforce negative metacognitions, but no studies have examined the presence of this feedback loop.

The grid lock of metacognitions, rumination and depression symptoms is claimed to be exacerbated by reduced executive control (Matthews and Wells 2004). Reduced metacognitive efficiency (i.e., subjective cognitive problems in executive functions and memory) has been proposed as an important depressogenic by-product, contributing to metacognitions (Papageorgiou and Wells 2003). Metacognitions and executive control are conceptually related (Fernandez-Duque et al. 2000; Wells and Matthews 1996). Reduced executive control is often experienced in conjunction with depression (Snyder 2013), and can predict risk of depression relapse (Majer et al. 2004).

Only one study has examined the association between metacognitions and executive control using objective neuropsychological tests (Kraft et al. 2017). This study, in a mixed clinical/non-clinical population, showed that negative metacognitions were associated with reduced ability shift between mental sets. This was attributed to the fact that individuals with low executive control tend to fail more often when attempting to stop rumination (Koster et al. 2011). However, it is unknown whether reduced executive control is associated with metacognitions in previously depressed individuals, and whether this contributes to symptom recurrence.

The relevance of other executive functions in the maintenance of metacognitions is largely unknown. Even though reduced inhibitory control has been proposed to be a gateway for negative thought intrusions and increase the tendency to ruminate (De Raedt and Koster 2010), Kraft et al. (2017) found no association between metacognitions and reduced inhibitory control as measured by the Stop-Signal Task. One explanation may be that inhibitory control is only relevant when regulating the influx of low-level stimuli, and not for higher-order cognition. Yet, this null-finding may also reflect methodological limitations with the particular cognitive task.

The present study tested the metacognitive model of depression in previously depressed individuals using a prospective design. In line with the metacognitive model, we hypothesized that positive metacognitions (beliefs about rumination as helpful) are associated with rumination, and that rumination and negative metacognitions (beliefs that rumination is uncontrollable and harmful) predict depression symptoms and symptom recurrence. In accordance with the feedback loop described in the metacognitive model, we hypothesized that depression symptoms and symptom recurrence predict positive and negative metacognitions. Finally, we examined whether there was an association between reduced executive control and metacognitions.

Method

Participants

We obtained data from an ongoing study of the effect of attention bias modification (ABM) on residual depression symptoms among patients with remitted depression (study no. NCT02648165 in ClinicalTrials.gov). Participants were recruited from an outpatient clinic in the Department of Psychiatry, Diakonhjemmet Hospital in Oslo, Norway, but also from other clinical sites, by local advertisements, and via social media. Participants provided written informed consent, and the study was approved by the Regional Ethical Committee for Medical and Health Research for Southern Norway (2014/217/REK sør-øst D).

In total, 105 participants were included in the present study. Inclusion criteria were recurrent major depressive disorder (history of > 1 depressive episode), age between 18 and 65 years, and fluency in Norwegian. Exclusion criteria were current major depressive disorder, the presence of current or former neurological disorder, substance use disorder, attention-deficit disorder, head trauma, psychosis, or bipolar disorder. These criteria were assessed by trained administrators using the *Mini-International Neuropsychiatric Interview* (Sheehan et al. 1998).

Measures

Depression Symptoms

Depression symptoms was assessed using the Beck Depression Inventory II (BDI). Good psychometric properties have been documented (Beck et al. 1996). The Cronbach's alpha was 0.89 in the present study, indicating good internal consistency.

Rumination

Rumination was assessed using the Ruminative Responses Scale (RRS; Nolen-Hoeksema and Morrow 1991). This 22-item scale assesses the tendency to ruminate in response to depressed mood. Cronbach's alpha was 0.89. Factor analysis of this scale has resulted in three factors (Treynor et al. 2003). We computed the brooding factor from five of the items (e.g.,"Thinking: What am I doing to deserve this?"), reflecting the tendency to engage in "passive comparison of one's current situation with some unachieved standard" (Treynor et al. 2003, p. 256). We chose to use the brooding factor, as it, compared to the full RRS, has been found to represent a more maladaptive form of rumination (Nolen-Hoeksema et al. 2008) and be stronger linked to executive control deficits (Koster et al. 2011).

Positive Metacognitions

Positive metacognitions were assessed using The Positive Beliefs about Rumination Scale (PBRS; Papageorgiou and Wells 2001b). This nine-item scale assesses beliefs about the benefits of rumination (e.g., "I need to ruminate about my problems to find the causes of my depression"). Good psychometric properties have previously been documented (Luminet 2004). The Cronbach's alpha in the present study was 0.90.

Negative Metacognitions

The Negative Beliefs about Rumination Scale (NBRS; Papageorgiou and Wells 2001a) was used to assess beliefs about the uncontrollability and harm (subscale 1, e.g., "Rumination about my problems is uncontrollable") and interpersonal consequences of rumination (subscale 2, e.g., "People will reject me if I ruminate"). Good psychometric properties have been documented (Luminet 2004). The Cronbach's alpha was 0.81.

Executive Control

A Stroop task, the Color-Word Interference Test (CWIT; Delis et al. 2001), was administered to measure executive control. The task measures important executive factors related to depression and rumination (inhibition and switching ability), and is the most common task reported in the depression literature (Miyake et al. 2000; Snyder 2013). In this test, the subject is asked to name the printed color of color-word names. This requires the subject to inhibit the pre-potent reading response of the word. We report completion time (seconds) on the inhibition task variant (which requires the participant to inhibit the pre-potent reading response), as well as the inhibition/switching variant (which also requires the participants to occasionally switch to the non-inhibitory responses).

Procedure

The study had a multi-wave follow-up design, where depression symptoms was assessed at five time points: baseline (Time 0), after 2 weeks (Time 1), 1 month (Time 2), 6 months (Time 3) and 12 months (Time 4). Positive metacognitinos, negative metacognitions, and brooding were assessed at Time 0 and Time 4. Executive control was assessed at Time 0. All participants attended the assessments at Time 0 and 1. Ten participants (9.5%) did not attend assessment at Time 2, 21 participants (20%) at Time 3, and 21 (20%) participants at Time 4.

Statistical Analyses

We used latent growth modelling (LGM) within a structural equation (SEM) framework to examine the bidirectional longitudinal effects between metacognitions and depression. Compared to traditional longitudinal models, LGM can handle unequally spaced time points and yields more statistical power (Curran et al. 2010). Moreover, LGM can also capture features of individual differences in depression over time, such as individual's average depression levels over time (random intercept) and the individual's average amount of linear change of depression over time, i.e., the symptom trajectory (random slope).

First, we estimated the intercept and slope of depression between Time 0 and Time 4. Second, we examined the effects of positive metacognitions, negative metacognitions,

and brooding at Time 0 (predictors) on depression levels and symptom trajectories (the dependent variables). A priori power analysis (three predictors, $R^2 = 0.15$, $\alpha = 0.05$, $1 - \beta = 0.80$) indicated a minimum sample size of 69 to refute the null-hypothesis. We examined the effect of each predictor separately, and then with all the predictors included as specified by the metacognitive model (Fig. 1): Depression levels and symptom trajectories regressed on negative metacognitions and brooding at Time 0, while simultaneously regressing negative metacognitions on brooding, and brooding on positive metacognitions. Third, we examined the feedback effects of depression levels and symptom trajectories (predictors) on positive metacognitions, negative metacognitions, and brooding at Time 4 (dependent variables). We examined the effects on each dependent variable separately, and then with all dependent variables included in the model simultaneously. Finally, we examined bivariate correlations between metacognitions, and executive control.

Single items missing on the BDI, RRS, PBRS and NBRS (< 0.2% of total items) were replaced by the mean of the scale or subscale for each subject. Missing data was handled by maximum likelihood estimation with robust standard errors (MLR) and pairwise deletion (correlational analysis). We used Mplus 7.4 to estimate the LGM and test the metacognitive model, while demographic and correlational statistics were obtained using IBM SPSS 25.

Results

Participant Characteristics

Demographic and clinical characteristics are presented in Table 1. Positive metacognitions, negative metacognitions, rumination, and depression symptoms at Time 0–4 are presented in Table 2. Correlations between positive metacognitions, negative metacognitions, brooding, and inhibition/switching are presented in Table 3.

Correlations between the variables at T0 was examined in a confirmatory factor analysis, and indicated that the various measures tap distinct constructs (BDI-brooding: r = .45, p < .01; BDI-PBRS: r = .20, p = .06; BDI-NBRS: r = .27, p < .01; PBRS-brooding: r = .50, p < .01, PBRS-NBRS: r = .17, p = .14; NBRS-brooding: r = .46, p < .01). There were no effects of ABM on brooding ($\beta = 0.14$, p = .22), positive metacognitions ($\beta = 0.08$, p = .45), or negative metacognitions (full scale: $\beta = 0.11$, p = .30; subscale 1: $\beta = 0.14$, p = .20; subscale 2: $\beta = 0.02$, p = .87) at T4.

Latent Growth Model of Depression Symptoms

The latent growth model showed that average depression level (intercept) was 11.9 BDI points. There was no statistical tendency in average symptom trajectory (slope, $b = -0.24$, p = .43). However, variance estimates for intercept and slope were statistical significant (p < .05), indicating meaningful between-person differences in depression level and symptom trajectory. There was a negative correlation between depression level and symptom trajectory ($\beta = -0.43$, p < .05). There were no effects of ABM on intercept ($\beta = 0.14$, p = .17) or slope ($\beta = -0.15$, p = .36).

Table 1 Demographic and clinical statistics

Variable	
Age, years, M (SD)	36.0 (13.0)
Sex (females), n (%)	76 (72%)
Educational level (ISCED), M (SD)	6.0 (1.1)
Use of SSRI/SNRI medication, n (%)	29 (28%)
Percentage of the sample reporting BDI score	
> 12 (clinical cut-off)	47%
4–12	39%
Number of depressive episodes, M (SD)	4.7 (7.5)
Inhibition, seconds to complete, M (SD)	51.2 (12.8)
Inhibition/switching, seconds to complete, M (SD)	58.7 (11.3)

ISCED level 6 corresponds to a bachelor's degree or equivalent

SSRI/SNRI serotonin specific/serotonin-norepinephrine reuptake inhibitor, *BDI-II* Beck's Depression Inventory II, *BAI* Beck's Anxiety Inventory, *ISCED* International Standard Classification of Education

Effects of Metacognitions and Brooding on Depression

Brooding ($\beta = 0.36$, p < .01), positive metacognitions ($\beta = 0.20$, p < .05), and negative metacognitions ($\beta = 0.34$, p < .01) predicted depression levels. Controlling for brooding, the effect of positive metacognitions disappeared ($\beta = 0.07$, p = .48), but the effect of negative metacognitions was still present ($\beta = 0.23$, p < .05). There were no effects of brooding ($\beta = -0.07$, p = .73), positive metacognitions ($\beta = -0.15$, p = .40), or negative metacognitions ($\beta = -0.21$, p = .26) on symptom trajectories.

When specifying the analysis in line with the metacognitive model, positive metacognitions predicted brooding, while negative metacognitions and brooding predicted depression levels, but not symptom trajectories (Fig. 2). Substituting brooding with the full-scale RRS or negative metacognitions with the two NBRS subscales, did not improve predictions.

Table 2 Variables means (and standard deviations) at Time 0–4

Variable	Baseline (T0)	2 weeks (T1)	1 month (T2)	6 months (T3)	12 months (T4)
Depression score (BDI)	12.7 (8.4)	9.9 (8.9)	10.1 (8.9)	11.6 (9.9)	14.8 (13.0)
Rumination (RRS)	51.5 (12.0)				49.5 (14.1)
Brooding subscale	11.1 (3.4)	–	–	–	10.8 (3.6)
Positive metacognitions (PBRS)	17.6 (5.9)	–	–	–	17.0 (5.3)
Negative metacognitions (NBRS)	21.8 (6.1)	–	–	–	20.8 (6.5)

BDI Beck's Depression Inventory-II, *RRS* ruminative responses scale, *PBRS* positive beliefs about rumination scale, *NBRS* negative beliefs about rumination subscale

Table 3 Correlations between rumination, metacognitions, and executive control

Variable	1	2	3	4	5	6	7
1. RRS brooding, Time 0	–						
2. RRS brooding, Time 4	0.44**	–					
3. PBRS, Time 0	0.39**	0.03	–				
4. NBRS, Time 0	0.38**	0.44**	0.22*	–			
5. PBRS, Time 4	0.40**	0.50**	0.39**	0.31**	–		
6. NBRS, Time 4	0.30*	0.70**	0.15	0.63**	0.43**	–	
7. Inhibition, Time 0	−0.01	−0.17	0.03	−0.05	0.16	−0.05	–
8. Inhibition/switching, Time 0	0.18	−0.07	0.01	−0.02	0.11	−0.05	0.63**

RRS ruminative responses scale, *PBRS* positive beliefs about rumination scale, *NBRS* negative beliefs about rumination subscale
**p < .01; *p < .05

Effects of Depression on Metacognitions and Brooding (Feedback Loop)

Depression levels predicted positive metacognitions ($\beta = 0.33$, $p < .05$), negative metacognitions ($\beta = 0.33$, $p < .05$), and brooding ($\beta = 0.53$, $p < .01$). Symptom trajectories predicted positive metacognitions ($\beta = 0.49$, $p < .05$) and brooding ($\beta = 0.62$, $p < .01$), and there was an effect on negative metacognitions at statistical trend-level ($\beta = 0.38$, $p = .10$).

Entering all dependent variables in the model simultaneously, resulted in medium effects of depression levels and symptom trajectories on positive metacognitions, negative metacognitions, and brooding (Fig. 3). However, the effect of depression levels on positive metacognitions, and the effect of symptom trajectories on negative metacognitions, were at statistical trend-level ($p < .10$).

Correlation Between Metacognitions and Inhibition/Switching

There was no correlation between inhibition and positive metacognitions ($r = .03$, $p = .77$) or negative metacognitions ($r = -.05$, $p = .64$), or between inhibition/switching and positive metacognitions ($r = .01$, $p = .92$) or negative metacognitions ($r = -.02$, $p = .82$).

Discussion

We examined the bidirectional links between rumination, metacognitions, and depression symptoms over a 12 month-period in currently remitted but previously depressed patients. This is the first study testing the metacognitive model of depression in a clinical sample using a prospective design. Results showed that positive metacognitions, negative metacognitions, and rumination predicted depression levels, but not symptom trajectories. Testing for feedback effects, we found that depression levels and symptom trajectories predicted positive metacognition, negative metacognitions and rumination, although some effects were at the statistical trend-level. There were no correlations between metacognitions and inhibition/switching.

We found that positive metacognitions predicted depression levels, but the effect was accounted for by rumination. Rumination and negative metacognitions had independent contributions to depression levels. This finding is in line with previous studies and the metacognitive model (e.g., Papageorgiou and Wells 2003, 2009; Solem et al. 2016). Thus, negative metacognitions explain the general susceptibility to experience depression symptoms.

Contrary to the metacognitive model, negative metacognitions and rumination did not predict symptom trajectories. Individuals with recurrent depression with lower levels of

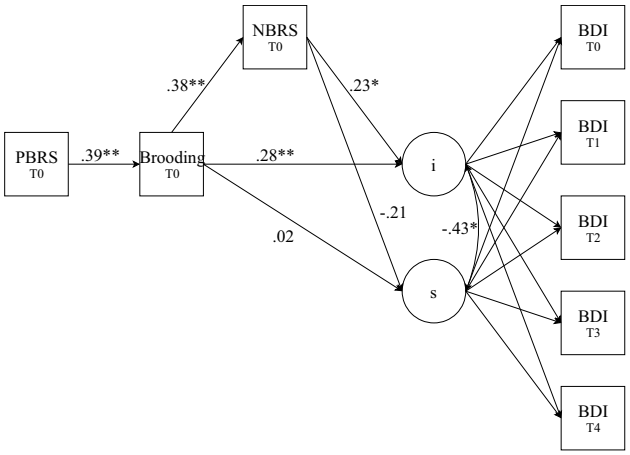

Fig. 2 Predictions of the metacognitive model (standardized coefficients) on depression levels (intercept) and symptom trajectories (slope) between time 0 and time 4. *i* intercept, *s* slope, *PBRS* positive beliefs about rumination, *NBRS* negative beliefs about rumination. **p < .01; *p < .05; ^p < .1

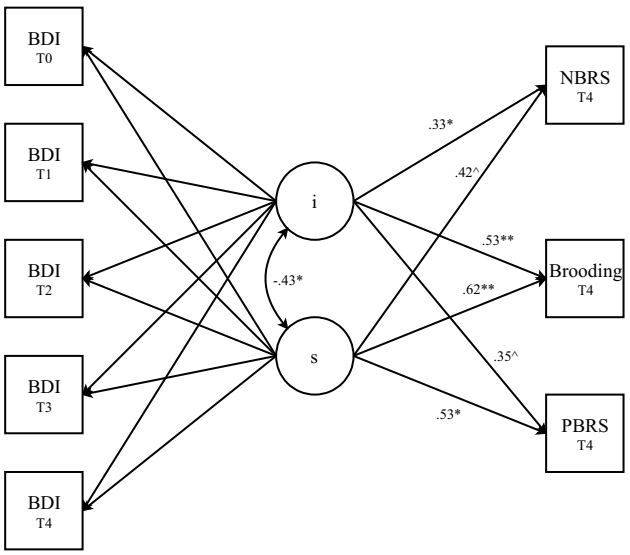

Fig. 3 Predictions of depression level (intercept) and trajectory (slope) on positive and negative metacognitions and brooding at time 4 (standardized coefficients). *PBRS* positive beliefs about rumination, *NBRS* negative beliefs about rumination. **p < .01; *p < .05; ^p < .1

metacognitions and rumination seem to be as vulnerable to symptom recurrence as individuals with higher levels of metacognitions and rumination. In other words, decreasing metacognitions is likely to reduce depression symptoms, but this is probably not enough to prevent the recurrence of symptoms in the next 12 months. It could be that metacognitions and rumination only convey a long-term risk for symptom recurrence (beyond one year). Short-term relapse prevention in these patients should go beyond targeting metacognitions and rumination (e.g., increase social functioning; Paykel 2008).

Because the present study also assessed metacognitions at 12-months follow-up, we were able to examine the hypothesized feedback effects of depression on metacognitions (Matthews and Wells 2004; Papageorgiou and Wells 2003), which has not been addressed in previous studies. Results suggest that remitted patients who have residual symptoms continue to ruminate, and continue to believe that rumination is uncontrollable, and harmful to their mental health and social relationships. Moreover, if they experience worsening, they also tend to believe that rumination is helpful.

Unfortunately, we could not examine whether depression leads to changes in metacognitions. Studies applying multiwave assessments of metacognitions could clarify whether symptom recurrence is followed by an intensification of metacognitions, which could point to a belief elaboration process (Wells and Matthews 1996).

Contrary to previous studies, there was no association between metacognitions and executive control. The present study assessed executive control using a task requiring the participant to stop a pre-potent response (inhibition), as well as shifting to an alternative response (inhibition/switching). Results showed that there was no association between ability to inhibit pre potent responses and metacognitions. This is in accordance with previous findings (Kraft et al. 2017). Together this indicate that metacognitions are not related to inhibitory control.

Although Kraft et al. (2017) found an association between metacognitions and a set-shifting task, there was no association between metacognitions and the inhibition/switching in the present study. One explanation could be that the inhibition/switching task taps different executive functions (Miyake and Friedman 2012). Moreover, different executive control measures tend not to correlate well, because of differences in reliability, strategy use, and task impurity problems (Friedman and Miyake 2017). This could explain the null finding. Differences in the sample characteristics may also be an explanation, as Kraft et al. (2017) examined the association in a mixed group of both healthy individuals and individuals with previous and current depression. This calls for more research on the relationship between metacognitions and set-shifting/switching tasks.

The present study has several strengths. The longitudinal design take us one step closer to establishing the causal effects of metacognitions. The sample is well defined and relevant to clinical practice. Because depression was assessed at multiple time points, we were able to examine effects on both stability and change in depression symptoms. Executive control was assessed using an objective neuropsychological test. Yet, the present study also has some limitations. The bidirectional links between metacognitions and depression needs to be confirmed in a currently

depressed population. Although there were no effect of the ABM condition on depression, brooding and metacognitions, we cannot rule out the possibility that the context of being enrolled in an intervention study affected the results. Studies in other, more naturalistic samples (e.g., clinically depressed population), are needed. Experimental studies are also needed to conclude regarding the causal and mediating role of metacognitions.

The present study lends partial support to the metacognitive model of depression. Positive beliefs about rumination are associated with rumination, while rumination and negative beliefs about rumination predicts depression. Contrary to the model, metacognitions does not seem to be a proximal vulnerability factor for worsening, nor related to reduced executive control. We found evidence for a feedback effect from depression to metacognitions: Depression symptoms leads to negative beliefs about rumination, while worsening leads to positive beliefs about rumination. Regardless, the present study cannot elucidate whether depression further exacerbates metacognitions, as claimed by the metacognitive model.

This study raises some important questions for future research. Future studies should include objective measures of executive control, to establish the relevancy of this factor. Even though the current findings suggests that metacognitions do not predict symptom recurrence in previously depressed individual, this relationship could be different in a treatment setting. A majority of the participants were women. However, the sample reflects the 1.7 increased incidence risk for depression observed in women (Albert 2015). The study is therefore not unreasonably skewed because of under-sampling of men. Nevertheless, metacognitive research could in general benefit from examining sex differences in metacognitions.

Emerging studies of metacognitive therapy (MCT) for depression, where metacognitions are the main target for treatment, shows promise (Hagen et al. 2017; Hjemdal et al. 2017). Future studies should assess metacognitions, rumination, and depression at several time points (pre, peri, and post treatment), as this will improve the statistical modelling of the lagged associations, and enable the analysis of bidirectionality, stability and change in these important variables.

Acknowledgements We want to thank the following research assistants: Inger Marie Andreassen, Adrian Dahl Askelund, Dani Beck, Sandra Aakjær Bruun, Jenny Tveit Kojan, Nils Eivind Holth Landrø, Elise Solbu Kleven and Julie Wasmuth. Further, we thank Kari Agnes Myhre and Senior Consultant, Research leader; MD, Phd Erlend Bøen at Diakonhjemmet Hospital, Division of Psychiatry, for help and support during the recruiting period. We also thank our external recruitment sites, Unicare, Coperiosenteret AS, Torgny Syrstad, MD, Synergi Helse AS and Lovisenberg Hospital.

Funding This work was supported by grants from the Research Council of Norway, Norges Forskningsråd (NO) project number 247372 (NIL) and The South East Norway Health Authority Research Funding, Helse Sør-Øst RHF project number 2015052 (NIL). The Department of Psychology, University of Oslo has also supported the project.

Compliance with Ethical Standards

Conflict of interest Nils Inge Landrø has received consultancy fees and travel expenses from Lundbeck. Brage Kraft, Rune Jonassen, Vidar Ulset and Tore Stiles declare that they have no conflict of interest.

Ethical Approval All procedures in the present study were in accordance with the ethical standards of the national research committee in Norway and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual subjects participating in the study.

Research Involving Animal Rights This article does not contain any studies with animals performed by any of the authors.

References

Albert, P. R. (2015). Why is depression more prevalent in women? *Journal of Psychiatry & Neuroscience: JPN, 40*(4), 219–221. https://doi.org/10.1503/jpn.150205.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Manual for the Beck Depression Inventory–II. San Antonio, TX: Psychological Corporation.

Curran, P. J., Obeidat, K., & Losardo, D. (2010). Twelve frequently asked questions about growth curve modeling. *Journal of Cognition and Development, 11*(2), 121–136. https://doi.org/10.1080/15248371003699969.

De Raedt, R., & Koster, E. H. (2010). Understanding vulnerability for depression from a cognitive neuroscience perspective: A reappraisal of attentional factors and a new conceptual framework. *Cognitive, Affective, & Behavioral Neuroscience, 10*(1), 50–70. https://doi.org/10.3758/CABN.10.1.50.

Delis, D. C., Kaplan, E., & Kramer, J. (2001). *Delis-Kaplan executive function system*. San Antonio: Psychological Corporation.

Fernandez-Duque, D., Baird, J. A., & Posner, M. I. (2000). Executive attention and metacognitive regulation. *Consciousness and Cognition, 9*(2), 288–307. https://doi.org/10.1006/ccog.2000.0447.

Friedman, N. P., & Miyake, A. (2017). Unity and diversity of executive functions: Individual differences as a window on cognitive structure. *Cortex, 86*, 186–204. https://doi.org/10.1016/j.cortex.2016.04.023.

Hagen, R., Hjemdal, O., Solem, S., Kennair, L. E. O., Nordahl, H. M., Fisher, P., & Wells, A. (2017). Metacognitive therapy for depression in adults: A waiting list randomized controlled trial with six months follow-up. *Frontiers in Psychology*. https://doi.org/10.3389/fpsyg.2017.00031.

Halvorsen, M., Hagen, R., Hjemdal, O., Eriksen, M. S., Sørli, Å. J., Waterloo, K., … Wang, C. E. (2015). Metacognitions and thought control strategies in unipolar major depression: A comparison of currently depressed, previously depressed, and never-depressed individuals. *Cognitive Therapy and Research, 39*(1), 31–40. https://doi.org/10.1007/s10608-014-9638-4.

Hjemdal, O., Hagen, R., & Solem, S. (2016). *A prospective study of metacognitive and cognitive predictors of depressive symptoms*. Paper presented at the The 3rd International Conference

of Metacognitive Therapy, Milan, Italy. http://www.mctconference.no//2016/MCT%20Conference%20Abstract%20Booklet.pdf.

Hjemdal, O., Hagen, R., Solem, S., Nordahl, H., Kennair, L. E. O., Ryum, T., … Wells, A. (2017). Metacognitive therapy in major depression: An open trial of comorbid cases. *Cognitive and Behavioral Practice, 24*(3), 312–318. https://doi.org/10.1016/j.cbpra.2016.06.006.

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. *Clinical Psychology Review, 31*(1), 138–145. https://doi.org/10.1016/j.cpr.2010.08.005.

Kraft, B., Jonassen, R., Stiles, T. C., & Landrø, N. I. (2017). Dysfunctional metacognitive beliefs are associated with decreased executive control. *Frontiers in Psychology*. https://doi.org/10.3389/fpsyg.2017.00593.

Luminet, O. (2004). Assessment and measurement of rumination. In C. Papageorgiou & A. Wells (Eds.), *Rumination: Nature, theory, and treatment of negative thinking in depression* (pp. 187–215). Chichester: Wiley.

Majer, M., Ising, M., Kunzel, H., Binder, E. B., Holsboer, F., Modell, S., & Zihl, J. (2004). Impaired divided attention predicts delayed response and risk to relapse in subjects with depressive disorders. *Psychological Medicine, 34*(8), 1453–1463. https://doi.org/10.1017/S0033291704002697.

Matthews, G., & Wells, A. (2004). Rumination, depression, and metacognition: The S-REF model. In C. Papageorgiou & A. Wells (Eds.), *Depressive rumination* (pp. 125–151). West Sussex: Wiley.

Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions: Four general conclusions. *Current Directions in Psychological Science, 21*(1), 8–14. https://doi.org/10.1177/0963721411429458.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*(1), 49–100. https://doi.org/10.1006/cogp.1999.0734.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*(4), 569–582. https://doi.org/10.1037/0021-843X.100.4.569.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta earthquake. *Journal of Personality and Social Psychology, 61*(1), 115–121. https://doi.org/10.1037/0022-3514.61.1.115.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science, 3*(5), 400–424. https://doi.org/10.1111/j.1745-6924.2008.00088.x.

Papageorgiou, C., & Wells, A. (2001a). Metacognitive beliefs about rumination in recurrent major depression. *Cognitive and Behavioral Practice, 8*(2), 160–164. https://doi.org/10.1016/S1077-7229(01)80021-3.

Papageorgiou, C., & Wells, A. (2001b). Positive beliefs about depressive rumination: Development and preliminary validation of a self-report scale. *Behavior Therapy, 32*(1), 13–26. https://doi.org/10.1016/S0005-7894(01)80041-1.

Papageorgiou, C., & Wells, A. (2003). An empirical test of a clinical metacognitive model of rumination and depression. *Cognitive Therapy and Research, 27*(3), 261–273. https://doi.org/10.1023/a:1023962332399.

Papageorgiou, C., & Wells, A. (2009). A prospective test of the clinical metacognitive model of rumination and depression. *International Journal of Cognitive Therapy, 2*(2), 123–131. https://doi.org/10.1521/ijct.2009.2.2.123.

Paykel, E. S. (2008). Partial remission, residual symptoms, and relapse in depression. *Dialogues in Clinical Neuroscience, 10*(4), 431–437.

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., … 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. *Journal of Clinical Psychiatry, 59*(Suppl 20), 22–33 (**quiz 34–57**).

Snyder, H. R. (2013). Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: A meta-analysis and review. *Psychological Bulletin, 139*(1), 81. https://doi.org/10.1037/a0028727.

Solem, S., Hagen, R., Hoksnes, J. J., & Hjemdal, O. (2016). The metacognitive model of depression: An empirical test in a large Norwegian sample. *Psychiatry Research, 242*, 171–173. https://doi.org/10.1016/j.psychres.2016.05.056.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*. https://doi.org/10.1023/a:1023910315561.

Wells, A. (2011). *Metacognitive therapy for anxiety and depression*. New York: Guilford press.

Wells, A., & Matthews, G. (1996). Modelling cognition in emotional disorder: The S-REF model. *Behaviour Research and Therapy, 34*(11), 881–888. https://doi.org/10.1016/S0005-7967(96)00050-2.