

# **The Interplay between Symptoms, Rumination, Attentional Bias and Control Processes in Depression**

Brage Kraft



Clinical Neuroscience Research Group

Department of Psychology

Faculty of Social Sciences

University of Oslo

2019

© Brage Kraft, 2019

*Series of dissertations submitted to the  
Faculty of Social Sciences, University of Oslo  
No. 747*

ISSN 1564-3991

All rights reserved. No part of this publication may be reproduced or transmitted, in any form or by any means, without permission.

Cover: Hanne Baadsgaard Utigard.  
Print production: Reprintsentralen, University of Oslo.

<b>Acknowledgements</b> .....	<b>1</b>
<b>General Summary</b> .....	<b>2</b>
<b>List of Papers</b> .....	<b>3</b>
<b>List of Abbreviations</b> .....	<b>4</b>
<b>Background</b> .....	<b>5</b>
Depression .....	5
Rumination .....	8
Attentional bias in depression and rumination .....	9
Cognitive control in depression and rumination .....	10
Integrative cognitive models of depression and rumination .....	11
<b>Aims of the Study</b> .....	<b>16</b>
<b>Methods</b> .....	<b>17</b>
Design.....	17
Participants .....	18
Assessments .....	20
Procedure.....	25
Ethical Considerations.....	26
Statistical Analyses .....	26
<b>Summary of Papers</b> .....	<b>28</b>
Paper I .....	28
Paper II .....	28
Paper III.....	29
<b>Discussion</b> .....	<b>29</b>
Main Findings .....	29
Methodological Considerations.....	39
Future Directions.....	41
<b>Conclusions</b> .....	<b>46</b>
<b>References</b> .....	<b>47</b>
<b>Papers I–III</b> .....	<b>67</b>



## Acknowledgements

Several colleagues and collaborators have given contributions to this thesis. First and foremost, I would especially like to thank my supervisor Professor Nils Inge Landrø for wise guidance, as well as theoretical and practical insights. Thanks to my co-authors Rune Jonassen, PhD, Professor Alexandre Heeren, Professor Tore Stiles and Professor Catherine Harmer, for their support in different parts of the study. I would also acknowledge the collaboration with Eva Hilland, MSc, and Luigi Maglanoc, MSc, as well as the test assistants, in particular Adrian Dahl Askelund, MPhil, and Dani Beck, MSc, for their conscientious effort and flexible attitude during data collection.

Special thanks goes to my friend and colleague Vidar Ulset, cand. psychol., for positive support throughout the PhD program, co-authoring one of the papers, co-deployment of the judicious use of the 'harelab' method, and all the fun we've had! A warm thanks to Ragnhild Bø, PhD, for mentoring and practical help, especially during the early days. Thanks also to all my other colleagues at the Department of Psychology, University of Oslo.

This work was made possible by the Department of Psychology and the Norwegian Research Council. Thanks also to Diakonhjemmet Hospital, Lovisenberg Hospital and all the participants for making this thesis possible.

Eternally thanks to my parents, Solveig and Pål, for providing me with my constitutional characteristics necessary for this thesis, giving me a secure and supportive upbringing, and inspiring my career in psychology.

Finally, and most importantly, thanks to Karoline for your endless support and affection, and Elida and Olav for your smiles and laughter!

## **General Summary**

Contemporary cognitive models incorporate an information-processing approach in explaining the causes of depression. The common core in these models is that depression is caused by a vicious cycle of negative thoughts and maladaptive behaviours, and maintained by an excessive focus on the causes and consequences of the depression symptoms (i.e. rumination). Using a variety of study designs, this thesis examines three aspects of cognition assumed to underlie this process: negative attentional bias, cognitive control and metacognitions. The thesis shows that using a computerized intervention to target negative attentional bias in currently remitted depression patients leads to reduced interaction between depression and anxiety symptoms, and that this is associated with increased interest and motivation for social involvement. Furthermore, the thesis indicate that there are bidirectional links between depression symptoms, rumination, and negative beliefs about rumination (negative metacognitions) in currently remitted depression patients, suggesting that negative metacognitions have a relevant role in the feedback loop between depression and rumination. The thesis also demonstrates that there is a basic association between negative metacognitions and reduced ability to shift between mental sets, suggesting a fundamental interaction between cognitive control processes and metacognitions. In sum, the findings presented in this thesis are in line with the notion of depression as a complex interplay of several cognitive and emotional mechanisms within and between different levels of processing.

## List of Papers

- I. Kraft, B., Jonassen, R., Heeren, A., Harmer, C. H., Stiles, T., & Landrø, N. I. (in press). Attention Bias Modification in Remitted Depression is Associated with Increased Interest and Leads to Reduced Adverse Impact of Anxiety Symptoms and Negative Cognition. *Clinical Psychological Science*.
- II. Kraft, B., Jonassen, R., Ulset, V., Stiles, T., & Landrø, N. I. (2018). A Prospective Test of the Metacognitive Model of Depression in Previously Depressed Individuals. *Cognitive Therapy and Research*, doi:10.1007/s10608-018-9972-z
- III. Kraft, B., Jonassen, R., Stiles, T. C., & Landrø, N. I. (2017). Dysfunctional Metacognitive Beliefs are Associated with Decreased Executive Control. *Frontiers in Psychology*, 8, 593. doi:10.3389/fpsyg.2017.00593

## **List of Abbreviations**

ABM	Attentional Bias Modification
BAI	Beck Anxiety Inventory
BDI	Beck Depression Inventory
CANTAB	Cambridge Neuropsychological Test Automated Battery
CWIT	Color-Word Interference Test
HDRS	Hamilton Depression Rating Scale
ID/ED	Intra-Extra Dimensional Task
MCT	Metacognitive Therapy
MCQ-30	Metacognitions Questionnaire-30
MDD	Major Depressive Disorder
NBRS	Negative Beliefs about Rumination Scale
PBRS	Positive Beliefs about Rumination Scale
RRS	Ruminative Responses Scale
S-REF	Self-Regulatory Executive Function (model of emotional disorder)
SST	Stop-signal Task
SWM	Spatial Working Memory



## **Background**

Depression is often dubbed the common cold of mental disorders. This implies that it is widespread in the population and that the unfortunate can catch it. However, a burgeoning amount of research has demonstrated that both internal factors (e.g. biological, psychological) and external factors (e.g. stress) are involved in the aetiology of depression (Feliciano & Renn, 2014). In his seminal works in the 1960s, Aaron T. Beck argued that depression is caused by automatic, spontaneous and apparently uncontrollable negative thoughts about the self, the world and the future. Based on this cognitive model, Beck developed cognitive-behavioural therapy, focusing on changing patients' negative thoughts (cognitions) and facilitating more adaptive behaviour. From this perspective, the negative *content* of thought is regarded as the main driving force behind depression. Beck urged his patients to identify the negative thoughts when they felt depressed and generate alternative positive thoughts. But why do some people experience more negative thoughts than others? In the early 1990s, Susan Nolen-Hoeksema observed that not only did depressed patients think through their problems and try to solve them, they also ruminated excessively on the possible causes of their depressed mood and the consequences of being depressed. Rumination is a negative *style* of thought as opposed to negative content (Joormann & Vanderlind, 2014). Experimental and longitudinal studies followed, and confirmed that rumination was causally related to more negative thoughts and an increased risk for experiencing depression symptoms (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Since then, clinical and cognitive scientists have tried to understand the cognitive mechanisms fuelling rumination and depression, and why this maladaptive process is not interrupted. This is the scope of this thesis.

### **Depression**

Two hallmarks of depression are sadness and loss of interest/pleasure in daily activities.

However, occasionally feeling sad or having reduced interest in everyday activities is normal.

It is a natural response to the ups and downs of life. Evolutionary psychologists have noted that sadness appears to be elevated in situations associated with decreased biological fitness (e.g. loss of status, loss of resources, social rejection or death of a loved one; Nesse, 1990). Loss of interest and pleasure has been observed in defeated and trapped rodents and in humans with a perceived fall in hierarchy and reduced access to resources (Gilbert, 2006). Together, these symptoms demotivate investing time and energy for future rewards. From an evolutionary perspective, this has likely been adaptive in situations where investments in vital resources were lost, and importantly, when the person could not mitigate the negative impacts of the loss (Beck & Bredemeier, 2016).

### ***Definition***

From here on, ‘depression’ refers to the clinical diagnosis of Major Depressive Disorder (MDD). Medical diagnosis are first and foremost practical models that aid the identification of specific individuals with specific mental ‘disorders’, predict how these individuals behave, provide a platform where mental disorders can be integrated with biological, psychological and cognitive theories, and to guide treatment (Kendler, Zachar, & Craver, 2011). According to the American Psychiatric Association’s *Diagnostic Manual of Mental Disorders V*, fulfilling the diagnostic criteria for a depressive episode requires at least two weeks of: 1) feeling depressed (e.g. sad, empty) or irritable, or 2) loss of interest or pleasure in daily activities. In addition to at least one of the aforementioned symptoms, additional symptoms (i.e. disturbances in weight/appetite/sleep/activity, fatigue/energy loss, feelings of guilt/worthlessness, concentration difficulties, suicidality) must be present nearly every day. In total, five of nine symptoms must be present, represent a substantial change from the person’s baseline, and impair normal social, occupational or educational functioning.

### ***Prevalence, development and impact***

Depression is one of the most prevalent mental disorders, with an estimated lifetime prevalence around 5% (Steel et al., 2014). However, estimates range 1.5–19% based on the country (Weissman et al., 1996), with high-income countries having an increased lifetime prevalence (9.9–19.2%; Kessler & Bromet, 2013). In urban Oslo, Norway, the lifetime prevalence is 17.8% (Kringlen, Torgersen, & Cramer, 2001), and is 8.3% in rural Sogn og Fjordane, Norway (Kringlen, Torgersen, & Cramer, 2006). The mean age of onset is 32 years (Kessler et al., 2005), and a depressive episode lasts about three to four months (Kessler et al., 2003). There are a number of risk factors for first-onset depression, for example, genetic, being female, low socioeconomic status, and experiencing stressful life events (Feliciano & Renn, 2014). Depression has a dramatic impact at the individual and the social level; it is the leading cause of disability worldwide and is a major contributor to the overall global burden of disease (WHO, 2017).

### ***Recurrence***

Depression is most often a recurring disorder. Recurrence rates are as high as 85% after 15 years, with a median time to relapse of about 2.5 years (Mueller et al., 1999). Depression tends to follow a relapsing-remitting course, often with the presence of residual symptoms between discrete episodes (Burcusa & Iacono, 2007). Previous research has identified risk factors for recurrence, such as stressful life events, comorbid psychopathology and lifetime number of depressive episodes, but not sex and low socioeconomic status. However, these risk factors do not seem to have a direct causal role in predicting depression recurrence, but rather reflect an underlying vulnerability (Burcusa & Iacono, 2007). A recent systematic review and meta-synthesis concluded that there was strong evidence for three factors that increase the risk of recurrence: history of childhood maltreatment, residual depression symptoms at the end of treatment and a history of recurrence (Buckman et al., 2018). In

addition, there was also good evidence that rumination, information-processing biases and reduced cognitive control are prognostic for recurrence (Buckman et al., 2018).

### **Rumination**

There is no unified definition of rumination or a gold-standard means of measurement. In a broad review of the concept, Smith and Alloy (2009) suggested that it is ‘best characterized as a stable, negative, broadly construed way of responding to discrepancies between current status and target status’ (p. 14). The most influential theory on rumination is the response styles theory (Nolen-Hoeksema, 1991), where rumination is defined as a trait-like cognitive response to negative affect, involving repetitive thinking about the causes, consequences and symptoms of one’s negative affect. Importantly, according to this definition, individuals who are ruminating are not engaging in constructive problem-solving, but focusing their attention on the problems and feelings about them. Rumination is a causal factor in depression leading to negative cognitions (i.e. hopelessness, pessimism, self-criticism), reduced problem-solving, inhibition of instrumental behaviour, reduced social support, exacerbation of sad mood, and predicts the onset of depression (Nolen-Hoeksema et al., 2008).

### ***Brooding***

The most frequently used self-report measure of rumination, the Ruminative Responses Scale (RRS), has been criticized for having items that largely overlap with depression symptoms. To delineate rumination from depression symptoms, Treynor, Gonzalez, and Nolen-Hoeksema (2003) examined the items in a factor analysis. One depression factor emerged, in addition two other factors which the authors conceptualized as adaptive (reflective pondering) and maladaptive (brooding). Brooding taps the negative and abstract aspects of self-reflection, focusing on obstacles (e.g. ‘Why can’t I handle problems better?’). Compared to reflection, the brooding factor is more closely related to depression, for example by higher levels of depression symptoms and a recurrent pattern (Joormann, Dkane, & Gotlib, 2006).

## **Attentional bias in depression and rumination**

In neuropsychological terms, rumination can be understood as excessive attentional focus on depression symptoms. Self-reports of rumination predict an attentional bias towards negative stimuli, even when depressive symptoms are statistically controlled for (Donaldson, Lam, & Mathews, 2007). Negative attentional bias has been demonstrated in currently depressed (Gotlib, Krasnoperova, Yue, & Joormann, 2004), previously depressed (Joormann & Gotlib, 2007) and never-depressed individuals with a family history of depression (Joormann, Talbot, & Gotlib, 2007; Kujawa et al., 2011).

A negative attentional bias can be demonstrated using the dot probe task. Here, paired stimuli (e.g. one negative and one positive face) are presented, followed by one or two probes (dots) appearing in the spatial location of one of the stimuli. Participants are then required to press one of two buttons as quickly as possible to indicate the number of dots in the probe. Thus, if the participant systematically orients towards a negative emotional stimulus (i.e. has a negative attentional bias), they respond faster to probes presented at the spatial location of the negative stimulus compared to probes at the location of the positive stimulus. A meta-analysis confirmed that depressed individuals respond faster on the dot probe task when probes replace negative stimuli compared to when the probes replace positive stimuli ( $d = .52$ ; Peckham, McHugh, & Otto, 2010). Such attentional biases have also been demonstrated in relation to rumination, especially brooding (Joormann et al., 2006).

Depression is also related to other cognitive biases for negative stimuli. For example, preferential recall of negative stimuli, overgeneral memory, negative interpretations of ambiguous stimuli, and difficulties identifying subtle positive emotional expressions (Gotlib & Joormann, 2010). It has been argued that future studies should examine the interplay between such biases (Everaert, Koster, Derakshan, Id, & Koster, 2012) and how this interplay is related to rumination and depression symptoms (Everaert, Grahek, Duyck, et al., 2017).

### ***Computerized modification of negative attentional bias***

Based on cognitive bias measures, such as the dot probe task, computerized tasks that aim to induce changes to the bias in question have been developed (Hertel & Mathews, 2011). Such cognitive ‘bias modification’ appears successful in reducing cognitive biases observed in depression, and may also lead to reductions in depression symptoms (Hallion & Ruscio, 2011). However, whether (and under which circumstances) these interventions have reliable effects on depression symptoms is under debate (Cristea, Kok, & Cuijpers, 2015; Cristea, Kok, & Cuijpers, 2017; Grafton et al., 2017).

### **Cognitive control in depression and rumination**

When automatic and habitual behaviour fail to serve long-term adaptive behaviour, cognitive control processes stop what you are doing and start another course. This requires effort and is more difficult than continuing with the initial behaviour. Cognitive control refers to a cluster of top-down processes needed when concentrating or paying attention and is often used interchangeably with ‘executive functions’ or ‘executive control’ (Diamond, 2013). These functions coordinate low-level cognitive processes to guide behaviour towards a goal (Banich, 2009).

### ***Executive functions***

The executive functions consist of three inter-related abilities: 1) inhibition of dominant responses (inhibition), 2) monitoring and updating information in working memory (updating), and 3) shifting between mental sets or tasks (shifting; Miyake et al., 2000; Friedman & Miyake, 2017). These functions predict clinically important behaviours relevant to mental disorders (Miyake & Friedman, 2012). A meta-analysis comparing depressed with healthy control participants (113 studies, 7007 participants) concluded that depression is associated with broad impairments in executive control (Snyder, 2013). Depression was negatively associated with inhibition ( $d = 0.58$ ), updating ( $d = 0.57$ ) and shifting ( $d = 0.47$ ). A

recent study has demonstrated that reduced inhibition (but not shifting and updating) is related to negative attentional bias and depression symptoms (Everaert, Grahek, & Koster, 2017).

The perseverative quality of rumination, in combination with the executive deficits in depressed patients, has led to the examination of whether rumination can be explained by reduced cognitive control (Koster, De Lissnyder, Derakshan, & De Raedt, 2011; Linville, 1996; Whitmer & Gotlib, 2013). A meta-analysis of 34 studies (3066 participants) revealed a negative association between rumination and inhibition ( $r = -.23$ ) and set-shifting ( $r = -.19$ ), but not updating (Yang, Cao, Shields, Teng, & Liu, 2017). Moreover, the results suggested that depression did not moderate the associations, implying a direct relationship between rumination and reduced cognitive control. However, there does not appear to be a reliable pattern in the associations between cognitive control deficits and brooding vs. reflection (Whitmer & Gotlib, 2013; but see also Grol, Hertel, Koster, & De Raedt, 2015).

### ***Cognitive control interventions***

Researchers have begun to develop cognitive control interventions for depression. There have been a multitude of intervention programs, which show promise for reducing cognitive control impairments and depression symptoms. A recent review concluded that cognitive control training has beneficial effects, but the effect is mainly evident in populations with clear cognitive control impairments and when training is extensive (Koster, Hoorelbeke, Onraedt, Owens, & Derakshan, 2017). Paradigms that are engaging and require attentional control while ignoring task-unrelated stressful thoughts appears most promising for reducing depression symptoms, and the effect is likely mediated by reduced rumination.

### **Integrative cognitive models of depression and rumination**

The aforementioned findings represent an information-processing approach to the underlying mechanism regulating rumination in depression (Ingram, 1984, 1986). Most of the

contemporary information-processing models of depression build on Beck's cognitive theory of depression (Gotlib & Joormann, 2010; LeMoult & Gotlib, 2018). These models aim to specify the basic cognitive 'mechanics' responsible for maintaining depression symptoms.

### ***Beck's unified model of depression***

Increasing knowledge of the cognitive processes underlying depression, along with new insights from neurobiological studies and evolutionary psychology, led Beck and Bredemeier (2016) to extend the original cognitive model of depression. Here, genetic risk and early experiences contribute to biological reactivity to stress and the development of information-processing biases (e.g. negative attentional bias), which, over time, lead to the development of the negative cognitive triad about the self, the world and the future (negative self-beliefs). It is assumed that activation of negative self-beliefs further exacerbates the cognitive biases, forming a feedback loop, which in turn maintains the depression symptoms. In addition, it has been posited that negative cognitions reinforce somatic depression symptoms (i.e. energy loss, reduced appetite) through autonomic and immune response alterations.

### ***The impaired disengagement hypothesis***

In integrated cognitive neurobiological models of depression, rumination and attentional biases are specified as crucial etiological and maintaining factors linking rumination with negative cognition in depression (De Raedt & Koster, 2010; Disner, Beevers, Haigh, & Beck, 2011). It has been assumed that negative attentional bias reflects increased engagement with negative information. However, researchers have increasingly started to consider the possibility that depressive rumination reflects a difficulty in *disengaging* attention from negative information, caused by reduced attentional control (Joormann, Yoon, & Zetsche, 2007; Koster et al., 2011). Supporting this 'impaired disengagement' account of rumination, Southworth, Grafton, MacLeod, and Watkins (2017) used a dot probe task capable of distinguishing between attentional engagement and disengagement and demonstrated that



greater impairment in disengaging attention from negative stimuli was associated with self-report and in vivo assessments of brooding.

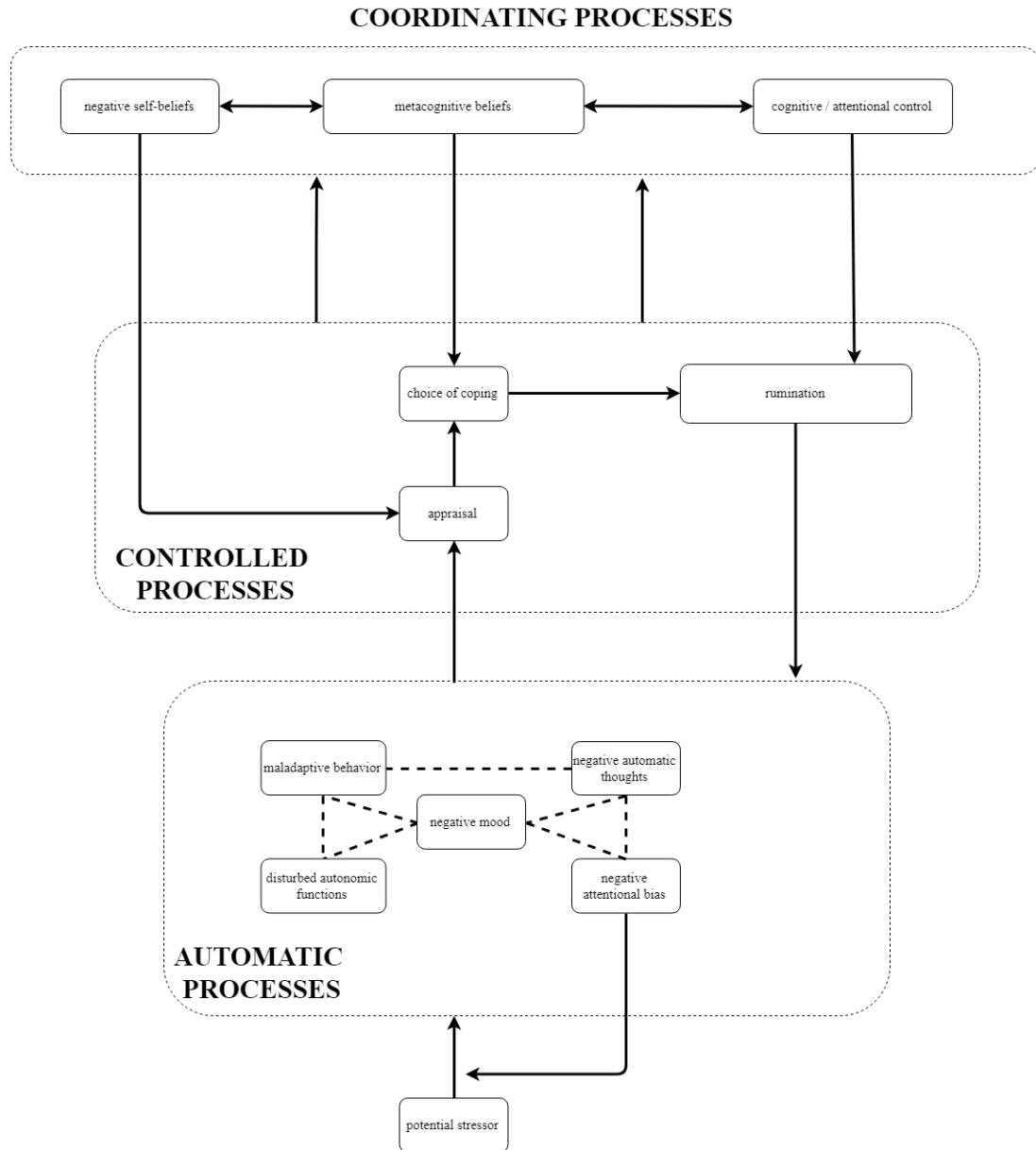
### ***The Self-Regulatory Executive Function model of emotional disorder***

Somewhat similar to the modern cognitive models of depression, the Self-Regulatory Executive Function (S-REF) model of emotional disorder (Wells & Matthews, 1996) integrates classical cognitive theory with an information-processing approach. In this model, input from low-level networks, such as negative thought intrusions, activates a coping response. The coping response (e.g. rumination) is controlled by an online process called the supervisory executive, seemingly overlapping with the concept of cognitive control. The target for this process is to reduce any discrepancy between the current status and some target status. It has been posited that this process is in itself regulated by a higher cognitive level (the 'meta'-level), by metacognitive beliefs. These provide generic procedures for coping (i.e. 'Should I ruminate about this?') and are informed by feedback from the supervisory executive (i.e. 'Are things better now; how is my mood?'). Common metacognitive beliefs in depression include thinking that ruminating is helpful (positive metacognitions), but also that rumination is dangerous and uncontrollable (negative metacognitions). Importantly, information flows back and forth between low-level processes (e.g. automatic negative thoughts), the supervisory executive (appraisal of mood/situation and initiation of rumination), and the meta-level (metacognitive beliefs). The model posits that positive and metacognitive beliefs, along with reduced cognitive control, contribute to a self-sustaining feedback loop between rumination and depression symptoms. Specifically, attentional capacity and reduced cognitive flexibility have been proposed as important regulatory factors of the influx of information to the supervisory executive, in terminating rumination, and in modifying metacognitive beliefs (Wells & Matthews, 1996). A psychological treatment method termed metacognitive therapy

(MCT) specifically aims to reduce metacognitive beliefs (Wells, 2011). MCT shows promise in reducing depression (Hagen et al., 2017).

### *Synthesis*

Contemporary cognitive models of depression emphasize the interplay between cognitive control and coordinating processes, rumination, negative attentional bias and depression symptoms. Figure 1 suggests a non-exhaustive synthesis of the aforementioned models. The overall framework is Well's three levels of cognition as described in the S-REF model, and the core processes in Beck's cognitive model, the impaired disengagement hypothesis, and the S-REF model are integrated within this framework.



**Figure 1.** A synthesis of contemporary cognitive models of depression. Potential stressors automatically activate negative thoughts, mood, etc. (symptoms). Self-beliefs guide appraisal of the situation, and triggers a coping response. Positive beliefs about rumination (metacognitions) motivate a ruminative coping style, which maintains depression symptoms and increases attentional bias for negative stimuli. Reduced cognitive control hampers attempts to disengage rumination. The coordinating processes monitor whether coping is successful. Unsuccessful coping and prolonged symptoms strengthen negative self-beliefs, negative beliefs about rumination and reduces cognitive flexibility.

## **Aims of the Study**

Using a variety of study designs, samples, measures and statistical approaches, this thesis examines some of the interactions depicted in the synthesis model (Figure 1).

In paper I, we comprehensively examine the impact of attentional bias modification (ABM) on residual depression symptoms in previously depressed patients. The study reanalyse data from a randomized controlled trial of ABM in previously depressed patients using a computational network approach. We aimed to examine whether ABM leads to changes in specific depression symptoms and in symptom–symptom interactions.

Paper II examines the interplay between depression symptoms and metacognitions using a longitudinal design. Data from a prospective follow-up study of previously depressed patients is analysed using latent growth modelling within a structural equation framework. We hypothesized that there are bidirectional effects between metacognitions, rumination and depression. Cross-sectional associations between metacognitions and a neuropsychological measure of cognitive control are also examined.

The relationship between metacognitions and cognitive control is addressed more thoroughly in paper III. Here, we examine associations between metacognitions and neuropsychological measures corresponding to the three-component model of executive functions (inhibition, updating, shifting). In line with the S-REF model, we hypothesized that metacognitions are associated with reduced cognitive control.

## Methods

### Design

#### *Paper I and II*

The data in paper I and II were obtained from a large, randomized double-blind controlled trial with follow-up assessments (Jonassen et al., 2018). The main aim of the trial was to examine the effects of computerized ABM on depression symptoms. The trial consisted of three phases. Phase one consisted of recruitment, pre-screening, enrolment in the study, assessment of various demographic and clinical variables (baseline) and random allocation to ABM intervention ('training') or a closely matched control condition. Phase two involved two weeks of ABM. In phase three, after the completion of ABM, participants returned for four assessments during the next 12 months (T1–T4).

Paper I examines the effect of ABM on depression symptoms from baseline to T1. The study design has several major methodological strengths. The control group provides a standard to which the ABM effect can be compared. Randomly allocating participants into each condition minimizes group differences in measured and unmeasured variables (confounding variables). The double-blind allocation reduces bias in both participants and assessors for potential expectancy effects.

Paper II examines the bidirectional associations between metacognitions, rumination and depression symptoms (baseline–T4). The longitudinal follow-up phase provides temporal control, which may be indicative of possible causal relationships between variables not subject to experimental manipulation.

#### *Paper III*

Data in paper III were obtained from a cross-sectional study aimed to examine cognitive control and brain function in subjects with MDD and in healthy subjects.

## **Participants**

### ***Paper I and II***

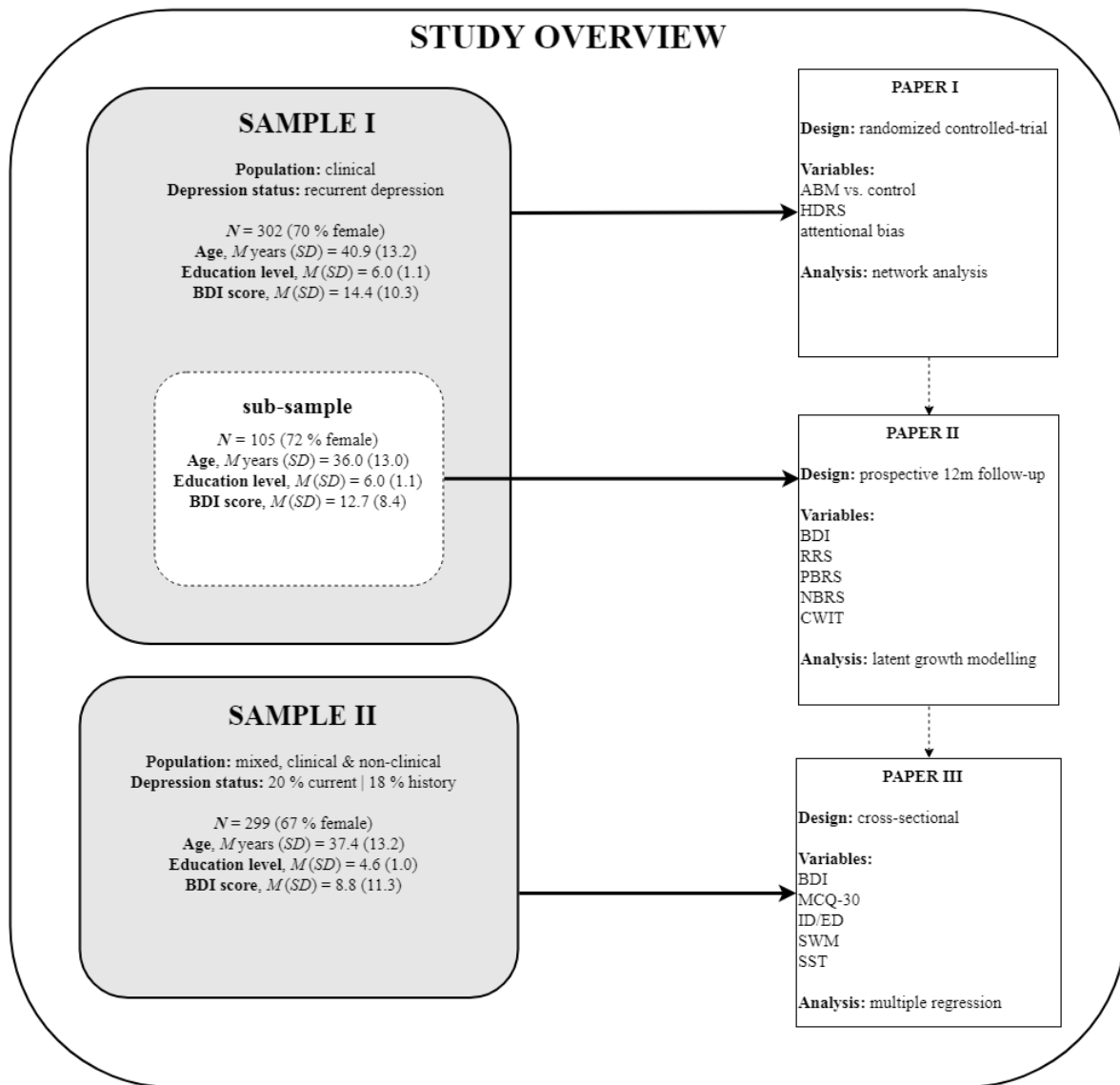
Paper I included 302 patients with a history of depression that were mainly recruited from an outpatient psychiatric clinic in the Department of Psychiatry, Diakonhjemmet Hospital, Oslo. Participants were also recruited from other clinical sites by local advertisements and via social media. The inclusion criteria were the presence of a remitted MDD, age 18–65 years, and fluency in Norwegian. The exclusion criteria were presence of current or former neurological disorder, substance use disorder, attention-deficit disorder, head trauma, psychosis, or bipolar disorder. The majority of the participants in this sample were women (70%) with a mean age of 40.9 years ( $SD = 13.2$ ); 82.7% had completed a bachelor's degree or higher. The mean score on the Beck Depression Inventory II (BDI; Beck, Steer, & Brown, 1996) was 14.4 ( $SD = 10.3$ ).

In paper II, the sample consisted of 105 patients who had completed ABM and returned for follow-up assessments in the next 12 months ('sub-sample' in Figure 2). This sub-sample had a recurrent pattern of MDD ( $\geq 2$  depressive episodes) and had completed additional measures relevant for paper II. Of the participants, 72% were female; the mean age was 36.0 years ( $SD = 13.0$ ), and 82.7% had completed a bachelor's degree or higher. The mean BDI score was 12.7 ( $SD = 8.4$ ).

### ***Paper III***

Paper III included 299 participants recruited through newspaper advertisements and posters and from outpatient psychiatric clinics in Norway. The inclusion criteria were age 18–65 years and fluency in Norwegian. The exclusion criteria were use of medication other than serotonin-specific/serotonin–norepinephrine reuptake inhibitors, history of neurological disorders, bipolar disorder, psychosis and present drug and/or alcohol abuse. Of the

participants, 67% were female; the mean age was 37.4 years ( $SD = 13.1$ ), and 61.3% had completed a bachelor's degree or higher. The mean BDI score was 8.8 ( $SD = 11.3$ ).



**Figure 2.** Study overview. *Note.* Education level = International Standard Classification of Education; BDI = Beck Depression Inventory II; ABM = Attentional Bias Modification; HDRS = Hamilton Depression Rating Scale; RRS = Ruminative Responses Scale; PBRs = Positive Beliefs about Rumination Scale; NBRs = Negative Beliefs about Rumination Scale; CWIT = Color-Word Interference Test; MCQ-30 = Metacognitions Questionnaire-30; ID/ED = Intra-Extra Dimensional Task; SWM = Spatial Working Memory Task; SST = Stop-signal Task.

## **Assessments**

### ***Diagnostic***

#### *Paper I and II*

Clinical psychologists and trained administrators with a psychology education assessed the presence of mental disorders using the *Mini-International Neuropsychiatric Interview* (Sheehan et al., 1998). Any uncertainties regarding diagnosis were discussed in the research team, which included a specialist in clinical psychology.

#### *Paper III*

Trained psychologists evaluated mental disorders and personality disorders using the *Structured Clinical Interviews for The Diagnostic Manual of Mental Disorders IV criteria I and II* (First & Gibbon, 2004). A specialist in clinical psychology validated all assessments, which had been audio-taped.

### ***Symptoms***

#### *Beck Depression Inventory II*

The BDI is a self-report measurement of depression symptoms (Beck et al., 1996). The BDI consists of 21 questions on common depression symptoms scored on a scale of 0–3. Higher total scores indicate more severe depression symptoms. Good psychometric properties have been documented (Dozois, Dobson, & Ahnberg, 1998).

#### *Hamilton Depression Rating Scale*

The Hamilton Depression Rating Scale (HDRS) is a clinician-rated assessment of depression (Hamilton, 1960). The HDRS contains 17 items; most are scored on a scale of 0–4 (some on a scale of 0–2). The scale has good psychometric properties (Trajković et al., 2011).



### *Beck Anxiety Inventory*

Beck Anxiety Inventory (BAI) is a self-report questionnaire of anxiety symptoms (Beck, Epstein, Brown, & Steer, 1988). The BAI consists of 21 items scored on a scale of 0–3. Higher total scores indicate more anxiety symptoms. Good psychometric properties have been documented (Beck et al., 1988).

### *Rumination*

The RRS is a self-report questionnaire measuring the tendency to ruminate in response to depressed mood (Nolen-Hoeksema, 1991). The scale consists of 22 items scored on a scale of 1–4. A higher total score reflects a stronger tendency to ruminate. The scale has good psychometric properties (Treynor et al., 2003). Factor analysis of the RRS has resulted in three factors: brooding, reflective pondering, and one factor mainly reflecting the presence of depression symptoms (Treynor et al., 2003).

### *Metacognitive beliefs (metacognitions)*

#### *The Positive and Negative Beliefs about Rumination*

Positive beliefs about rumination were assessed using the Positive Beliefs about Rumination Scale (PBRS; Papageorgiou & Wells, 2001b). This is a nine-item scale assessing beliefs about the benefits of rumination (e.g. ‘I need to ruminate about my problems to find the causes of my depression’). Each item is scored on a scale of 1–4. Higher scores reflect positive beliefs about rumination. The scale has acceptable psychometric properties (Luminet, 2003).

Negative metacognitive beliefs were assessed using the Negative Beliefs about Rumination Scale (NBRS; Papageorgiou & Wells, 2001a). This scale assess beliefs about the uncontrollability and harm of rumination (e.g. ‘Rumination about my problems is uncontrollable’), as well as the interpersonal consequences of rumination (e.g. ‘People will reject me if I ruminate’). Each item is scored on a scale of 1–4. Higher scores reflect negative

beliefs about rumination. The scale consists of two subscales (uncontrollability/harm, interpersonal consequences), both with acceptable psychometric properties (Luminet, 2003).

### *Metacognitions Questionnaire-30*

The Metacognitions Questionnaire-30 (MCQ-30) is a 30-item questionnaire that assesses metacognitive beliefs about worry (Wells & Cartwright-Hatton, 2004). Although worry is considered the core process of anxiety, both worry and rumination are conceived as similar cognitive processes described in different research contexts (Watkins, Moulds, & Mackintosh, 2005). Both can be conceptualized collectively as persistent negative thinking (Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014). Moreover, individuals with a history of depression often have a comorbid anxiety disorder (Brown, Campbell, Lehman, Grisham, & Mancill, 2001) and tend to worry about relapsing (Sariso et al., 2014; Spada, Nikčević, Moneta, & Wells, 2008; Wells & Cartwright-Hatton, 2004). Metacognitive beliefs about worry have also been proposed as a promising vulnerability marker of depressive relapse (Halvorsen et al., 2015). Thus, this questionnaire is also relevant in depression research.

MCQ-30 items are answered on a Likert response scale from 1 to 4. The items generate five subscales: positive beliefs about worry (believing that perseverative thinking is useful), negative beliefs about the uncontrollability and danger of worry (believing that perseverative thinking is uncontrollable and dangerous), cognitive confidence (in attention and memory), beliefs about the need to control thoughts (whether certain thoughts should be suppressed), and cognitive self-consciousness (the tendency to monitor one's thoughts and focus attention inwards). The scale has acceptable psychometric properties (Spada, Mohiyeddini, & Wells, 2008; Wells & Cartwright-Hatton, 2004).

## *Cognitive assessments*

### *Dot probe task*

Attentional bias was measured using a standard computerized visual probe procedure (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002) presented on laptop computers. Paired images of faces (the stimuli) were presented, followed by one or two dots (a probe), which appeared behind one of the stimuli. Participants were asked to press one of two buttons as quickly as possible (while also being correct) to indicate the number of dots in the probe. No other instruction was given. The stimuli were pictures of emotional faces (Karolinska Directed Emotional Faces; Lundqvist, Flykt, & Öhman, 1998) of three valences: positive, neutral or negative (angry and fearful). The task comprised 96 trials with equal numbers of the three stimulus pair types. There were equal numbers of trials in which the stimuli were randomly presented for 500 or 1000 milliseconds before the probe was displayed. Stimuli from two valences were displayed in each trial in one of the following pairing types: positive–neutral, positive–negative and negative–neutral. The probe was located behind the positive stimulus in half of the trials, and behind the negative stimulus in the other half of the trials. We calculated a total attentional bias score, and three valence-specific attentional bias scores based on the difference in reaction between trials in which the probe replaced the relatively more: 1) positive face vs. negative face, 2) positive face vs. neutral face, 3) negative face vs. neutral face.

### *The Color-Word Interference Test*

Two tasks from the Color-Word Interference Test (CWIT; Delis, Kaplan, & Kramer, 2001) are relevant to this study. In the inhibition task, the subject is required to name the printed colour of colour word names. Longer completion times reflect reduced ability to inhibit pre-potent responses (i.e. reading the word). In the inhibition/switching task, the subject names the printed colour but is also occasionally required to switch to the non-inhibitory response

(reading the word). Longer completion times reflect reduced ability to inhibit pre-potent responses and switch to another response. These tasks are especially common in the depression literature (Miyake et al., 2000; Snyder, 2013).

#### *Stop-signal Task*

The Stop-signal Task (SST) is a computerized task from the Cambridge Neuropsychological Test Automated Battery (CANTAB, 2009). The SST requires the subject to override a pre-potent go-response when presented with an infrequent stop signal (a beep). Inhibitory efficiency is operationalized as a stop-signal reaction time, which is estimated through the automatic adjustment of the delay between the go stimulus and the stop signal. A higher stop-signal reaction time represents reduced inhibitory control (Logan, Schachar, & Tannock, 1997).

#### *The Intra-Extra Dimensional Task*

The Intra-Extra Dimensional Task (ID/ED) requires participants to pay attention to different examples within a stimulus dimension and shift attention from one set of stimuli to a new, formerly unimportant set of stimuli across nine stages. Shifting ability is operationalized as total errors adjusted for whether the entire task is completed. A high total error adjusted score represents reduced shifting ability (Kaplan et al., 2006).

#### *The Spatial Working Memory Task*

The Spatial Working Memory Task (SWM) requires participants to search through several boxes to locate tokens. A token that has been located will not reappear in the same box during that same trial. Accuracy of working memory is operationalized as the between-trial errors score, which is calculated when the subject searches for a token in a box where a token had been found in a previous trial. High between-trial error scores represent working memory failures (Owen, Downes, Sahakian, Polkey, & Robbins, 1990).

### ***Attentional bias modification***

The ABM is a modified, validated visual dot probe procedure (Browning, Holmes, Charles, Cowen, & Harmer, 2012). The ‘training task’ was in every way identical to the dot probe task used to measure attentional bias, except that probes were located behind positive (valid) emotional stimuli in 87% of the trials. Thus, when completing the task, participants should implicitly learn to deploy their attention towards positive stimuli, and in this way develop a relatively more positive attentional bias. In the control condition, the probe was located behind the positive (valid) stimuli in 50% of the trials.

### **Procedure**

#### ***Paper I and II***

The study protocol was retrospectively registered at [clinicaltrials.gov](https://clinicaltrials.gov) (NCT02648165). Potential participants were pre-screened by phone according to the exclusion criteria prior to in-person formal clinical evaluation and study enrolment. Participants were informed that the study aimed at examining ‘attention focus, how this changes over time and how this is related to mood and depression symptoms’ (the specific rationale underlying ABM was not explained). A trained administrator blinded to the condition allocation assessed depression symptoms and demonstrated ABM. The participants were then instructed to perform the task at home twice a day for two weeks (28 sessions in total), before they returned for assessments of depression symptoms and other clinical variables immediately after ABM (T1), as well as one month, six months, and 12 months after ABM (T2–T4).

#### ***Paper III***

After recruitment, participants were clinically evaluated, filled out self-report measures of depression symptoms and other clinical variables, and completed a full CANTAB assessment in one session. The participants were required to be medication fasting on the day of the assessment.

## **Ethical Considerations**

The studies were conducted in compliance with the Helsinki Declaration and approved by the Regional committees for medical and health research ethics (Paper I-II: REK South East 2014/217; Paper III: REK North t6/2006). Participants were provided with written and oral description of the studies, and informed that they could withdraw from the study at any time, without any explanation, and have their personal data deleted. Participants were given a short verbal summary of their test results immediately after assessment. In cases where the participant requested (or clearly needed) advice regarding their mental health, it was provided by a clinical psychologist. Participants provided written informed consent and all data were collected and stored according to required standards.

## **Statistical Analyses**

### ***Paper I***

We used network analysis (Borsboom, 2017) to characterize symptom changes following ABM. Symptom items from the HDRS were graphically visualized as nodes, while between-symptoms associations were visualized as edges (lines) between nodes. First, a baseline network was estimated for the whole sample, where edges represented the partial correlations between each of the 17 HDRS items before ABM. Thus, the edges represented conditional independence relationships between symptoms when controlling for the effects of all other nodes (Epskamp, Borsboom, & Fried, 2017). The theoretical underlying assumption for this is that each depressive symptom has independent causal powers that influence other symptoms (e.g. insomnia causes fatigue), and is not only assumed to be a passive indicator of an underlying disease ('the depression', Borsboom & Cramer, 2013). After estimating the baseline network, we estimated two networks based on the symptom change scores from baseline to post-ABM: 1) a symptom change network for the whole sample, including a node representing the ABM condition, and 2) symptom change networks for the training and

control group separately. Finally, we estimated networks separately for the training and control groups, where nodes represented symptom scores post-ABM. We used the *R* package *qgraph* (Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012) to estimate the symptom networks, and the *R* packages *bootnet* (Epskamp et al., 2017) and *NetworkComparisonTest* (van Borkulo, 2016) to assess statistical reliability.

### ***Paper II***

We used latent growth modelling within a structural equation modelling framework to examine the bidirectional longitudinal effects between metacognitions, rumination and depression. First, we examined the effects of positive metacognitions (PBRS), negative metacognitions (NBRS) and brooding (RRS) at baseline on depression levels and individual symptom trajectories (baseline–T4). Second, we examined the feedback effects of depression levels and symptom trajectories on positive metacognitions, negative metacognitions and brooding at T4. Finally, we examined bivariate correlations between metacognitions and cognitive control variables (inhibition and inhibition/switching). Statistical analyses were performed using *Mplus 7.4* and *IBM SPSS 24*.

### ***Paper III***

First, we explored the bivariate correlations between the five MCQ-30 subscales and test variables from SST (inhibition), ID/ED (shifting) and SWM (updating). Any statistically significant correlations were examined further using hierarchical multiple linear regression analyses, with the MCQ-30 subscale as the dependent variable and the test measure as the predictor variable. The control variables were age, education level, general cognitive functioning, current symptoms of depression (BDI) and anxiety (BAI) and history of depression. Control variables were entered in step 1, and the test variable in step 2. Statistical analyses were performed in *IBM SPSS 22*.

## Summary of Papers

### **Paper I**

The clinical effects of ABM for depression have commonly been evaluated based on sum scores from depression rating scales such as the HDRS or BDI. However, it is likely that ABM is associated with changes in specific depression symptoms, and that aggregating symptoms into a sum score masks this effect, which may potentially conceal important insights (Fried & Nesse, 2015). The paper reconsiders the model of depression and how the outcome of ABM is assessed, and re-examines data demonstrating the effect of ABM in residual depression (Jonassen et al., 2018) at the symptom-level, using recent innovations in theoretical and computational network analysis. ABM was associated with improvements in interest, which were in turn associated with improvements in other depression symptoms. Although global network strength was unchanged following ABM, comparison of symptom change in the ABM and control groups suggested that ABM led to a reduction of the association between anxiety, depressed mood and guilt. The findings suggest that reductions in depression symptoms following ABM may have been set in motion by increased interest and involvement in everyday activities, leading to a reduction of the adverse impact of anxiety and negative cognition. Paper I provides new knowledge on the interplay between specific depression symptoms and negative attentional bias, and may inform further development of ABM.

### **Paper II**

According to the S-REF model, depression symptoms are maintained because patients hold beliefs that motivate rumination (positive metacognitive beliefs) and beliefs that demotivate the stopping of rumination (negative metacognitive beliefs). The model also posits that worsening of depression symptoms reinforces negative metacognitive beliefs (a feedback effect). However, no studies have confirmed the prospective effects of metacognitive beliefs



on depression or the hypothesized feedback effect in a clinical population. We found that positive metacognitive beliefs were associated with rumination, while negative metacognitive beliefs and rumination predicted higher depression levels but not symptom worsening. Depression levels and symptom worsening predicted positive and negative metacognitions, as well as rumination. The study also examines the hypothesized relationship between metacognitive beliefs and cognitive control (inhibition and inhibition/switching). We found no association between metacognitions and cognitive control. Paper II lends partial support for the metacognitive model, but raises questions on the relevance of metacognitions as a proximal vulnerability marker for symptom recurrence.

### **Paper III**

Metacognition is conceptually closely related to cognitive control (Fernandez-Duque, Baird, & Posner, 2000), and cognitive control has been proposed as important factors in the modification of metacognitive beliefs (Wells & Matthews, 1996). However, whether metacognitive beliefs and objective measures of executive control are empirically related has not been studied systematically. Controlling for current symptoms and history of depression, decreased ability to shift between mental sets was associated with increased negative beliefs about the uncontrollability and danger of worry and beliefs about the need to control thoughts. Paper III demonstrates that there is a basic association between certain metacognitive beliefs and cognitive control.

## **Discussion**

### **Main Findings**

#### ***ABM reduces the interplay between depression symptoms***

Consistent with the existing depression literature (e.g. Boschloo, van Borkulo, Borsboom, & Schoevers, 2016; Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2014; Fried,

Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016), paper I shows that the most central symptoms before ABM are depressed mood, interest, guilt and anxiety. These symptoms are, according to the network theory of mental disorders (Borsboom, 2017), assumed to be responsible for the maintenance of other depression symptoms. It has been hypothesized that worsening of these symptoms may trigger a downstream cascade of worsening in other depression symptoms (Borsboom & Cramer, 2013; Valente, 2012). We found that interplay between these symptoms were reduced after ABM, and that improvements in depression symptoms were associated with improvements in interest and motivation for social involvement. This provides an important basis for formulating new hypotheses on how ABM may alter depression symptoms.

The ABM procedure aims to render patients more focused on positive social stimuli. This mechanism may have been set in motion by implicit relearning of emotional associations, where ambiguous events or stimuli are perceived more positively and thus increase patients' motivation to engage with their social environment (Harmer, Duman, & Cowen, 2017). Hence, when participants' attention is nudged towards the positive aspects of everyday situations (e.g. a smiling face or an encouraging comment), this may increase pro-social interaction, which in turn may increase positive feedback and ultimately reinforces approach behaviour towards social interaction (Fox, 2005). The association between ABM and improvements in interest and motivation for social interaction may reflect the endpoint of this mechanism.

An alternative interpretation is that interest and motivation for social interaction is a moderator of the ABM effect. This explanation is in line with Shiroma, Thuras, Johns, and Lim (2014) demonstrating that early changes in emotional processing of faces predict remission and response to antidepressants, but only when controlling for perceived social support.

### *ABM and the positive affective system*

The association between positive affectivity (i.e. interest) and reduced interplay between depression symptoms invites the possibility that the ABM-related improvements in depression reflect a remediation of the positive affective system. Importantly, positive affect does not merely reflect the opposite end of negative affect (Cacioppo & Berntson, 1999). Positive and negative affect are regarded as distinct dimensions reflecting the broader biobehavioural systems of approach and withdrawal (Watson, Wiese, Vaidya, & Tellegen, 1999). Together, these affective systems influence cognitive appraisals of stimuli and inform decisions regarding appropriate actions.

Positive affect reflects a person's level of pleasurable engagement with the environment (e.g. high energy levels, mental alertness, interest, determination), while negative affect reflects subjective distress (e.g. anxiety and sadness). Depression is characterized by low positive affect and high negative affect (Watson, Clark, & Tellegen, 1988).

Reduced interest and motivation for previously pleasurable activities (i.e. positive affect) is a core symptom of depression. This symptom overlaps with what has been termed 'motivational anhedonia', which refers to diminished motivation or drive to pursue rewards (Treadway & Zald, 2011). Anhedonia is associated with impairments in the reward-related processes, specifically approach motivation, reward anticipation and reward learning (Dillon et al., 2014).

Compared to other depression symptoms, interest and anhedonia appear especially important for social functioning (Fried & Nesse, 2014). It has been demonstrated that naturally occurring life stress impacts more strongly on interest/anhedonia than other depression symptoms (Fried, Nesse, Guille, & Sen, 2015). The evolutionary function of these symptoms

may be to conserve resources after a perceived loss or a hopeless situation (Beck & Bredemeier, 2016; Nesse, 2000).

Given that ABM directs attention towards the positive aspects of social situations, we therefore speculate that patients become more likely reach out to and elicit positive feedback from their environment. This process may in turn reinforce approach behaviour, increase anticipation of social rewards and result in more positive affect.

Although assumed to be related to the negative affective system, rumination may also play a pivotal role in maintaining anhedonia. This is because continued rumination provides a stream of thought that convinces patients that action is futile, that they cannot overcome their problems regardless, and justifies withdrawal from previously pleasurable activities (Nolen-Hoeksema et al., 2008). A negative attentional bias could be considered the manifestation of this process at the automatic level. Accordingly, a negative attentional bias may function to focus attention on stimuli congruent with the idea that one should remain inactive. However, whether there is a specific link between anhedonia and negative attentional bias (and the potential mediating role of rumination) has not been examined.

#### *ABM and the negative affective system*

Interestingly, our study found reduced interplay between anxiety and other depression symptoms after ABM. Moreover, improvements in anxiety were associated with attentional bias change from pre- to post-ABM. This may be explained by the fact that the current ABM procedure actually involves shifting attention in the context of threat-related stimuli (angry and fearful faces). This invites the possibility that attenuating vigilance for potential threats may have mediated improvements in depression.

Although threat vigilance is assumed to originate from the negative affective system (Cuthbert, 2015; Dillon et al., 2014), patients with depression do not appear to demonstrate

heightened threat vigilance (Armstrong & Olatunji, 2012; Mogg & Bradley, 2005).

Heightened threat vigilance has first and foremost been established as a causal process in anxiety (Mathews, Mackintosh, & Fulcher, 1997). However, it is important to note that the current study did not exclude patients with anxiety disorders. In fact, a majority of the sample fulfilled the diagnostic criteria for a comorbid mental disorder (often an anxiety disorder), and reported anxiety symptom levels bordering or exceeding clinical cut-off. Threat vigilance may therefore be a relevant target mechanism in this specific sample.

Moreover, the possibility of a depressive relapse is likely to be a very threatening prospect for patients who have experienced repeated episodes of depression. Hypervigilance towards negative social cues and the resulting increase in negative affect may easily trigger rumination in these patients, as this may ‘forecast’ a depressive relapse (Lyubomirsky & Tkach, 2004).

The S-REF model refers to this process as threat monitoring (Mathews & Wells, 2004; Wells & Mathews, 1996). Threat monitoring is motivated by negative metacognitive beliefs about rumination as damaging to the individual (Papageorgiou & Wells, 2001a). Given this, and that ABM might buffer against depression symptoms through reduced threat monitoring, we speculate whether negative metacognitions could moderate the ABM effect.

Research in affective science suggests that there is interplay between positive and negative affectivity. For example, the presence of acute threats negatively impacts reward processing, leading to blunted behavioural and neural responses to pleasant activities and reward tasks (Dillon et al., 2014). Our finding, that ABM is associated with changes in both anxiety- and anhedonia-related processes, may therefore also be ascribed to the seemingly fundamental interaction between the positive and the negative affective systems.

Compared to changes in anxiety, ABM resulted in less pronounced changes related to depressed mood and guilt. Depressed mood (e.g. sadness) and guilt reflect the negative

affective system. Loss of resources (for example a relationship, status, or income) appears to trigger these emotions (Wolpert, 2008), as well as rumination (Nolen-Hoeksema et al., 2008). These are therefore considered loss-related processes (Owens & Gibb, 2017; Woody & Gibb, 2015). In the present study, guilt actually refers to ‘self-reproach and ideas of guilt or rumination over past errors’, which is similar to brooding rumination (Treyner et al., 2003). It may therefore be the case that reductions in the interplay of depressed mood and guilt reflect a reduced impact of rumination in the context of other depression symptoms. However, given that these symptom changes were only secondary to improvements in interest, we speculate whether ABM does not directly remediate loss-related processes.

### *Clinical implications*

The current ABM procedure may prove more efficient for problems originating from the positive affective system. The effect of ABM may increase if patients are encouraged to activate the positive affective system between training sessions (e.g. approach social situations). In addition, ABM may be more effective in patients where the interplay between interest and anxiety symptoms dominates the symptom network. Training in the context of negative mood-related stimuli (e.g. sad faces) instead may result in more pronounced changes involving the negative affective system.

### ***The interplay between metacognitions, rumination and depression***

Cross-sectional studies have shown support for the metacognitive model in depressed and non-depressed individuals (Papageorgiou & Wells, 2003; Solem, Hagen, Hoksnes, & Hjemdal, 2016). However, only one previous study has used a prospective design to investigate the metacognitive model, but only in a non-clinical sample (Papageorgiou & Wells, 2009). Negative metacognitions are increased in individuals with residual depression symptoms, and has therefore been proposed as a vulnerability factor for depressive relapse (Halvorsen et al., 2015). Thus, according to the metacognitive model, negative

metacognitions are assumed to increase the probability of symptom recurrence in previously depressed individuals. Moreover, it is proposed that prolonged experiences of depression symptoms reinforce metacognitions (Wells & Matthews, 1996).

The results presented in paper II partially support the hypothesized causal effects of metacognitions. The results indicated bidirectional links between rumination, metacognitions and depression symptoms over a one-year period in currently remitted depression patients. Positive metacognitions predicted depression levels, and rumination accounted for this effect. Rumination and negative metacognitions contributed independently to depression levels. However, contrary to the metacognitive model, metacognitions did not predict symptom trajectories. Negative metacognitions therefore only appear to explain the general susceptibility to experiencing depression symptoms. That is, individuals with lower levels of metacognitions appear to be as vulnerable to symptom recurrence as individuals with higher levels of metacognitions.

Thus, the findings suggest that metacognitions do not predict symptom recurrence in previously depressed individual. However, this relationship could be different in a treatment setting. It is also important to note that the present study was limited to one year. It is possible that metacognitions convey a risk for symptom recurrence in the long-term.

#### *Feedback effects from depression symptoms to metacognitions*

We found evidence for the hypothesized feedback effect of depression on metacognitions, which has not been examined in previous studies. The results showed that a worsening depression trajectory predicted levels of positive metacognition, negative metacognitions and rumination. Thus, patients with worsening residual depression symptoms continue to ruminate and continue to believe that rumination is uncontrollable and harmful to their mental health

and social relationships. Our study suggests that negative metacognitions have a relevant role in the positive feedback loop between depression and rumination.

Unfortunately, we were unable to delineate in the present study the exact mechanism behind the feedback effect. We speculate whether metacognitions may foster a self-reinforcing ‘perceptual sampling bias’ (Smith, Alkozei, Killgore, & Lane, 2018) that promotes social avoidance and attentional focus towards stimuli that is in line with a person’s depressive expectations. Thus, even if the patient’s situation has many positive aspects, negative metacognitions promote behaviours (e.g. rumination and threat monitoring) that exaggerate the ability to learn from negative outcomes and reduce the ability to learn from positive outcomes. These maladaptive behaviours are likely to maintain negative trends in the patient’s life and provide further evidence in favour of the patient’s negative self-beliefs and metacognitions.

Future studies should assess metacognitions, rumination and depression at several time points, along with MCT. This will improve the statistical modelling of the lagged associations and enable the analysis of bidirectionality, stability and change in these important variables. Such multi-wave designs can clarify whether reductions in metacognitions leads to reduced depression symptoms, and to what extent this will feed back and reduce metacognitions further.

### *Clinical implications*

Patients that experience worsening depression symptoms are likely to have increased levels of metacognitions and rumination. Identifying and discussing this vicious cycle may be helpful for patients with a high risk for relapse. However, proximal relapse prevention should probably go beyond the reduction of metacognitions and rumination. For example, remediating social functioning (Paykel, 2008) and increasing positive affect (Craske, Meuret,



Ritz, Treanor, & Dour, 2016) could be a more effective treatment focus for patients with a currently remitted recurrent depression.

### ***Possible interplay between negative metacognitions and cognitive control***

The S-REF model assumes a basic interplay between metacognitions and cognitive control. Reduced cognitive control is posited to exacerbate the impact of metacognitions and rumination on depression symptoms (Matthews & Wells, 2004; Papageorgiou & Wells, 2003). Moreover, it has been proposed that cognitive control is important in the modification of metacognitions (Wells & Matthews, 1996). Previous studies have demonstrated that metacognitions are associated with self-reports of decreased ability to shift and focus attention (Spada, Georgiou, & Wells, 2010). However, self-reports of cognitive control and performance on objective neuropsychological tests show weak or no correlation (Løvstad et al., 2016). Thus, the present study set out to examine the relationship between metacognitions and cognitive control based on objective neuropsychological tests.

Paper III shows that negative metacognitions are associated with decreased ability to shift between mental sets. The association persisted after controlling for age, education level, general cognitive function, and depression and anxiety symptoms, suggesting a basic relationship between shifting ability and negative metacognitions.

Individuals who have high levels of negative metacognitions may therefore struggle to disengage from rumination because of reduced shifting ability. However, the relationship could also be in the opposite direction, as failing to successfully stop rumination may lead to the conclusion that one is not very good at it, which may reinforce metacognitions (Fernandez-Duque et al., 2000). This is referred to as ‘belief elaboration’ in the S-REF model (Wells & Matthews, 1996). The cross-sectional design of paper III precludes conclusions on

the direction of the relationship. In any case, it is clear that future research on this matter is needed to delineate the relationship between cognitive control and metacognitions.

On the contrary, paper II showed no association between metacognitions and the combined inhibition/switching task. This task requires the participant to stop a pre-potent response (inhibition), as well as shift to an alternative response (inhibition/switching). The conflicting results could be explained by the tasks tapping different aspects of executive functions, or differences in the sample characteristics. In general, correlations between different executive control tasks tend to be low because of reliability differences, task impurity problems and strategy use (Friedman & Miyake, 2017). Therefore, there is a great need for replication of the findings in paper III using the same measures.

Cognitive control could play a special role as depression symptoms and metacognitions fluctuate over time. For example, cognitive control may moderate the effects of metacognitions and rumination on depression trajectories. Feedback effects from depression to metacognitions could be mediated by changes in cognitive control. Successful modification of attentional biases has also been linked to increased cognitive control (Heeren, De Raedt, Koster, & Philippot, 2013). Thus, future studies of these processes should examine the temporal interplay between metacognitions, cognitive control and negative attentional bias.

#### *Clinical implications*

Effective MCT (which aim to weaken metacognitions and stop rumination) may increase shifting ability. However, the opposite may also be true; cognitive training that targets shifting ability might increase the ability to stop rumination and weaken metacognitions, and in turn increase the effectiveness of MCT. Individuals with low executive control might benefit from training of shifting ability before they attempt to stop rumination. On the other hand, individuals with better executive control might not require explicit training at all. It might also

be the case that MCT requires a minimum of executive control, and that other treatments are more appropriate for individuals with low executive control.

### **Methodological Considerations**

The strengths of the present study are as follows: participants were mostly recruited from a clinical population, sample sizes were large, we used designs that can address causality, and used well-validated and objective measures of cognitive control. However, we cannot overlook the study limitations: The samples varied in terms of the inclusion and exclusion criteria, and consisted of individuals with mixed symptomatology, often reporting other mental health problems than depression. There were also large variations within each sample regarding depression status (ongoing, remission, or never-depressed), number of depressive episodes, symptom severity, and other clinically relevant variables. This provides a rather broad approach to the phenomenon of depression, but may preclude claims that are specific to the clinical setting. The generalizability of the overall findings in this thesis are questionable. It is also likely that the quality and strength of interplay between the relevant processes vary as a result of depression status.

A majority of the participants in this study were women (approximately 70%), which reflects the increased incidence risk for depression observed in women (Albert, 2015). The study is therefore not unreasonably skewed because of under-sampling of men. Nonetheless, the demonstrated interplay between variables may, to a greater extent, reflect processes that are more relevant for women. Future studies should consider examining whether these processes are different between men and women.

The primary outcome measure was self-reports of depression symptoms. Paper I and II report results using only one depression inventory, even though the participants reported depression symptoms using two inventories (BDI and HDRS). Reporting results using both scales in both

papers would have addressed questions regarding the findings' reliability more appropriately. A more general problem with self-reports is the tendency for participants to report inaccurately (response biases). Other unquestionably very important aspects of depression, such as relational or vocational functioning, were not examined at all.

Symptom associations in the network analyses of ABM were calculated using cross-sectional data, precluding strong inference regarding cause–effect relationships among the variables (Maurage, Heeren, & Pesenti, 2013). These associations represent both within- and between-subjects effects that cannot be disentangled, and the network trajectory over time may vary across individuals. The statistical adequacy of considering the ABM condition within a network of continuous measures has not been established. Even though ABM was associated with improved interest, it was not associated with changes in attentional bias. Also mind that the dot probe task exhibits poor psychometric properties (McNally, 2018). Symptom change networks were estimated based on individual difference scores, assuming that there would be no symptom changes in the absence of intervention. An alternative model would be to assume that symptom scores post-ABM are a linear function of the symptom scores at baseline. Even though the results from the non-parametric bootstrap approach (*bootnet*-analysis) reinforce the generalizability of the present findings in similar samples, cross-sample validations in larger samples are required to draw firm conclusions. We cannot determine whether the observed network changes are specific to ABM, or could be the result of any form of intervention. Uncertainty still abounds regarding the best way to interpret the relevance of the rather small improvement in HDRS score in the present sample (that is, among patients not currently fulfilling the diagnostic criteria for depression). We cannot exclude the premise that the present effects of ABM on symptom networks would have been different with larger effects size.

Experimental studies are still needed to draw conclusions regarding the causal and mediating role of metacognitions. The bidirectional links between metacognitions and depression need to be confirmed in a currently depressed population. Although there were no effects of the ABM condition on depression, rumination and metacognitions, we cannot rule out the possibility that the context of being enrolled in an intervention study affected the results. The present study cannot elucidate whether depression further exacerbates metacognitions, as claimed by the metacognitive model. The study should have included the same measures of cognitive control as used in paper III to enable more direct comparison of the results. Metacognitions and rumination were only assessed at baseline and at the last follow-up, and cognitive control was only assessed at baseline. Assessing these variables at all follow-ups would have enabled better analysis of the interplay between these variables.

There were only small to medium associations between metacognitions and objective measures of cognitive control. Moreover, the overall generalizability of these findings are questionable, as the sample is not representative of the general population. The mechanism underlying the relationship between metacognitions and executive control could have been illuminated by analysing the data with participants separated into sub-groups (e.g. based on diagnosis, number of depressive episodes, etc.). This was not possible due to low statistical power. Experimental designs should examine whether reductions in metacognitions leads to better cognitive control, and whether cognitive control training results in decreasing metacognitions. Future research should also examine how cognitive control and metacognitions interact during the course of depression and if this is related to treatment.

## **Future Directions**

### ***Controlled processing and the positive valence system***

Psychological treatments for depression largely aim to reduce negative affect (Craske et al., 2016). This has also been the primary focus of cognitive control research in depression,

mainly examining the processing of negative material. There is no question that cognitive control is implicated in rumination and depression. However, researchers have recently started to question to what extent the problem of dysregulated mood can be explained by deficits in cognitive control, or whether the problem rather is a failure to wilfully engage in controlled processing that supports goal-directed behaviour (i.e. low motivation).

Grahek, Everaert, Krebs, and Koster (2018), for example, critique the lack of integration of the positive valence system in cognitive models of depression. Specifically, cognitive control appears to share some basic neurobiological mechanisms with reward-based processes (Botvinick & Braver, 2015). These processes include decreased willingness to modify behaviour to obtain rewards, impaired ability to learn from obtaining rewards and dissociation between experienced pleasure and willingness to invest effort into achieving pleasure (Grahek et al., 2018). Depression-related cognitive control ‘deficits’ may therefore rather reflect reduced motivation to engage in controlled attainment of rewards.

Future research should aim to examine the interplay between cognitive control and symptoms that are grounded in the positive affective system, such as the experience of low interest/anhedonia and other reward-related processes. One possibility is to examine this question using network analysis, for example by including objective measures of cognitive control or behavioural measures of reward-related processes in a symptom network. Analyses could also include items from the Positive and Negative Affect Schedule, which are more specific to the positive and negative affective systems (Watson et al., 1988). Exploration of the relationship between metacognitions and specific depression symptoms, and positive and negative affective processes, should also be considered.

### *Depression as a complex system of interacting mechanisms*

Traditionally, depression studies are based on individuals who experience a substantial presence of symptoms, meaning they fulfil the diagnostic criteria of MDD. The main rationale behind this approach is the assumption that a specific constellation of symptoms reflects specific mental disorders and that these are the result of specific pathogenic pathways. The vast majority of research on the cognitive mechanisms of depression has been conducted using this approach. However, recent years have documented that this is problematic.

Research based on diagnostic categories does not align with findings from clinical neuroscience and genetics, is not predictive of treatment response, and may not capture the underlying mechanisms of dysfunction. Relying on the traditional diagnostic system is likely constraining new scientific discoveries in clinical psychology and psychiatry (Insel et al., 2010).

There has been a call for multi-level aetiological models involving multiple causal processes at the micro and macro level, within and outside the individual (Kendler, 2008). This has sparked a paradigmatic shift in psychopathology research, where the emerging focus is to use dimensional classifications of mental disorders (Kotov, Krueger, & Watson, 2018) and delineate how causal mechanisms interact to produce symptom clusters (Cuthbert, 2015).

The network approach (utilized in paper I) offers a promising conceptual framework to explore the complex interplay between important psychopathological processes at the dimensional level (Hofmann, Curtiss, & McNally, 2016). The essence of this approach is that psychiatric symptoms co-occur because they are causally related to one another (i.e. sleep problems cause fatigue, fatigue causes loss of interest); symptoms are not mere reflections of an underlying mental disorder (Borsboom, 2017).

This approach has primarily been used to characterize symptom patterns using self-report assessment. Recently, however, researchers have advocated for the inclusion of non-symptom variables that are assumed to be plausible causal candidates in the aetiology or maintenance of disorders (Jones, Heeren, & McNally, 2017). Metacognitions, rumination, attentional bias and cognitive control are examples of relevant variables that can be included in network analyses of depression. To date, only a few studies (in addition to paper I) have included such variables (e.g. Heeren & McNally, 2016; Hoorelbeke, Marchetti, De Schryver, & Koster, 2016).

Including variables that enable more optimal assessment of attentional bias, such as eye-tracking, might also be more appropriate in future research (for examples, see Price et al., 2015; Sanchez-Lopez, Vanderhasselt, Allaert, Baeken, & De Raedt, 2018; Zvielli, Bernstein, & Koster, 2014).

Complex models of depression emphasize the importance of positive feedback loops to explain how symptoms are worsened or maintained. Various reinforcing loops activated by external factors (e.g. stress or loss) may push some individuals towards a trajectory of increasingly negative cognition, more attentional bias for negative information, decreasing social contact and impaired cognitive control. Once established, it may be difficult to mitigate this feedback loop even if the triggering stress dissipates. Subtle individual differences in positive feedback mechanisms may explain why some individuals develop very different symptomatologic trajectories in response to stress or loss, and why others are more resilient. Some patients may be trapped in cognitive feedback loops, others by biological or social ones, or any combination of these (Wittenborn, Rahmandad, Rick, & Hosseinichimeh, 2016). The specific feedback mechanisms may be unique between different individuals, and may not necessarily involve all mechanisms depicted in the synthesis model presented in the Introduction (Figure 1).



### *Towards a personalized approach to depression*

There is a shift towards understanding the interplay between the causal mechanisms at the individual level. Previous research on depression has almost exclusively examined the proposed causal mechanisms using between-subject designs. However, generalizing from the group level to a specific individual is far less accurate and valid than assumed (Fisher, Medaglia, & Jeronimus, 2018; Molenaar, 2004). This problem is particularly disturbing for depression research, as depression is a very heterogenic disorder (Goldberg, 2011) with numerous plausible aetiological and maintaining pathways (e.g. Charney & Manji, 2004; Harrington, Rutter, & Fombonne, 1996; Hasler, 2010; Wittenborn et al., 2016).

With smart phones and other wearables, researchers can assess relevant psychological, physiological and behavioural variables in real-time (Miller, 2012). Analysing these data can reveal dynamic processes within the person (Trull & Ebner-Priemer, 2009). This perspective provides an exciting possibility for examining the dynamic interplay between causal mechanisms in depression at the individual level. Using intensive time series data in combination graphical vector autoregressive modelling, researchers can capture the within- and between-person temporal dynamics of individual symptom networks (e.g. Wichers, Groot, & Psychosystems, 2016; Wild et al., 2010). This may provide a more fine-grained examination of the interplay between causal mechanisms in a specific patient. In this manner, symptoms and other variables can be tracked over time to optimally model the continuous dynamics of the interplay between processes during interventions or a depressive relapse.

This approach provides a promising conceptual framework for personalized assessment and treatment (Fried et al., 2017) and may have the potential to inform clinicians in choosing treatments that are tailor-made to address each patient's problem (Epskamp et al., 2018; Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017). For example, based on these estimations, the symptom profiles for which MCT or ABM might be the most effective treatment approach

can be identified. Consequently, future research should aim to understand the interplay of causal mechanisms of mental disorders at individual level (Barlow & Nock, 2009; Fisher, 2015; Molenaar, 2004).

## **Conclusions**

Contemporary cognitive models of depression emphasize the interplay between symptoms, rumination, attentional bias and the control and coordination of cognition. This thesis examined three aspects of this interplay using novel methodological and statistical approaches. The main findings were: 1) targeting negative attentional bias using a computerized intervention leads to reduced interaction between depression and anxiety symptoms, 2) we found evidence for a feedback loop between depression symptoms and metacognitions, and 3) that there is an apparent basic interplay between metacognitions and cognitive control. In sum, using a variety of methodological approaches, this thesis demonstrates that depression is characterized by an interplay within and between different levels of cognitive processing. Examining these processes in parallel, as they unfold, within a complex system perspective, and at the level of the individual could provide new avenues for developing treatments that target the pathogenic processes underlying depression more precisely.

## References

- Albert, P. R. (2015). Why Is Depression More Prevalent in Women? *Journal of Psychiatry & Neuroscience, 40*(4), 219-221. doi:10.1503/jpn.150205
- Armstrong, T., & Olatunji, B. O. (2012). Eye Tracking of Attention in the Affective Disorders: A Meta-Analytic Review and Synthesis. *Clinical Psychology Review, 32*(8), 704-723. doi:10.1016/j.cpr.2012.09.004
- Banich, M. T. (2009). Executive Function: The Search for an Integrated Account. *Current Directions in Psychological Science, 18*(2), 89-94. doi:10.1111/j.1467-8721.2009.01615.x
- Barlow, D. H., & Nock, M. K. (2009). Why Can't We Be More Idiographic in Our Research? *Perspectives on Psychological Science, 4*(1), 19-21. doi:10.1111/j.1745-6924.2009.01088.x
- Beck, A. T., & Bredemeier, K. (2016). A Unified Model of Depression. *Clinical Psychological Science, 4*(4), 596-619. doi:10.1177/2167702616628523
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An Inventory for Measuring Clinical Anxiety: Psychometric Properties. *Journal of Consulting and Clinical Psychology, 56*(6), 893. doi:10.1037/0022-006X.56.6.893
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.
- Beckwé, M., Deroost, N., Koster, E. H., De Lissnyder, E., & De Raedt, R. (2014). Worrying and Rumination Are Both Associated with Reduced Cognitive Control. *Psychological Research, 78*(5), 651-660. doi:10.1007/s00426-013-0517-5
- Borsboom, D. (2017). A Network Theory of Mental Disorders. *World Psychiatry, 16*(1), 5-13. doi:10.1002/wps.20375

- Borsboom, D., & Cramer, A. O. J. (2013). Network Analysis: An Integrative Approach to the Structure of Psychopathology. *Annual Review of Clinical Psychology*, 91-121.  
doi:10.1146/annurev-clinpsy-050212-185608
- Boschloo, L., van Borkulo, C. D., Borsboom, D., & Schoevers, R. A. (2016). A Prospective Study on How Symptoms in a Network Predict the Onset of Depression. *Psychotherapy and Psychosomatics*, 85(3), 183-184. doi:10.1159/000442001
- Botvinick, M., & Braver, T. (2015). Motivation and Cognitive Control: From Behavior to Neural Mechanism. *Annual Review of Psychology*, 66(1), 83-113.  
doi:10.1146/annurev-psych-010814-015044
- Bringmann, L. F., Lemmens, L. H. J. M., Huibers, M. J. H., Borsboom, D., & Tuerlinckx, F. (2014). Revealing the Dynamic Network Structure of the Beck Depression Inventory-II. *Psychological Medicine*, 45(4), 747-757. doi:10.1017/S0033291714001809
- Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and Lifetime Comorbidity of the Dsm-IV Anxiety and Mood Disorders in a Large Clinical Sample. *Journal of Abnormal Psychology*, 110(4), 585-599.  
doi:10.1037/0021-843X.110.4.585
- Browning, M., Holmes, E. A., Charles, M., Cowen, P. J., & Harmer, C. J. (2012). Using Attentional Bias Modification as a Cognitive Vaccine against Depression. *Biological Psychiatry*, 72(7), 572-579. doi:10.1016/j.biopsych.2012.04.014
- Buckman, J. E. J., Underwood, A., Clarke, K., Saunders, R., Hollon, S. D., Fearon, P., & Pilling, S. (2018). Risk Factors for Relapse and Recurrence of Depression in Adults and How They Operate: A Four-Phase Systematic Review and Meta-Synthesis. *Clinical Psychology Review*, 64, 13-38. doi:10.1016/j.cpr.2018.07.005
- Burcusa, S. L., & Iacono, W. G. (2007). Risk for Recurrence in Depression. *Clinical Psychology Review*, 27(8), 959-985. doi:10.1016/j.cpr.2007.02.005

- Cacioppo, J. T., & Berntson, G. G. (1999). The Affect System: Architecture and Operating Characteristics. *Current Directions in Psychological Science*, 8(5), 133-137.  
doi:10.1111/1467-8721.00031
- CANTAB. (2009). The Cambridge Neuropsychological Test Automated Battery. Cambridge Cognition Limited: Cambridge, UK.
- Charney, D. S., & Manji, H. K. (2004). Life Stress, Genes, and Depression: Multiple Pathways Lead to Increased Risk and New Opportunities for Intervention. *Science's STKE*, 2004(225), re5. doi:10.1126/stke.2252004re5
- Craske, M. G., Meuret, A. E., Ritz, T., Treanor, M., & Dour, H. J. (2016). Treatment for Anhedonia: A Neuroscience Driven Approach. *Depression and Anxiety*, 33(10), 927-938. doi:10.1002/da.22490
- Cristea, I. A., Kok, R. N., & Cuijpers, P. (2015). Efficacy of Cognitive Bias Modification Interventions in Anxiety and Depression: Meta-Analysis. *The British Journal of Psychiatry*, 206(1), 7-16. doi:10.1192/bjp.bp.114.146761
- Cristea, I. A., Kok, R. N., & Cuijpers, P. (2017). Confusing Procedures with Process in Cognitive Bias Modification Research. *The British Journal of Psychiatry*, 211(5), 272. doi:10.1192/bjp.bp.117.200972
- Cuthbert, B. N. (2015). Research Domain Criteria: Toward Future Psychiatric Nosologies. *Dialogues in Clinical Neuroscience*, 17(1), 89-97.
- 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. doi:10.3758/CABN.10.1.50
- Delis, D. C., Kaplan, E., & Kramer, J. (2001). *Delis-Kaplan Executive Function System*. San Antonio, TX: Psychological Corporation.

- Diamond, A. (2013). Executive Functions. *Annual Review of Psychology*, *64*, 135-168.  
doi:10.1146/annurev-psych-113011-143750 23020641
- Dillon, D. G., Rosso, I. M., Pechtel, P., Killgore, W. D. S., Rauch, S. L., & Pizzagalli, D. A. (2014). Peril and Pleasure: An Rdoc-Inspired Examination of Threat Responses and Reward Processing in Anxiety and Depression. *Depression and Anxiety*, *31*(3), 233-249. doi:10.1002/da.22202
- Disner, S. G., Beevers, C. G., Haigh, E. A. P., & Beck, A. T. (2011). Neural Mechanisms of the Cognitive Model of Depression. *Nature Reviews Neuroscience*, *12*(8), 467-477.
- Donaldson, C., Lam, D., & Mathews, A. (2007). Rumination and Attention in Major Depression. *Behaviour Research and Therapy*, *45*(11), 2664-2678.  
doi:10.1016/j.brat.2007.07.002
- Dozois, D. J. A., Dobson, K. S., & Ahnberg, J. L. (1998). A Psychometric Evaluation of the Beck Depression Inventory–II. *Psychological Assessment*, *10*(2), 83-89.  
doi:10.1037/1040-3590.10.2.83
- Epskamp, S., Borsboom, D., & Fried, E. I. (2017). Estimating Psychological Networks and Their Accuracy: A Tutorial Paper. *Behavioral Research Methods*, *50*(1), 195-212.  
doi:10.3758/s13428-017-0862-1
- Epskamp, S., Cramer, A. O. J., Waldorp, L. J., Schmittmann, V. D., & Borsboom, D. (2012). Qgraph: Network Visualizations of Relationships in Psychometric Data. *Journal of Statistical Software*, *48*(4), 1-18. doi:10.18637/jss.v048.i04
- Epskamp, S., van Borkulo, C. D., van der Veen, D. C., Servaas, M. N., Isvoranu, A.-M., Riese, H., & Cramer, A. O. J. (2018). Personalized Network Modeling in Psychopathology: The Importance of Contemporaneous and Temporal Connections. *Clinical Psychological Science*, *6*(3), 416-427. doi:10.1177/2167702617744325

- Everaert, J., Grahek, I., Duyck, W., Buelens, J., Van den Bergh, N., & Koster, E. H. W. (2017). Mapping the Interplay among Cognitive Biases, Emotion Regulation, and Depressive Symptoms. *Cognition and Emotion*, *31*(4), 726-735.  
doi:10.1080/02699931.2016.1144561
- Everaert, J., Grahek, I., & Koster, E. H. W. (2017). Individual Differences in Cognitive Control over Emotional Material Modulate Cognitive Biases Linked to Depressive Symptoms. *Cognition and Emotion*, *31*(4), 736-746.  
doi:10.1080/02699931.2016.1144562
- Everaert, J., Koster, E. H. W., & Derakshan, N. (2012). The Combined Cognitive Bias Hypothesis in Depression. *Clinical Psychology Review*, *32*(5), 413-424.  
doi:10.1016/j.cpr.2012.04.003 22681914
- Feliciano, L., & Renn, B. N. (2014). Mood Disorders: Depressive Disorders. In D. C. Beidel, B. C. Frueh, & M. Hersen (Eds.), *Adult psychopathology and diagnosis* (pp. 253-289). Hoboken, New Jersey: John Wiley & Sons.
- Fernandez-Duque, D., Baird, J. A., & Posner, M. I. (2000). Executive Attention and Metacognitive Regulation. *Consciousness and Cognition*, *9*(2), 288-307.  
doi:10.1006/ccog.2000.0447
- First, M. B., & Gibbon, M. (2004). The Structured Clinical Interview for Dsm-IV Axis I Disorders (Scid-I) and the Structured Clinical Interview for Dsm-IV Axis II Disorders (Scid-II). In M. J. Hilsenroth & D. L. Segal (Eds.), *Comprehensive handbook of psychological assessment, Vol. 2. Personality assessment* (pp. 134-143). Hoboken, New Jersey: John Wiley & Sons Inc.
- Fisher, A. J. (2015). Toward a Dynamic Model of Psychological Assessment: Implications for Personalized Care. *Journal of Consulting and Clinical Psychology*, *83*(4), 825-836.  
doi:10.1037/ccp0000026

- Fisher, A. J., Medaglia, J. D., & Jeronimus, B. F. (2018). Lack of Group-to-Individual Generalizability Is a Threat to Human Subjects Research. *Proceedings of the National Academy of Sciences*, *115*(27), E6106- E6115. doi:10.1073/pnas.1711978115
- Fisher, A. J., Reeves, J. W., Lawyer, G., Medaglia, J. D., & Rubel, J. A. (2017). Exploring the Idiographic Dynamics of Mood and Anxiety Via Network Analysis. *Journal of Abnormal Psychology*, *126*(8), 1044-1056. doi:10.1037/abn0000311
- Fox, E. (2005). The Role of Visual Processes in Modulating Social Interactions. *Visual Cognition*, *12*(1), 1-11. doi:10.1080/13506280444000067
- Fried, E. I., Epskamp, S., Nesse, R. M., Tuerlinckx, F., & Borsboom, D. (2016). What Are 'Good' Depression Symptoms? Comparing the Centrality of DSM and Non-DSM Symptoms of Depression in a Network Analysis. *Journal of Affective Disorders*, *189*, 314-320. doi:10.1016/j.jad.2015.09.005 26458184
- Fried, E. I., & Nesse, R. M. (2014). The Impact of Individual Depressive Symptoms on Impairment of Psychosocial Functioning. *PLOS ONE*, *9*(2), e90311. doi:10.1371/journal.pone.0090311
- Fried, E. I., & Nesse, R. M. (2015). Depression Sum-Scores Don't Add Up: Why Analyzing Specific Depression Symptoms Is Essential. *BMC Medicine*, *13*(72). doi:10.1186/s12916-015-0325-4
- Fried, E. I., Nesse, R. M., Guille, C., & Sen, S. (2015). The Differential Influence of Life Stress on Individual Symptoms of Depression. *Acta Psychiatrica Scandinavica*, *131*(6), 465-471. doi:10.1111/acps.12395
- Fried, E. I., van Borkulo, C. D., Cramer, A. O. J., Boschloo, L., Schoevers, R. A., & Borsboom, D. (2017). Mental Disorders as Networks of Problems: A Review of Recent Insights. *Social Psychiatry and Psychiatric Epidemiology*, *52*(1), 1-10. doi:10.1007/s00127-016-1319-z



- Friedman, N. P., & Miyake, A. (2017). Unity and Diversity of Executive Functions: Individual Differences as a Window on Cognitive Structure. *Cortex*, *86*, 186-204. doi:10.1016/j.cortex.2016.04.023
- Gilbert, P. (2006). Evolution and Depression: Issues and Implications. *Psychological Medicine*, *36*(3), 287-297. doi:10.1017/S0033291705006112
- Goldberg, D. (2011). The Heterogeneity of "Major Depression". *World Psychiatry*, *10*(3), 226-228.
- Gotlib, I. H., & Joormann, J. (2010). Cognition and Depression: Current Status and Future Directions. *Annual Review of Clinical Psychology*, *6*, 285. doi:10.1146/annurev.clinpsy.121208.131305
- Gotlib, I. H., Krasnoperova, E., Yue, D. N., & Joormann, J. (2004). Attentional Biases for Negative Interpersonal Stimuli in Clinical Depression. *Journal of Abnormal Psychology*, *113*(1), 127-135. doi:10.1037/0021-843X.113.1.121
- Grafton, B., MacLeod, C., Rudaizky, D., Holmes, E. A., Salemink, E., Fox, E., & Notebaert, L. (2017). Confusing Procedures with Process When Appraising the Impact of Cognitive Bias Modification on Emotional Vulnerability. *The British Journal of Psychiatry*, *211*(5), 266.
- Grahek, I., Everaert, J., Krebs, R. M., & Koster, E. H. W. (2018). Cognitive Control in Depression: Toward Clinical Models Informed by Cognitive Neuroscience. *Clinical Psychological Science*, *6*(4), 464-480. doi:10.1177/2167702618758969
- Grol, M., Hertel, P. T., Koster, E. H. W., & De Raedt, R. (2015). The Effects of Rumination Induction on Attentional Breadth for Self-Related Information. *Clinical Psychological Science*, *3*(4), 607-618. doi:10.1177/2167702614566814
- 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*, 8(31). doi: 10.3389/fpsyg.2017.00031
- Hallion, L. S., & Ruscio, A. M. (2011). A Meta-Analysis of the Effect of Cognitive Bias Modification on Anxiety and Depression. *Psychological Bulletin*, 137(6), 940-958. doi:10.1037/a0024355 21728399
- 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. doi:10.1007/s10608-014-9638-4
- Hamilton, M. (1960). A Rating Scale for Depression. *Journal of Neurology, Neurosurgery & Psychiatry*, 23(1), 56-62. doi:10.1136/jnnp.23.1.56
- Harmer, C. J., Duman, R. S., & Cowen, P. J. (2017). How Do Antidepressants Work? New Perspectives for Refining Future Treatment Approaches. *The Lancet Psychiatry*, 4(5), 409-418. doi:10.1016/S2215-0366(17)30015-9
- Harrington, R., Rutter, M., & Fombonne, E. (1996). Developmental Pathways in Depression: Multiple Meanings, Antecedents, and Endpoints. *Development and Psychopathology*, 8(4), 601-616. doi:10.1017/S095457940000732X
- Hasler, G. (2010). Pathophysiology of Depression: Do We Have Any Solid Evidence of Interest to Clinicians? *World Psychiatry*, 9(3), 155-161.
- Heeren, A., De Raedt, R., Koster, E. H. W., & Philippot, P. (2013). The (Neuro)Cognitive Mechanisms Behind Attention Bias Modification in Anxiety: Proposals Based on Theoretical Accounts of Attentional Bias. *Frontiers in Human Neuroscience*, 7, 119-119. doi:10.3389/fnhum.2013.00119

- Heeren, A., & McNally, R. J. (2016). An Integrative Network Approach to Social Anxiety Disorder: The Complex Dynamic Interplay among Attentional Bias for Threat, Attentional Control, and Symptoms. *Journal of Anxiety Disorders, 42*, 95-104. doi:10.1016/j.janxdis.2016.06.009
- Hertel, P. T., & Mathews, A. (2011). Cognitive Bias Modification: Past Perspectives, Current Findings, and Future Applications. *Perspectives on Psychological Science, 6*(6), 521-536. doi:10.1177/1745691611421205
- Hofmann, S. G., Curtiss, J., & McNally, R. J. (2016). A Complex Network Perspective on Clinical Science. *Perspectives on Psychological Science, 11*(5), 597-605. doi:10.1177/1745691616639283 27694457
- Hoorelbeke, K., Marchetti, I., De Schryver, M., & Koster, E. H. W. (2016). The Interplay between Cognitive Risk and Resilience Factors in Remitted Depression: A Network Analysis. *Journal of Affective Disorders, 195*, 96-104. doi:10.1016/j.jad.2016.02.001
- Ingram, R. E. (1984). Toward an Information-Processing Analysis of Depression. *Cognitive Therapy and Research, 8*(5), 443-477. doi:10.1007/BF01173284
- Ingram, R. E. (1986). *Information Processing Approaches to Clinical Psychology. Personality, Psychopathology, and Psychotherapy Series*. San Diego, California: Academic Press.
- Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D. S., Quinn, K., . . . Wang, P. (2010). Research Domain Criteria (RDOC): Toward a New Classification Framework for Research on Mental Disorders. *American Journal of Psychiatry, 167*(7), 748-751. doi:10.1176/appi.ajp.2010.09091379
- Jonassen, R., Harmer, C. J., Hilland, E., Maglanoc, L. A., Kraft, B., Browning, M., . . . Landro, N. I. (2018). Effects of Attentional Bias Modification on Residual Symptoms

- in Depression. A Randomized Controlled Trial. *bioRxiv (Preprint: 318279)*.  
doi:10.1101/318279
- Jones, P. J., Heeren, A., & McNally, R. J. (2017). Commentary: A Network Theory of Mental Disorders. *Frontiers in Psychology, 8*, 1305. doi:10.3389/fpsyg.2017.01305
- Joormann, J., Dkane, M., & Gotlib, I. H. (2006). Adaptive and Maladaptive Components of Rumination? Diagnostic Specificity and Relation to Depressive Biases. *Behavior Therapy, 37*(3), 269-280. doi:10.1016/j.beth.2006.01.002
- Joormann, J., & Gotlib, I. H. (2007). Selective Attention to Emotional Faces Following Recovery from Depression. *Journal of Abnormal Psychology, 116*(1), 80-85.  
doi:10.1037/0021-843X.116.1.80
- Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased Processing of Emotional Information in Girls at Risk for Depression. *Journal of Abnormal Psychology, 116*(1), 135-143.  
doi:10.1037/0021-843x.116.1.135
- Joormann, J., & Vanderlind, W. M. (2014). Emotion Regulation in Depression: The Role of Biased Cognition and Reduced Cognitive Control. *Clinical Psychological Science, 2*(4), 402-421. doi:10.1177/2167702614536163
- Joormann, J., Yoon, K. L., & Zetsche, U. (2007). Cognitive Inhibition in Depression. *Applied and Preventive Psychology, 12*(3), 128-139. doi:10.1016/j.appsy.2007.09.002
- Kaplan, J. S., Erickson, K., Luckenbaugh, D. A., Weiland-Fiedler, P., Geraci, M., Sahakian, B. J., . . . Neumeister, A. (2006). Differential Performance on Tasks of Affective Processing and Decision-Making in Patients with Panic Disorder and Panic Disorder with Comorbid Major Depressive Disorder. *Journal of Affective Disorders, 95*(1), 165-171. doi:10.1016/j.jad.2006.04.016
- Kendler, K. S. (2008). Explanatory Models for Psychiatric Illness. *American Journal of Psychiatry, 165*(6), 695-702. doi:10.1176/appi.ajp.2008.07071061

- Kendler, K. S., Zachar, P., & Craver, C. (2011). What Kinds of Things Are Psychiatric Disorders? *Psychological Medicine*, *41*(6), 1143-1150.  
doi:10.1017/S0033291710001844 20860872
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., . . . National Comorbidity Survey, R. (2003). The Epidemiology of Major Depressive Disorder: Results from the National Comorbidity Survey Replication (Ncs-R). *JAMA*, *289*(23), 3095-3105. doi:10.1001/jama.289.23.3095
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K., & Walters, E. E. (2005). Lifetime Prevalence and Age-of-Onset Distributions of Dsm-Iv Disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*.  
doi:10.1001/archpsyc.62.6.593
- Kessler, R. C., & Bromet, E. J. (2013). The Epidemiology of Depression across Cultures. *Annual Review of Public Health*, *34*, 119-138. doi:10.1146/annurev-publhealth-031912-114409
- 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.  
doi:10.1016/j.cpr.2010.08.005
- Koster, E. H. W., Hoorelbeke, K., Onraedt, T., Owens, M., & Derakshan, N. (2017). Cognitive Control Interventions for Depression: A Systematic Review of Findings from Training Studies. *Clinical Psychology Review*, *53*, 79-92. doi:  
10.1016/j.cpr.2017.02.002
- Kotov, R., Krueger, R. F., & Watson, D. (2018). A Paradigm Shift in Psychiatric Classification: The Hierarchical Taxonomy of Psychopathology (Hitop). *World Psychiatry*, *17*(1), 24-25. doi:10.1002/wps.20478

- Kringlen, E., Torgersen, S., & Cramer, V. (2001). A Norwegian Psychiatric Epidemiological Study. *American Journal of Psychiatry*, *158*(7), 1091-1098.  
doi:10.1176/appi.ajp.158.7.1091
- Kringlen, E., Torgersen, S., & Cramer, V. (2006). Mental Illness in a Rural Area: A Norwegian Psychiatric Epidemiological Study. *Social Psychiatry and Psychiatric Epidemiology*, *41*(9), 713-719. doi:10.1007/s00127-006-0080-0
- Kujawa, A. J., Torpey, D., Kim, J., Hajcak, G., Rose, S., Gotlib, I. H., & Klein, D. N. (2011). Attentional Biases for Emotional Faces in Young Children of Mothers with Chronic or Recurrent Depression. *Journal of Abnormal Child Psychology*, *39*(1), 125-135.  
doi:10.1007/s10802-010-9438-6
- LeMoult, J., & Gotlib, I. H. (2018). Depression: A Cognitive Perspective. *Clinical Psychology Review*. doi:10.1016/j.cpr.2018.06.008
- Linville, P. (1996). Attention Inhibition: Does It Underlie Ruminative Thought? In R. S. Wyer (Ed.), *Ruminative Thoughts: Advances in Social Cognition* (pp. 121–133). Mahwah, New Jersey: Erlbaum.
- Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and Inhibitory Control. *Psychological Science*, *8*(1), 60-64. doi:10.1111/j.1467-9280.1997.tb00545.x
- Luminet, O. (2003). Measurement of Depressive Rumination and Associated Constructs. In C. Papageorgiou & A. Wells (Eds.), *Depressive Rumination* (pp. 187-216). West Sussex, England: John Wiley & Sons.
- Lundqvist, D., Flykt, A., & Öhman, A. (1998). The Karolinska Directed Emotional Faces - Kdef [CD-ROM]. Department of Clinical Neuroscience, Psychology section, Karolinska Institutet.

- Lyubomirsky, S., & Tkach, C. (2004). The Consequences of Dysphoric Rumination. In C. Papageorgiou & A. Wells (Eds.), *Depressive Rumination* (pp. 21-41). West Sussex, England: John Wiley & Sons.
- Løvstad, M., Sigurdardottir, S., Andersson, S., Grane, V., Moberget, T., Stubberud, J., & Solbakk, A. (2016). Behavior Rating Inventory of Executive Function Adult Version in Patients with Neurological and Neuropsychiatric Conditions: Symptom Levels and Relationship to Emotional Distress. *Journal of the International Neuropsychological Society*, 22, 682–694. doi:10.1017/S135561771600031X
- MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective Attention and Emotional Vulnerability: Assessing the Causal Basis of Their Association through the Experimental Manipulation of Attentional Bias. *Journal of Abnormal Psychology*, 111(1). doi:10.1037/0021-843X.111.1.107 11866165
- Mathews, A., Mackintosh, B., & Fulcher, E. P. (1997). Cognitive Biases in Anxiety and Attention to Threat. *Trends in Cognitive Sciences*, 1(9), 340-345. doi:10.1016/S1364-6613(97)01092-9
- 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, England: John Wiley & Sons.
- Maurage, P., Heeren, A., & Pesenti, M. (2013). Does Chocolate Consumption Really Boost Nobel Award Chances? The Peril of Over-interpreting Correlations in Health Studies. *Journal of Nutrition*, 143(6), 931-933. doi:10.3945/jn.113.174813
- McNally, R. J. (2018). Attentional Bias for Threat: Crisis or Opportunity? *Clinical Psychology Review*. doi:10.1016/j.cpr.2018.05.005
- Miller, G. (2012). The Smartphone Psychology Manifesto. *Perspectives on Psychological Science*, 7(3), 221-237. doi:10.1177/1745691612441215

- 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. doi: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. doi:10.1006/cogp.1999.0734
- Mogg, K., & Bradley, B. P. (2005). Attentional Bias in Generalized Anxiety Disorder Versus Depressive Disorder. *Cognitive Therapy and Research*, 29(1), 29-45. doi:10.1007/s10608-005-1646-y
- Molenaar, P. C. M. (2004). A Manifesto on Psychology as Idiographic Science: Bringing the Person Back into Scientific Psychology, This Time Forever. *Measurement: Interdisciplinary Research and Perspectives*, 2(4), 201-218. doi:10.1207/s15366359mea0204\_1
- Mueller, T. I., Leon, A. C., Keller, M. B., Solomon, D. A., Endicott, J., Coryell, W., . . . Maser, J. D. (1999). Recurrence after Recovery from Major Depressive Disorder During 15 Years of Observational Follow-Up. *The American Journal of Psychiatry*, 156(7), 1000-1006.
- Nesse, R. M. (1990). Evolutionary Explanations of Emotions. *Human Nature*, 1(3), 261-289. doi:10.1007/BF02733986 24222085
- Nesse, R. M. (2000). Is Depression an Adaptation? *Archives of General Psychiatry*, 57(1), 14-20. doi:10.1001/archpsyc.57.1.14
- Nolen-Hoeksema, S. (1991). Responses to Depression and Their Effects on the Duration of Depressive Episodes. *Journal of Abnormal Psychology*, 100(4), 569-582. doi:10.1037/0021-843X.100.4.569



- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking Rumination. *Perspectives on Psychological Science*, 3(5), 400-424. doi:10.1111/j.1745-6924.2008.00088.x
- Owen, A. M., Downes, J. J., Sahakian, B. J., Polkey, C. E., & Robbins, T. W. (1990). Planning and Spatial Working Memory Following Frontal Lobe Lesions in Man. *Neuropsychologia*, 28(10), 1021-1034. doi:10.1016/0028-3932(90)90137-D
- Owens, M., & Gibb, B. E. (2017). Brooding Rumination and Attentional Biases in Currently Non-Depressed Individuals: An Eye-Tracking Study. *Cognition and Emotion*, 31(5), 1062-1069. doi:10.1080/02699931.2016.1187116
- Papageorgiou, C., & Wells, A. (2001a). Metacognitive Beliefs About Rumination in Recurrent Major Depression. *Cognitive and Behavioral Practice*, 8(2), 160-164. doi: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. doi: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. doi: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. doi: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.

- Peckham, A. D., McHugh, R., & Otto, M. W. (2010). A Meta-Analysis of the Magnitude of Biased Attention in Depression. *Depression and Anxiety*, 27(12), 1135-1142.  
doi:10.1002/da.20755
- Price, R. B., Kuckertz, J. M., Siegle, G. J., Ladouceur, C. D., Silk, J. S., Ryan, N. D., . . . Amir, N. (2015). Empirical Recommendations for Improving the Stability of the Dot-Probe Task in Clinical Research. *Psychological Assessment*, 27(2), 365-376.  
doi:10.1037/pas0000036
- Sanchez-Lopez, A., Vanderhasselt, M.-A., Allaert, J., Baeken, C., & De Raedt, R. (2018). Neurocognitive Mechanisms Behind Emotional Attention: Inverse Effects of Anodal Tdcs over the Left and Right Dlpfc on Gaze Disengagement from Emotional Faces. *Cognitive, Affective, & Behavioral Neuroscience*, 18(3), 485-494.  
doi:10.3758/s13415-018-0582-8
- Sarisoy, G., Pazvantoğlu, O., Özturan, D. D., Ay, N. D., Yilman, T., Mor, S., . . . Gümüş, K. (2014). Metacognitive Beliefs in Unipolar and Bipolar Depression: A Comparative Study. *Nordic Journal of Psychiatry*, 68(4), 275-281.  
doi:10.3109/08039488.2013.814710
- 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.
- Shiroma, P. R., Thuras, P., Johns, B., & Lim, K. O. (2014). Emotion Recognition Processing as Early Predictor of Response to 8-Week Citalopram Treatment in Late-Life Depression. *International Journal of Geriatric Psychiatry*, 29(11), 1132-1139.  
doi:10.1002/gps.4104

- Smith, J. M., & Alloy, L. B. (2009). A Roadmap to Rumination: A Review of the Definition, Assessment, and Conceptualization of This Multifaceted Construct. *Clinical Psychology Review, 29*(2), 116-128. doi:10.1016/j.cpr.2008.10.003 19128864
- Smith, R., Alkozei, A., Killgore, W. D. S., & Lane, R. D. (2018). Nested Positive Feedback Loops in the Maintenance of Major Depression: An Integration and Extension of Previous Models. *Brain, Behavior, and Immunity, 67*, 374-397. doi:10.1016/j.bbi.2017.09.011
- 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. doi: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. doi:10.1016/j.psychres.2016.05.056
- Southworth, F., Grafton, B., MacLeod, C., & Watkins, E. (2017). Heightened Ruminative Disposition Is Associated with Impaired Attentional Disengagement from Negative Relative to Positive Information: Support for the “Impaired Disengagement” Hypothesis. *Cognition and Emotion, 31*(3), 422-434. doi:10.1080/02699931.2015.1124843
- Spada, M. M., Georgiou, G. A., & Wells, A. (2010). The Relationship among Metacognitions, Attentional Control, and State Anxiety. *Cognitive Behaviour Therapy, 39*(1), 64-71. doi:10.1080/16506070902991791
- Spada, M. M., Mohiyeddini, C., & Wells, A. (2008). Measuring Metacognitions Associated with Emotional Distress: Factor Structure and Predictive Validity of the Metacognitions Questionnaire 30. *Personality and Individual Differences, 45*(3), 238-242. doi:10.1016/j.paid.2008.04.005

- Spada, M. M., Nikčević, A. V., Moneta, G. B., & Wells, A. (2008). Metacognition, Perceived Stress, and Negative Emotion. *Personality and Individual Differences, 44*(5), 1172-1181. doi:10.1016/j.paid.2007.11.010
- Steel, Z., Marnane, C., Iranpour, C., Chey, T., Jackson, J. W., Patel, V., & Silove, D. (2014). The Global Prevalence of Common Mental Disorders: A Systematic Review and Meta-Analysis 1980-2013. *International Journal of Epidemiology, 43*(2), 476-493. doi:10.1093/ije/dyu038
- Trajković, G., Starčević, V., Latas, M., Leštarević, M., Ille, T., Bukumirić, Z., & Marinković, J. (2011). Reliability of the Hamilton Rating Scale for Depression: A Meta-Analysis over a Period of 49 years. *Psychiatry Research, 189*(1), 1-9. doi: 10.1016/j.psychres.2010.12.007
- Treadway, M. T., & Zald, D. H. (2011). Reconsidering Anhedonia in Depression: Lessons from Translational Neuroscience. *Neuroscience and Biobehavioral Reviews, 35*(3), 537-555. doi:10.1016/j.neubiorev.2010.06.006
- Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination Reconsidered: A Psychometric Analysis. *Cognitive Therapy and Research, 27*(3), 247-259. doi:10.1023/a:1023910315561
- Trull, T. J., & Ebner-Priemer, U. W. (2009). Using Experience Sampling Methods/Ecological Momentary Assessment (Esm/Ema) in Clinical Assessment and Clinical Research: Introduction to the Special Section. *Psychological Assessment, 21*(4), 457-462. doi:10.1037/a0017653 19947780
- Valente, T. W. (2012). Network Interventions. *Science, 337*(6090), 49-53. doi:10.1126/science.1217330
- van Borkulo, C. (2016). NetworkComparisonTest. Retrieved from <https://cran.r-project.org/web/packages/NetworkComparisonTest/>

- Watkins, E. R., Moulds, M., & Mackintosh, B. (2005). Comparisons between Rumination and Worry in a Non-Clinical Population. *Behaviour Research and Therapy*, *43*(12), 1577-1585. doi:10.1016/j.brat.2004.11.008
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and Validation of Brief Measures of Positive and Negative Affect: The PANAS Scales. *Journal of Personality and Social Psychology*, *54*(6), 1063-1070. doi:10.1037/0022-3514.54.6.1063
- Watson, D., Wiese, D., Vaidya, J., & Tellegen, A. (1999). The Two General Activation Systems of Affect: Structural Findings, Evolutionary Considerations, and Psychobiological Evidence. *Journal of Personality and Social Psychology*, *76*(5), 820-838. doi:10.1037/0022-3514.76.5.820
- Weissman, M. M., Bland, R. C., Canino, G. J., Faravelli, C., Greenwald, S., Hwu, H. G., . . . Yeh, E. K. (1996). Cross-National Epidemiology of Major Depression and Bipolar Disorder. *JAMA*, *276*(4), 293-299.
- Wells, A. (2011). *Metacognitive Therapy for Anxiety and Depression*. New York: Guilford press.
- Wells, A., & Cartwright-Hatton, S. (2004). A Short Form of the Metacognitions Questionnaire: Properties of the MCQ-30. *Behaviour Research and Therapy*, *42*(4), 385-396. doi:10.1016/S0005-7967(03)00147-5
- Wells, A., & Matthews, G. (1996). Modelling Cognition in Emotional Disorder: The S-REF Model. *Behaviour Research and Therapy*, *34*(11), 881-888. doi:10.1016/S0005-7967(96)00050-2
- Whitmer, A. J., & Gotlib, I. H. (2013). An Attentional Scope Model of Rumination. *Psychological Bulletin*, *139*(5), 1036-1061. doi:10.1037/a0030923
- WHO. (2017). Fact Sheet on Depression. Retrieved from <https://www.who.int/news-room/fact-sheets/detail/depression>

- Wichers, M., Groot, P. C., & Psychosystems. (2016). Critical Slowing Down as a Personalized Early Warning Signal for Depression. *Psychotherapy and Psychosomatics*, 85(2), 114-116. doi:10.1159/000441458
- Wild, B., Eichler, M., Friederich, H.-C., Hartmann, M., Zipfel, S., & Herzog, W. (2010). A Graphical Vector Autoregressive Modelling Approach to the Analysis of Electronic Diary Data. *BMC Medical Research Methodology*, 10(1), 28. doi:10.1186/1471-2288-10-28
- Wittenborn, A. K., Rahmandad, H., Rick, J., & Hosseinichimeh, N. (2016). Depression as a Systemic Syndrome: Mapping the Feedback Loops of Major Depressive Disorder. *Psychological Medicine*, 46(3), 551-562. doi:10.1017/S0033291715002044
- Wolpert, L. (2008). Depression in an Evolutionary Context. *Philosophy, Ethics, and Humanities in Medicine*, 3(8). doi:10.1186/1747-5341-3-8
- Woody, M. L., & Gibb, B. E. (2015). Integrating NIMH Research Domain Criteria (RDOC) into Depression Research. *Current Opinion in Psychology*, 4, 6-12. doi:10.1016/j.copsyc.2015.01.004
- Yang, Cao, S., Shields, G. S., Teng, Z., & Liu, Y. (2017). The Relationships between Rumination and Core Executive Functions: A Meta-Analysis. *Depression and Anxiety*, 34(1), 37-50. doi:10.1002/da.22539
- Zvielli, A., Bernstein, A., & Koster, E. H. W. (2014). Temporal Dynamics of Attentional Bias. *Clinical Psychological Science*, 3(5), 772-788. doi:10.1177/2167702614551572

## **Papers I–III**









Attention Bias Modification in Remitted Depression is Associated with Increased Interest and  
Leads To Reduced Adverse Impact of Anxiety Symptoms and Negative Cognition

Brage Kraft

University of Oslo

Rune Jonassen

University of Oslo

Alexandre Heeren

Université Catholique de Louvain

Catherine Harmer

University of Oxford

Tore Stiles

Norwegian University of Science and Technology

Nils Inge Landrø

University of Oslo

## Author Note

Brage Kraft, Department of Psychology, University of Oslo; Alexandre Heeren, Psychological Sciences Research Institute, Université Catholique de Louvain, Institute of Neuroscience, Université Catholique de Louvain; Rune Jonassen, Department of Psychology, University of Oslo, Akershus University Hospital, Division of Psychiatry, Oslo, Norway; Catherine Harmer, Department of Psychiatry, University of Oxford, Department of Psychology, University of Oslo; Tore Stiles, Department of Psychology, Norwegian University of Science and Technology; Nils Inge Landrø, Department of Psychology, University of Oslo, Department of Psychiatry, Diakonhjemmet Hospital.

This work was supported by grants from the Research Council of Norway (229135) to NIL, and The South East Norway Health Authority Research Funding (2015052) to NIL. The Department of Psychology, University of Oslo has also supported the project.

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. We thank Giulio Costantini and Eiko Fried for initial comments regarding the analysis.

CH has received consultancy fees from Johnson and Johnson Inc., P1vital, and Lundbeck. NIL has received consultancy fees and travel expenses from Lundbeck.

Correspondence concerning this article should be addressed to Brage Kraft, Department of Psychology, University of Oslo, PB 1094 Blindern, 0317 Oslo, Norway. E-mail:

brage.kraft@psykologi.uio.no; [bragekb@gmail.com](mailto:bragekb@gmail.com)

### Abstract

Using a computational network approach, we reanalyzed data from a randomized controlled-trial of attention bias modification task (ABM) on residual depression symptoms. The main aim was to characterize the symptom-to-symptom changes following ABM. ABM was associated with improvements in interest, which was, in turn, associated with improvements in other depression symptoms. Although there were no changes in the global network strength following ABM, the comparison between symptom change in the ABM and control group suggests that ABM lead to a reduction of the association between anxiety, depressed mood, and guilt. Findings suggest that reduction in depression symptoms following ABM may have been set in motion by increased interest and involvement in everyday activities, leading to a reduction of the adverse impact of anxiety and negative cognition. ABM may be more effective in patients where these symptoms are prominent.

*Keywords:* attention bias modification, depression, network analysis, attentional bias

Depression is among the most prevalent and severe mental disorders and is related to substantial individual suffering (Cuijpers & Schoevers, 2004; Demyttenaere et al., 2004). Both currently (Gotlib, Krasnoperova, Yue, & Joormann, 2004) and previously depressed patients (Joormann & Gotlib, 2007) show an *attentional bias* towards negative stimuli. This negative attentional bias can be demonstrated in visual probe tasks, where depressed individuals are faster to respond when probes replace negative stimuli, compared to when probes replace positive stimuli (Peckham, McHugh, & Otto, 2010). This is also the case for never-depressed individuals who are at high risk because of a family history of depression (Joormann, Talbot, & Gotlib, 2007; Kujawa et al., 2011), implying attentional bias as a causal risk factor for depression debut, recurrence and consolidation (Beck & Bredemeier, 2016; Disner, Beevers, Haigh, & Beck, 2011; Mathews & MacLeod, 2005).

Attention bias modification tasks (ABM) aim to reduce attentional bias by automatically directing attention towards more positive stimuli (Hakamata et al., 2010). The sparse studies in depression have shown that ABM has the potential to reduce residual symptoms after depression (Beevers, Clasen, Enock, & Schnyer, 2015; Browning, Holmes, Charles, Cowen, & Harmer, 2012; Wells, Beevers, Id, & Wells, 2010; Yang, Ding, Dai, Peng, & Zhang, 2015). As residual symptoms after a depressive episode are among the strongest predictors for recurrence (Paykel, 2008), ABM might prove effective in preventing relapse. However, as argued in a number of meta-analyses (see for example Cristea, Kok, & Cuijpers, 2015; Mogoase, David, & Koster, 2014), effect sizes are small and definitive conclusions are limited due to small sample size and poor trial methodology employed in many studies. Even so, emotional vulnerability seem to decrease in studies where ABM is followed by a successful shift in attention bias (Grafton et al., 2017, but see also Cristea, Kok, & Cuijpers, 2017).

Recently, in a large preregistered randomized clinical trial of effects of ABM on residual symptoms in depression (Jonassen et al., 2018), a two-week ABM program resulted in a positive change in attentional bias and a small but statistically significant effect on depression symptoms as measured by the clinician-rated interview Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). Importantly, the degree of symptom improvement increased with degree of positive bias modification within the ABM group, in line with the mechanism emphasized by Grafton et al. (2017).

To increase the effect of ABM, researchers are urged to explore novel ways to conceptualize and modify attentional bias in depression (Koster & Bernstein, 2015; Mogg, Waters, & Bradley, 2017). Another approach is to reconsider the model of depression vulnerability and how outcome of ABM is assessed. Clinical effects of ABM for depression has commonly been evaluated based on sum scores on depression rating scales, such as the HRSD or the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996). These have high construct validity and are extensively used in the clinic, but aggregating symptoms into a sum can conceal potentially important insights (Fried & Nesse, 2015). Individual depression symptoms are differentially related to risk factors, impaired psychological functioning, biomarkers, and antidepressant efficacy (for a review, see Fried, Nesse, Zivin, Guille, & Sen, 2014). It is likely that ABM is associated with changes in specific depression symptoms, but that global assessments of symptom change masks this effect. Obtaining this knowledge may inform further development of ABM. Along these lines, the present study reexamines data demonstrating the effect of ABM in residual depression (Jonassen et al., 2018) on the symptom level, using recent innovations in theoretical and computational *network analysis* (Borsboom, 2017; Borsboom & Cramer, 2013; Epskamp, Cramer, Waldorp, Schmittmann, & Borsboom, 2012).

Over the last two years, the network approach has been increasingly used to understand mental disorders as systems of interacting symptoms (Hofmann & Curtiss, 2018; Hofmann, Curtiss, & McNally, 2016; McNally, 2016). According to this approach, each depressive symptom is not reflective of a “depressive disorder”, but possess independent causal powers that influence other symptoms (e.g., *insomnia causes fatigue*); symptoms are not merely passive indicators of an underlying disease (Borsboom & Cramer, 2013). Thus, a depressive episode emerges because of the pairwise associations between depression symptoms. These associations can be visualized in a *network model*, where symptoms are represented by *nodes* and associations are represented by *edges* between the nodes (Borsboom, 2017).

Network analysis can determine which symptoms are the most *central* (i.e., influential) based on the amount of influence that flows from one symptom to another (Borgatti, 2005; Valente, 2012). Central symptoms are those that have many strong relations with other symptoms, and the presence of a central symptom easily spread to other symptoms, potentially producing a *cascade* of activation (Borsboom & Cramer, 2013; Valente, 2012). In depression, low mood and loss of interest appear to be the most central symptoms (Fried et al., 2017), along with anxiety (Fried, Epskamp, Nesse, Tuerlinckx, & Borsboom, 2016). Networks with many strong connections between symptoms (*dense networks*) are more likely pathogenic than networks characterized by weaker connections – i.e., more “vulnerable” (Borsboom, 2017; Borsboom & Cramer, 2013; Fried et al., 2017; Heeren & McNally, 2018).

Individuals with depression show a more densely connected network compared to healthy subjects (Pe et al., 2015), and this predicts difficulty recovering from a depressive episode (van Borkulo et al., 2015, but see also Schweren, van Borkulo, Fried, & Goodyer, 2017). Simulation studies show that in dense networks, only a minimal of worsening in a central symptom may trigger a downstream cascade of symptoms. This might lead to a “vicious cycle” of negative



cognition and symptoms (Teasdale, 1988; Wichers, 2014), with a depressed state as the result (Cramer et al., 2016).

We used network analysis to characterize the symptom changes following ABM. We believe that traditional approaches for conceptualizing symptom changes (i.e., sum scores of depression scales; Fried et al., 2017) could be occluding symptom changes following ABM. Our main aim is thus to provide a comprehensive exploration of ABM's impact on residual depression, and evaluate whether ABM changes specific symptoms or other aspects of the symptom network. *First*, we estimated the initial symptom network before ABM, and compared symptom centrality with previous network studies on depression to assess the generalizability of the current sample. *Second*, we estimated networks based on symptom changes from pre to post ABM, and examined whether specific symptoms are improved or the symptom cascade is changed (i.e., the dynamic changes in symptom-symptom relations over time). We examined whether ABM is associated with improvements in individual symptoms, and decreases the connectivity between symptoms. *Finally*, we examined symptom networks post ABM, and hypothesized that ABM weakens connections among symptoms, that is, ABM reduces the overall network connectivity.

## Method

### Participants

We obtained data from a randomized double-blind placebo-controlled trial of ABM on residual depression symptoms among patients with remitted depression. The study was preregistered at ClinicalTrials.gov (*NCT02648165*). For details regarding design, methods, and results, see Jonassen et al. (2018). ABM training led to significantly greater decrease in clinician rated, but not subjective rated, symptoms of depression as compared to the control condition.

The sample included 322 participants recruited mainly from an outpatient clinic in the Department of Psychiatry, Diakonhjemmet Hospital in Oslo. Participants were also recruited from other clinical sites, by local advertisements, and via social media. Inclusion criteria were the presence of a remitted major depressive disorder, age between 18-65 years, and fluency in Norwegian. Exclusion criteria were the presence of current or former neurological disorder, substance use disorder, attention-deficit disorder, head trauma, psychosis, or bipolar disorder. These criteria were assessed using the *Mini-International Neuropsychiatric Interview* (Sheehan et al., 1998). A total of 302 patients were included per protocol. 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).

### **Measures and Materials**

**Symptoms.** Depression symptoms were measured using HRSD (Hamilton, 1960), which is an observer-rated interview with 17 items. The internal reliability of HRSD was acceptable in the current sample ( $\alpha = .77$ ).

**Attention bias modification task.** The ABM was a computerized, validated visual dot-probe procedure (Browning et al., 2012), presented on laptop computers (14" HP Elitebook 840, 1600x900, 8GB, Intel Core i5-4310U). Paired images of faces (the stimuli) were presented followed by one or two dots (a probe), which appeared behind one of the stimuli. Participants were required to press one of two buttons as quickly as possible to indicate the number of dots in the probe. Stimuli were pictures of emotional faces (Karolinska Directed Emotional Faces, Lundqvist, Flykt, & Öhman, 1998) of three valences: positive, neutral, or negative (angry and fearful). The task comprised 96 trials with equal numbers of the three stimulus pair types. There were equal numbers of trials in which the stimuli were randomly presented for 500 or 1000 ms before the probe was displayed. Stimuli from two valences were displayed in each trial of the task

in one of the following pairing types: positive-neutral, positive-negative, and negative-neutral. Probes were located behind positive (valid) emotional stimuli in 87 % of the trials in the training condition. Thus, when completing training, participants should learn to deploy their attention towards positive stimuli, and in this way develop a relatively more positive attentional bias. The control condition was identical in every respect, other than the location of the probe, which was located behind the positive (valid) stimuli in only 50% of the trials.

### **Procedure**

Participants were explained that the study aimed at examining “attention focus, how this changes over time, and how this is related to mood and depression symptoms” (the specific rationale underlying ABM was not explained). After providing written consent, participants were randomly allocated to either training or control condition by an independent lab technician (not involved in the day to day collection of data) who prepared laptop computers to deliver either training or control treatment according to a randomization list in a 1:1 ratio. A trained administrator, who was blind to condition allocation, assessed depression symptoms and demonstrated ABM. Participants were then instructed to do the task at home twice a day for two weeks (28 sessions in total), before returning for a second assessment of depression symptoms.

### **Network Analysis**

**Network estimation.** We used a Graphical Gaussian Model (GGM) to estimate the networks. In the presented networks, nodes represent depression symptoms and edges represent conditional independence relationships between nodes when controlling for the effects of all other nodes (Epskamp, Borsboom, & Fried, 2017). It is common to regularize GGMs via the graphical LASSO (least absolute shrinkage and selection operator). The graphical LASSO estimates a maximum likelihood solution in which the likelihood is penalized for the sum of absolute parameter estimates (Epskamp & Fried, 2018; Friedman, Hastie, & Tibshirani, 2008).

First, it computes regularized partial correlations between pairs of nodes, thereby limiting the sum of absolute partial correlation coefficients. This eliminates spurious associations (edges) attributable to the influence of other nodes in the network. Second, it shrinks trivially small associations to zero, thereby removing potentially “false positive” edges from the graph and producing a sparse graph comprising only the strongest edges. We used the *R* package *qgraph* (Epskamp et al., 2012), that automatically implements the graphical LASSO regularization, in combination with an extended Bayesian Information Criterion (EBIC) model selection (Foygel & Drton, 2011). In this approach, 100 different network models are estimated with different degrees of sparsity. Then, the model with the lowest EBIC value is selected, given a certain value of the hyperparameter gamma ( $\gamma$ ); this procedure strikes a balance between including false-positive edges and removing true edges. The hyperparameter  $\gamma$  is usually set between zero and 0.5 (Epskamp et al., 2017). As the value of  $\gamma$  nears 0.5, the EBIC will favor a simpler model that contains fewer edges. As the value of  $\gamma$  nears zero, the EBIC will favor a model with a greater number of edges. Given the exploratory nature of the study, we set  $\gamma$  to zero to maximize the sensitivity.

A baseline network was estimated for the whole sample, where edges represent the partial correlations between each of the 17 HRSD items before ABM. This network highlights possible pathways between depression symptoms – in other words, the symptomatological “landscape” which ABM in some way must operate in. Node placement was determined by Fruchterman and Reingold’s (1991) algorithm, whereby nodes nearer to the center of the graph tend to have the strongest connections with other nodes. A thicker edge denotes a larger association. Green edges represent positive partial correlations, whereas red ones represent negative partial correlations.

We estimated two networks based on symptom change scores from baseline to post ABM (post ABM minus baseline). A positive symptom score indicates an improvement in symptom

severity from pre to post, and a negative score indicates a worsening in symptom severity from pre to post. These networks give a visual presentation of how changes in one symptom relates to changes in other symptoms. First, we estimated a symptom change network for the whole sample, including a node representing ABM condition (control = 0, training = 1). This network indicates whether ABM is related to changes in specific symptoms. Second, we estimated symptom change networks for each group (training vs. control) separately, indicating whether ABM induces a changed symptom cascade from baseline to post ABM. Node placement in these training and control networks were determined by the node layout from the baseline network, to ease comparison.

Finally, we estimated networks separately for each group (training vs. control), where nodes represented symptom scores post ABM. Node positioning in these networks were also according to the baseline network layout. We compared these networks in global strength, symptom centrality, and specific edge differences.

**Centrality indices.** For each network, we computed centrality indices to quantify the importance of each node (Opsahl, Agneessens, & Skvoretz, 2010). We focused on “*strength*” centrality, defined as the sum of the weights of the edges attached to that node, because previous network research in psychopathology has indicated that this is the most stable and reliable centrality metric (e.g., Beard et al., 2016; Bernstein, Heeren, & McNally, 2017). Higher values reflect greater centrality in the network. We created centrality plots that depict these values as *z*-scores for ease of interpretation.

We evaluated the stability of the centrality indices by using the R package *bootnet* (Epskamp et al., 2017) by implementing a subset bootstrap procedure (Costenbader & Valente, 2003). The procedure is described in the Supplementary Materials.

**Networks comparisons.** Network differences were evaluated based on global strength, defined as the weighted sum of the absolute connections within a network (Barrat, Barthélemy, Pastor-Satorras, & Vespignani, 2004). Higher values reflect greater interconnectivity among nodes. We used the *NetworkComparisonTest* (NCT) to test for differences between the training and control network in global strength. The NCT is a two-tailed permutation-based test in which the difference between two groups is calculated repeatedly (10,000 times) for randomly regrouped individuals (van Borkulo, 2016). This produces a distribution of values under the null hypothesis (i.e., assuming equality between the groups) that enables one to test whether the observed difference in global network strength differs significantly ( $p < .05$ ) between groups.

To clarify potential significant differences between groups, we also tested whether edge weights were significantly different between groups for each edge of the networks. To do so, we relied on permutation-based tests using NCT (van Borkulo, 2016), and applied the Holm-Bonferroni correction to control for the large number of tests (i.e., minimizing the risk of Type 1-error).

## Results

### Participants

The majority of the participants were women (70 %). Mean age for the whole sample was 40.9 ( $SD = 13.2$ ), and participants had on average experienced 4.1 ( $SD = 5$ ) depressive episodes. Mean HRSD score at baseline was 8.8 ( $SD = 5.6$ ). Twenty-seven percent of the participants used a serotonin specific/serotonin-norepinephrine reuptake inhibitor anti-depressant. Demographic and clinical characteristics of the participants are presented in *Table 1*. There were no significant differences between the training and control group, except that HDRS scores from baseline to post ABM improved by 1.1 points ( $SD = 5.3$ , corresponding to 12 % improvement) in the

training group and worsened by 0.5 points ( $SD = 4.6$ , corresponding to 6 % worsening) in the control group (Cohen's  $d = 0.32$ ), as reported by Jonassen et al. (2018).

### **Baseline network**

The baseline network is presented in *Figure S1* (Supplementary Materials available online). Depressed mood, interest, guilt, and anxiety have many strong edges and are centrally located in the network. Insomnia symptoms and other vegetative symptoms (e.g., weight loss, agitation, psychomotor retardation) are located in the periphery, and primarily connected to the rest of the network through anxiety. Centrality indices (*Figure S2*, Supplementary Materials available online) indicate that anxiety (somatic [1.84]), depressed mood (1.70), guilt (0.81), and interest (0.70) have high strength centrality, meaning that they are strongly connected to the rest of the network.

### **Symptom change networks**

*Figure 1* presents the symptom change network for the whole sample, and strength centrality for each node. The ABM node represents ABM condition (0 = control, 1 = training). The other nodes represent change scores from baseline to post ABM (positive score = improvement, negative score = worsening). This network indicates that ABM condition covaries with improvement in interest, that is, when ABM condition changes from control to training, interest improves. Improvement in interest is in turn related to improvement in other symptoms. Strength centrality index indicate that ABM is a relatively unimportant node (-0.60) compared to most other nodes in the network, but, strikingly, its connection is to what appears as the second most central node in the network (interest [2.12]).

We conducted additional network analyses (i.e., post-hoc) to examine whether changes in attentional bias scores were associated with changes in symptoms (see *Supplementary Materials Figure S9-14*). Overall, results showed that there were edges appearing between, on the one hand,

changes in positive vs. negative attentional bias and, on the other hand, anxiety (somatic) and insight.

*Figure 2* denotes symptoms change networks and strength centrality for each node in the training and control group respectively. Depressive mood, low interest, and anxiety are closely related in both networks. Global network strength was significantly weaker for the training network (0.09) than in the control network (2.28,  $p < .05$ ). Anxiety (somatic) is the most central symptom in the control network (2.32), and markedly more central than in the training network (0.24). Permutation-based tests of edge differences revealed significant differences between the two networks. Indeed, five of the edges present in the control network were absent in the training network. Specifically, interest was connected to guilt ( $p = .01$ ), anxiety (somatic) was connected with suicidal thoughts ( $p < .01$ ) and somatic symptoms ( $p < .01$ ), agitation was connected to loss of insight ( $p < .05$ ), and retardation with loss of libido ( $p < .05$ ). Guilt was also connected to anxiety (somatic) at borderline significance level ( $p = .05$ ). No other edge was statistically different between the networks.

### **Post ABM networks**

Symptom networks and strength centrality for training and control groups post ABM are shown in *Figure 3*. There was no statistical significant difference in network density ( $p = .62$ ) between the training network (Global strength = 5.58) and the control network (Global strength = 5.06). Centrality indices were highly similar. Permutation-based tests revealed several significant edge differences between the training and the control network. In the control network, guilt was more strongly connected to anxiety (somatic;  $p < .05$ ) and retardation ( $p < .05$ ), and depressed mood was more strongly connected to anxiety (psychic;  $p < .05$ ). The training network had two edges present which were not present in the control network: interest was connected to gastro-



intestinal symptoms ( $p < .05$ ), and retardation was connected to late insomnia ( $p < .05$ ). No other differences in edges were statistically significant.

### **Stability of the centrality indices**

Stability analyses of the strength centrality index for all networks are depicted in *Figure S3-S8* (Supplementary Materials). The average correlation remained high after 50 % of the cases were dropped, indicating stability in the centrality measure estimates (Epskamp et al., 2017).

## **Discussion**

This is the first study examining a randomized clinical trial using network analysis, and the first network analysis on the ABM's impacts. Depressed mood, interest, guilt, and anxiety were the most central symptoms before ABM, supporting the generalizability of the current sample (e.g., Boschloo, van Borkulo, Borsboom, & Schoevers, 2016; Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2014; Fried et al., 2016). ABM was associated with improvement in interest and involvement in everyday activities, which, in turn, was associated with improvement in other depression symptoms. Post hoc analyses suggested that changes in attentional bias for positive vs. negative stimuli was associated with changes in anxiety (somatic) and insight. Moreover, results suggested that ABM changes the symptom cascade: the training network was less densely connected, and connections between interest, anxiety, guilt, suicidal ideation, and somatic symptoms were absent, compared to the control network. However, ABM did not change the global network strength, albeit connections between anxiety, depressed mood, and guilt were reduced in the training network.

The aim of the present ABM procedure was to render patients more focused on positive social stimuli. It has been hypothesized that this mechanism may set in motion an implicit relearning of a range of emotional associations, where ambiguous events or stimuli are perceived

more positively, and thus increase patients' motivation to engage in their social environment (Harmer, Duman, & Cowen, 2017). Thus, as participants' attention is nudged towards positive aspects of everyday situations (e.g., a smiling face, an encouraging comment), it may increase the probability for prosocial interaction, which, in turn, may increase the frequency of positive feedback and ultimately reinforces approach behavior towards social interaction (for a review, see Fox, 2005). Although this interpretation remains speculative, the association between ABM and interest may reflect the endpoint of this mechanism.

On the other hand, post-hoc analyses showed that changes in attentional bias (as measured by the dot-probe task) were not associated with interest. However, like most extant procedures for assessing attentional bias, the dot-probe task exhibits poor psychometric properties (for a review, see McNally, 2018). Recently developed experimental paradigms enabling optimal assessment of attentional bias might be more appropriate in future research agendas (e.g., Price et al., 2015; Sanchez-Lopez, Vanderhasselt, Allaert, Baeken, & De Raedt, 2018; Zvielli, Bernstein, & Koster, 2014).

An alternative interpretation is that interest is a moderator of the ABM effect. This hypothesis is in keeping with Shiroma, Thuras, Johns, and Lim (2014) who found that antidepressant response and remission was predicted by early changes in emotional processing of faces when considered along with perceived social support. Thus, helping patients to approach social situations during training may increase the effect of ABM.

Guilt has been found to be the most important symptom in explaining differences in overall network connectivity between persisters and remitters (van Borkulo et al., 2015). We found that ABM reduces connectivity between guilt and other depression symptoms. The guilt item in HRSD refers to "self-reproach and ideas of guilt or rumination over past errors", and is thematically similar to the brooding facet of rumination (Treyner, Gonzalez, & Nolen-Hoeksema,

2003). Rumination has been identified as a critical mechanism of depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). It is related to negative emotional processing (Gotlib & Joormann, 2010) and decreased cognitive control (Yang, Cao, Shields, Teng, & Liu, 2017). Specifically, brooding is characterized by attention being more firmly held on negative compared to positive information (Southworth, Grafton, MacLeod, & Watkins, 2017). Moreover, rumination has been found to mediate the effect of ABM on depression symptoms (Yang et al., 2015). Thus, the decoupling of guilt may represent a reduced impact of rumination in the context of other depression symptoms.

The present study provides an important basis to formulate new hypotheses on how ABM may alter depression symptoms. Our findings suggest that ABM reduces residual depression by increasing interest and motivation for social interaction, which in turn buffers against anxiety symptoms and negative cognitions. Whether these changes are of a sufficient magnitude to prevent relapse of a depressive episode needs to be examined in follow-up studies.

ABM resulted in few changes related to depressed mood, compared to changes involving anxiety symptoms. This may be explained by the fact that attention was trained towards positive stimuli in the context of threatening or anxiety-related stimuli (i.e., angry and fearful faces). This observation is further corroborated by the complementary post hoc analyses of changes in attentional bias scores, showing that fostering attentional bias for positive stimuli in the presence of negative stimuli was associated with a reduction of anxiety symptoms. Training in a context of negative mood-related stimuli (e.g., sad faces) may result in more pronounced changes involving depressed mood. Since depressed mood is the most central symptom, it is possible that this can increase the overall effect of ABM.

On the other hand, post hoc analyses, showing that changes in anxiety were related to changes in attentional bias towards angry/fearful faces, also point to anxiety as a potential

mediator of ABM's beneficial impacts. Thus, a reduction in the orientation towards threatening stimuli may have improved anxiety symptoms, which, in turn, may have lead to improvements in other depression symptoms. Unfortunately, the exact sequence of change in the symptom cascade cannot be inferred from the present study and future experiments are clearly needed to do so.

The network approach offers a promising conceptual framework to personalized treatment (Fried et al., 2017). The present study invites the hypothesis that ABM may be more effective in patients where interest, anxiety symptoms and negative cognitions dominate the symptom network, and less effective in patients where other symptoms are central (e.g., sleep problems and vegetative symptoms). Estimating individual symptom networks based on intensive time-series data can provide a more fine grained evaluation of ABM effects in individuals (see for example Fisher, Reeves, Lawyer, Medaglia, & Rubel, 2017). Based on these estimations, one can identify symptom profiles for which ABM might be the most effective treatment approach.

Including non-symptom variables that are assumed to play a causal role in depression in the network models has the potential to bring more knowledge of how and for whom and ABM can alter depression course (Jones, Heeren, & McNally, 2017). Relevant variables here are rumination and attentional control, because these are regarded as core causal mechanisms of depression (De Raedt & Koster, 2010; Nolen-Hoeksema et al., 2008), and are related to attentional bias change (Arditte & Joormann, 2014; Basanovic, Notebaert, Grafton, Hirsch, & Clarke, 2017) and symptom relief (Yang et al., 2015) after ABM.

The present study has several limitations. First, to the best of our knowledge, this is the first study considering an experimental condition within a network of continuous measures. Thus, the statistical adequacy of this approach has yet to be established. Emerging computational tools enabling the combination of categorical and continuous variables within a network, such as mixed graphical network modelling (Haslbeck, 2015), may therefore be more appropriate for

future research in the field. Second, the edges were calculated with cross-sectional data, precluding strong inference regarding cause-effect relationships among the variables (Maurage, Heeren, & Pesenti, 2013). Cross-sectional edges represent both within- and between-subjects effects that cannot be disentangled, and the network trajectory over time may vary across individuals. Third, symptom change networks were estimated based on individual difference scores, making the assumption that there would be no symptom changes in the absence of intervention. An alternative model would be to assume that symptom scores at post ABM is a linear function of the symptom scores at baseline (i.e., residual change scores; for a discussion, see Gollwitzer, Christ, & Lemmer, 2014). However, to best capture the within- and between-person temporal dynamics of individual networks, one would need to apply graphical vector autoregressive modeling approaches on intensive time-series data from individual participants (e.g., Wichers, Groot, & Psychosystems, 2016; Wild et al., 2010). In this way, one can track change in symptoms and attentional bias over time to optimally model the continuous dynamics of the interplay between the different changes occurring during ABM. Fourth, although a sample size of 302 patients is usually not regarded as a small sample for a clinical study in depression, network models estimate a very large number of parameters, and cross-sample validations in larger samples are thus required to draw firm conclusions. On the other hand, results from the non-parametric bootstrap approach reinforce the generalizability of the present findings in similar samples. Fifth, as highlighted by Jonassen et al. (2018), uncertainty still abounds regarding the best way to interpret the relevance of the rather small improvement in HRSD score in the present sample (that is, among patients who are not currently fulfilling diagnostic criteria for depression). We cannot exclude that the present effects on symptom networks would have been different with larger effects size. Finally, this study cannot determine whether the observed network changes are specific to ABM, or could be the result of any form of intervention. Other forms of treatments

(i.e., psychological, pharmacological) may yield very different impacts on the symptoms network. Moreover, each patient's particular network structure may point to different treatments, or a combination of treatments. Future research is thus clearly needed to clarify these issues.

In conclusion, by applying a computational network approach, we found that ABM improved depression by reducing the adverse impact of anxiety symptoms and negative cognitions on other depression symptoms. This change was associated with increased interest and involvement in everyday activities.

#### Author contributions

BK developed the study concept. BK and RJ collected the data. BK performed the data analysis and interpretation under the supervision of AH. BK and AH drafted the paper, and RJ, CH, TS and NIL provided critical revisions. All authors approved the final version of the paper for submission.

## References

- Arditte, K. A., & Joormann, J. (2014). Rumination Moderates the Effects of Cognitive Bias Modification of Attention. *Cognitive therapy and research*, *38*(2), 189-199.  
doi:10.1007/s10608-013-9581-9
- Barrat, A., Barthélemy, M., Pastor-Satorras, R., & Vespignani, A. (2004). The architecture of complex weighted networks. *Proceedings of the National Academy of Sciences of the United States of America*, *101*(11), 3747-3752. doi:10.1073/pnas.0400087101
- Basanovic, J., Notebaert, L., Grafton, B., Hirsch, C. R., & Clarke, P. J. F. (2017). Attentional control predicts change in bias in response to attentional bias modification. *Behaviour Research and Therapy*, *99*, 47-56. doi:10.1016/j.brat.2017.09.002
- Beard, C., Millner, A. J., Forgeard, M. J. C., Fried, E. I., Hsu, K. J., Treadway, M. T., . . . Bjorgvinsson, T. (2016). Network analysis of depression and anxiety symptom relationships in a psychiatric sample. *Psychological medicine*, *46*(16), 3359-3369.  
doi:10.1017/S0033291716002300
- Beck, A. T., & Bredemeier, K. (2016). A Unified Model of Depression. *Clinical Psychological Science*, *4*(4), 596-619. doi:10.1177/2167702616628523
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck depression inventory-II*. San Antonio, TX: Psychological Corporation.
- Beevers, C. G., Clasen, P. C., Enock, P. M., & Schnyer, D. M. (2015). Attention bias modification for major depressive disorder: Effects on attention bias, resting state connectivity, and symptom change. *Journal of abnormal psychology*, *124*(3), pp. doi:10.1037/abn0000049 25894440

- Bernstein, E. E., Heeren, A., & McNally, R. J. (2017). Unpacking Rumination and Executive Control: A Network Perspective. *Clinical Psychological Science, 5*(5), 816-826.  
doi:10.1177/2167702617702717
- Borgatti, S. P. (2005). Centrality and network flow. *Social Networks, 27*(1), 55-71.  
doi:10.1016/j.socnet.2004.11.008
- Borsboom, D. (2017). A network theory of mental disorders. *World Psychiatry, 16*(1), 5-13.  
doi:10.1002/wps.20375
- Borsboom, D., & Cramer, A. O. J. (2013). Network analysis: An integrative approach to the structure of psychopathology. *Annual review of clinical psychology, 91*-121.  
doi:10.1146/annurev-clinpsy-050212-185608
- Boschloo, L., van Borkulo, C. D., Borsboom, D., & Schoevers, R. A. (2016). A prospective study on how symptoms in a network predict the onset of depression. *Psychotherapy and Psychosomatics, 85*(3), 183-184. doi:10.1159/000442001
- Bringmann, L. F., Lemmens, L. H. J. M., Huibers, M. J. H., Borsboom, D., & Tuerlinckx, F. (2014). Revealing the dynamic network structure of the Beck Depression Inventory-II. *Psychological medicine, 45*(4), 747-757. doi:10.1017/S0033291714001809
- Browning, M., Holmes, E. A., Charles, M., Cowen, P. J., & Harmer, C. J. (2012). Using Attentional Bias Modification as a Cognitive Vaccine Against Depression. *Biological Psychiatry, 72*(7), 572-579. doi:10.1016/j.biopsych.2012.04.014
- Costenbader, E., & Valente, T. W. (2003). The stability of centrality measures when networks are sampled. *Social Networks, 25*(4), 283-307. doi:10.1016/S0378-8733(03)00012-1
- Cramer, A. O., van Borkulo, C. D., Giltay, E. J., van der Maas, H. L., Kendler, K. S., Scheffer, M., & Borsboom, D. (2016). Major Depression as a Complex Dynamic System. *PLoS ONE, 11*(12), e0167490. doi:10.1371/journal.pone.0167490



- Cristea, I. A., Kok, R. N., & Cuijpers, P. (2015). Efficacy of cognitive bias modification interventions in anxiety and depression: meta-analysis. *Br J Psychiatry*, *206*(1), 7-16.  
doi:10.1192/bjp.bp.114.146761
- Cristea, I. A., Kok, R. N., & Cuijpers, P. (2017). Confusing procedures with process in cognitive bias modification research. *The British Journal of Psychiatry*, *211*(5), 272.
- Cuijpers, P., & Schoevers, R. A. (2004). Increased mortality in depressive disorders: a review. *Curr Psychiatry Rep*, *6*(6), 430-437.
- 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.  
doi:10.3758/CABN.10.1.50
- Demyttenaere, K., Bruffaerts, R., Posada-Villa, J., Gasquet, I., Kovess, V., Lepine, J. P., . . . WHO. (2004). Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. *JAMA*, *291*(21), 2581-2590. doi:10.1001/jama.291.21.2581
- Disner, S. G., Beevers, C. G., Haigh, E. A. P., & Beck, A. T. (2011). Neural mechanisms of the cognitive model of depression. *Nat Rev Neurosci*, *12*(8), 467-477.
- Epskamp, S., Borsboom, D., & Fried, E. I. (2017). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*. doi:10.3758/s13428-017-0862-1
- Epskamp, S., Cramer, A. O. J., Waldorp, L. J., Schmittmann, V. D., & Borsboom, D. (2012). qgraph: Network Visualizations of Relationships in Psychometric Data. *2012*, *48*(4), 18.  
doi:10.18637/jss.v048.i04
- Epskamp, S., & Fried, E. I. (2018). A tutorial on regularized partial correlation networks. *Psychological Methods*(Pagination). doi:10.1037/met0000167

- Fisher, A. J., Reeves, J. W., Lawyer, G., Medaglia, J. D., & Rubel, J. A. (2017). Exploring the idiographic dynamics of mood and anxiety via network analysis. *Journal of abnormal psychology, 126*(8), 1044-1056. doi:10.1037/abn0000311
- Fox, E. (2005). The role of visual processes in modulating social interactions. *Visual Cognition, 12*(1), 1-11. doi:10.1080/13506280444000067
- Foygel, R., & Drton, M. (2011). *Bayesian model choice and information criteria in sparse generalized linear models*. arXiv:1112.5635. Retrieved from <https://arxiv.org/abs/1112.5635>
- Fried, E. I., Epskamp, S., Nesse, R. M., Tuerlinckx, F., & Borsboom, D. (2016). What are 'good' depression symptoms? Comparing the centrality of DSM and non-DSM symptoms of depression in a network analysis. *Journal of affective disorders, 189*, 314-320. doi:10.1016/j.jad.2015.09.005 26458184
- Fried, E. I., & Nesse, R. M. (2015). Depression sum-scores don't add up: why analyzing specific depression symptoms is essential. *BMC Med, 13*, 72. doi:10.1186/s12916-015-0325-4
- Fried, E. I., Nesse, R. M., Zivin, K., Guille, C., & Sen, S. (2014). Depression is more than the sum score of its parts: individual DSM symptoms have different risk factors. *Psychological medicine, 44*(10), 2067-2076. doi:10.1017/S0033291713002900
- Fried, E. I., van Borkulo, C. D., Cramer, A. O. J., Boschloo, L., Schoevers, R. A., & Borsboom, D. (2017). Mental disorders as networks of problems: A review of recent insights. *Social Psychiatry and Psychiatric Epidemiology, 52*(1), 1-10. doi:10.1007/s00127-016-1319-z
- Friedman, J., Hastie, T., & Tibshirani, R. (2008). Sparse inverse covariance estimation with the graphical lasso. *Biostatistics, 9*(3), 432-441. doi:10.1093/biostatistics/kxm045

- Gollwitzer, M., Christ, O., & Lemmer, G. (2014). Individual differences make a difference: On the use and the psychometric properties of difference scores in social psychology. *European Journal of Social Psychology*, *44*(7), pp. doi:10.1002/ejsp.2042
- Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: current status and future directions. *Annual review of clinical psychology*, *6*, 285.  
doi:10.1146/annurev.clinpsy.121208.131305
- Gotlib, I. H., Krasnoperova, E., Yue, D. N., & Joormann, J. (2004). Attentional Biases for Negative Interpersonal Stimuli in Clinical Depression. *Journal of abnormal psychology*, *113*(1), 127-135. doi:10.1037/0021-843X.113.1.121
- Grafton, B., MacLeod, C., Rudaizky, D., Holmes, E. A., Salemink, E., Fox, E., & Notebaert, L. (2017). Confusing procedures with process when appraising the impact of cognitive bias modification on emotional vulnerability. *The British Journal of Psychiatry*, *211*(5), 266.
- Hakamata, Y., Lissek, S., Bar-Haim, Y., Britton, J. C., Fox, N. A., Leibenluft, E., . . . Pine, D. S. (2010). Attention Bias Modification Treatment: A meta-analysis toward the establishment of novel treatment for anxiety. *Biological Psychiatry*, *68*(11), 982-990.  
doi:10.1016/j.biopsych.2010.07.021
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery & Psychiatry*, 56-61. doi:10.1136/jnnp.23.1.56
- Harmer, C. J., Duman, R. S., & Cowen, P. J. (2017). How do antidepressants work? New perspectives for refining future treatment approaches. *The Lancet Psychiatry*, *4*(5), 409-418. doi:10.1016/S2215-0366(17)30015-9
- Haslbeck, J. M. B. W., L. J. (2015). Structure estimation for mixed graphical models in high-dimensional data. *arXiv (Preprint: 1510.05677)*. doi:<https://arxiv.org/abs/1510.05677>

- Heeren, A., & McNally, R. J. (2018). Social Anxiety Disorder as a Densely Interconnected Network of Fear and Avoidance for Social Situations. *Cognitive therapy and research*. doi:10.1007/s10608-017-9876-3
- Hofmann, S. G., & Curtiss, J. (2018). A complex network approach to clinical science. *Eur J Clin Invest*, 48(8), e12986. doi:10.1111/eci.12986
- Hofmann, S. G., Curtiss, J., & McNally, R. J. (2016). A complex network perspective on clinical science. *Perspectives on psychological science*, 11(5), pp. doi:10.1177/1745691616639283 27694457
- Jonassen, R., Harmer, C. J., Hilland, E., Maglanoc, L. A., Kraft, B., Browning, M., . . . Landro, N. I. (2018). Effects of Attentional Bias Modification on Residual Symptoms in depression. A Randomized Controlled Trial. *bioRxiv (Preprint: 318279)*. doi:10.1101/318279
- Jones, P. J., Heeren, A., & McNally, R. J. (2017). Commentary: A network theory of mental disorders. *Frontiers in Psychology*, 8, 1305. doi:10.3389/fpsyg.2017.01305
- Joormann, J., & Gotlib, I. H. (2007). Selective attention to emotional faces following recovery from depression. *J Abnorm Psychol*, 116(1), 80-85. doi:10.1037/0021-843X.116.1.80
- Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of abnormal psychology*, 116(1), 135-143. doi:10.1037/0021-843x.116.1.135
- Koster, E. H. W., & Bernstein, A. (2015). Introduction to the special issue on Cognitive bias modification: Taking a step back to move forward? *J Behav Ther Exp Psychiatry*, 49(Pt A), 1-4. doi:10.1016/j.jbtep.2015.05.006
- Kujawa, A. J., Torpey, D., Kim, J., Hajcak, G., Rose, S., Gotlib, I. H., & Klein, D. N. (2011). Attentional biases for emotional faces in young children of mothers with chronic or

- recurrent depression. *J Abnorm Child Psychol*, 39(1), 125-135. doi:10.1007/s10802-010-9438-6
- Lundqvist, D., Flykt, A., & Öhman, A. (1998). The Karolinska Directed Emotional Faces - KDEF [CD-ROM]. Department of Clinical Neuroscience, Psychology section, Karolinska Institutet.
- Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annu Rev Clin Psychol*, 1(1), 167-195. doi:10.1146/annurev.clinpsy.1.102803.143916
- Maurage, P., Heeren, A., & Pesenti, M. (2013). Does chocolate consumption really boost Nobel Award chances? The peril of over-interpreting correlations in health studies. *J Nutr*, 143(6), 931-933. doi:10.3945/jn.113.174813
- McNally, R. J. (2016). Can network analysis transform psychopathology? *Behaviour Research and Therapy*, 86, 95-104. doi:10.1016/j.brat.2016.06.006
- McNally, R. J. (2018). Attentional bias for threat: Crisis or opportunity? *Clinical psychology review*. doi:10.1016/j.cpr.2018.05.005
- Mogg, K., Waters, A. M., & Bradley, B. P. (2017). Attention Bias Modification (ABM): Review of Effects of Multisession ABM Training on Anxiety and Threat-Related Attention in High-Anxious Individuals. *Clin Psychol Sci*, 5(4), 698-717. doi:10.1177/2167702617696359
- Mogoase, C., David, D., & Koster, E. H. (2014). Clinical efficacy of attentional bias modification procedures: an updated meta-analysis. *J Clin Psychol*, 70(12), 1133-1157. doi:10.1002/jclp.22081
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on psychological science*, 3(5), 400-424. doi:10.1111/j.1745-6924.2008.00088.x

- Opsahl, T., Agneessens, F., & Skvoretz, J. (2010). Node centrality in weighted networks: Generalizing degree and shortest paths. *Social Networks*, *32*(3), 245-251.  
doi:10.1016/j.socnet.2010.03.006
- Paykel, E. S. (2008). Partial remission, residual symptoms, and relapse in depression. *Dialogues in Clinical Neuroscience*, *10*(4), 431-437.
- Pe, M. L., Kircanski, K., Thompson, R. J., Bringmann, L. F., Tuerlinckx, F., Mestdagh, M., . . . Gotlib, I. H. (2015). Emotion-Network Density in Major Depressive Disorder. *Clinical Psychological Science*, *3*(2), 292-300. doi:10.1177/2167702614540645
- Peckham, A. D., McHugh, R. K., & Otto, M. W. (2010). A meta-analysis of the magnitude of biased attention in depression. *Depression and Anxiety*, *27*(12), 1135-1142.  
doi:10.1002/da.20755
- Price, R. B., Kuckertz, J. M., Siegle, G. J., Ladouceur, C. D., Silk, J. S., Ryan, N. D., . . . Amir, N. (2015). Empirical recommendations for improving the stability of the dot-probe task in clinical research. *Psychol Assess*, *27*(2), 365-376. doi:10.1037/pas0000036
- Sanchez-Lopez, A., Vanderhasselt, M.-A., Allaert, J., Baeken, C., & De Raedt, R. (2018). Neurocognitive mechanisms behind emotional attention: Inverse effects of anodal tDCS over the left and right DLPFC on gaze disengagement from emotional faces. *Cognitive, Affective, & Behavioral Neuroscience*, *18*(3), 485-494. doi:10.3758/s13415-018-0582-8
- Schweren, L., van Borkulo, C. D., Fried, E., & Goodyer, I. M. (2017). Assessment of symptom network density as a prognostic marker of treatment response in adolescent depression. *JAMA Psychiatry*. doi:10.1001/jamapsychiatry.2017.3561
- 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. *J Clin Psychiatry*, 59 Suppl 20, 22-33;quiz 34-57.
- Shiroma, P. R., Thuras, P., Johns, B., & Lim, K. O. (2014). Emotion recognition processing as early predictor of response to 8-week citalopram treatment in late-life depression. *International Journal of Geriatric Psychiatry*, 29(11), 1132-1139. doi:10.1002/gps.4104
- Southworth, F., Grafton, B., MacLeod, C., & Watkins, E. (2017). Heightened ruminative disposition is associated with impaired attentional disengagement from negative relative to positive information: support for the “impaired disengagement” hypothesis. *Cognition and Emotion*, 31(3), 422-434. doi:10.1080/02699931.2015.1124843
- Teasdale, J. D. (1988). Cognitive Vulnerability to Persistent Depression. *Cognition and Emotion*, 2(3), 247-274. doi:10.1080/02699938808410927
- Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination Reconsidered: A Psychometric Analysis. *Cognitive therapy and research*, 27(3), 247-259. doi:10.1023/a:1023910315561
- Valente, T. W. (2012). Network interventions. *Science*, 337(6090), 49-53. doi:10.1126/science.1217330
- van Borkulo, C. (2016). NetworkComparisonTest. Retrieved from <https://cran.r-project.org/web/packages/NetworkComparisonTest/>
- van Borkulo, C., Boschloo, L., Borsboom, D., Penninx, B. H., Waldorp, L. J., & Schoevers, R. A. (2015). Association of symptom network structure with the course of depression. *JAMA Psychiatry*, 72(12), 1219-1226. doi:10.1001/jamapsychiatry.2015.2079
- Wells, T. T., Beevers, C. G., Id, & Wells, T. T. O. h. o. o. X. (2010). Biased attention and dysphoria: Manipulating selective attention reduces subsequent depressive symptoms. *Cognition and Emotion*, .24(4), pp. doi:10.1080/02699930802652388

- Wichers, M. (2014). The dynamic nature of depression: a new micro-level perspective of mental disorder that meets current challenges. *Psychol Med*, *44*(7), 1349-1360.  
doi:10.1017/S0033291713001979
- Wichers, M., Groot, P. C., & Psychosystems. (2016). Critical Slowing Down as a Personalized Early Warning Signal for Depression. *Psychotherapy and Psychosomatics*, *85*(2), 114-116. doi:10.1159/000441458
- Wild, B., Eichler, M., Friederich, H.-C., Hartmann, M., Zipfel, S., & Herzog, W. (2010). A graphical vector autoregressive modelling approach to the analysis of electronic diary data. *BMC Medical Research Methodology*, *10*(1), 28. doi:10.1186/1471-2288-10-28
- Yang, Cao, S., Shields, G. S., Teng, Z., & Liu, Y. (2017). The relationships between rumination and core executive functions: A meta-analysis. *Depress Anxiety*, *34*(1), 37-50.  
doi:10.1002/da.22539
- Yang, Ding, Z., Dai, T., Peng, F., & Zhang, J. X. (2015). Attention Bias Modification training in individuals with depressive symptoms: A randomized controlled trial. *J Behav Ther Exp Psychiatry*, *49*(Pt A), 101-111. doi:10.1016/j.jbtep.2014.08.005
- Zvielli, A., Bernstein, A., & Koster, E. H. W. (2014). Temporal Dynamics of Attentional Bias. *Clinical Psychological Science*, *3*(5), 772-788. doi:10.1177/2167702614551572

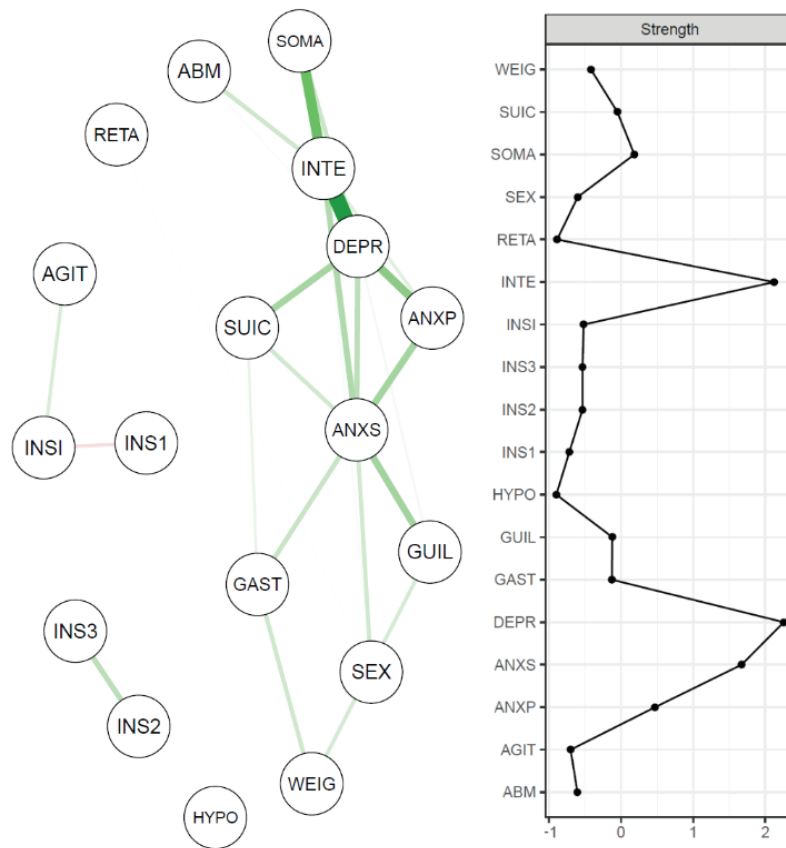


Table 1

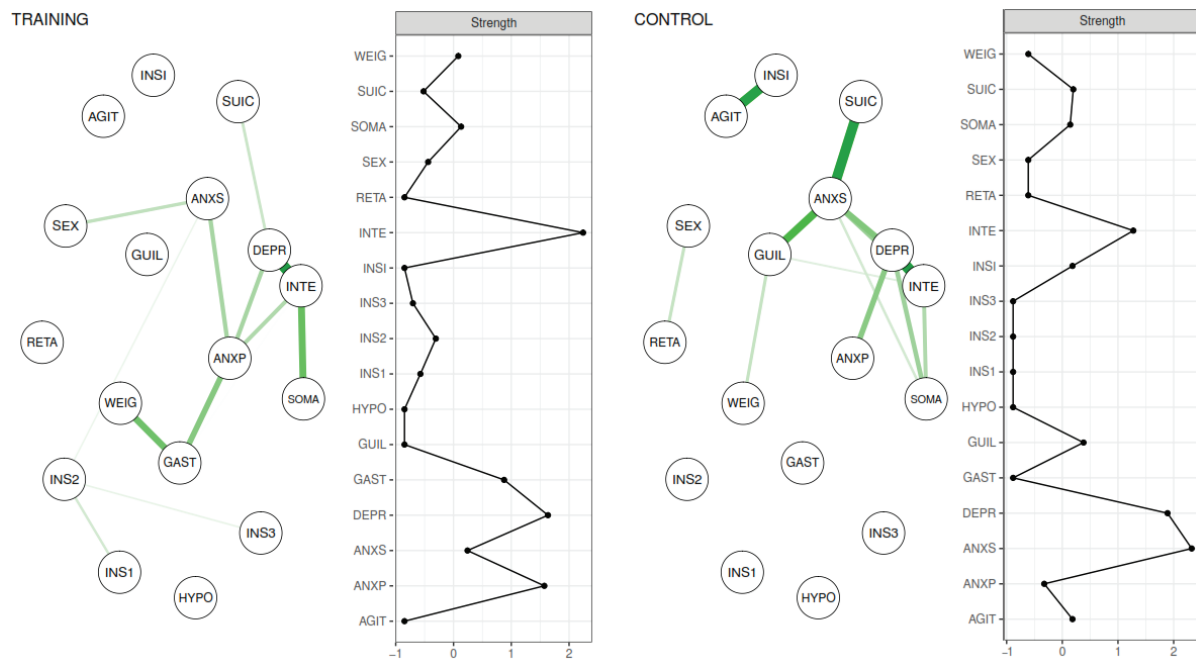
*Demographic and Clinical Characteristics*

Variables	Control ( <i>n</i> = 149)	Training ( <i>n</i> = 153)
Age, years, <i>M</i> ( <i>SD</i> )	41.5 (13.6)	40.2 (12.7)
Sex (females), <i>n</i>	103	109
Educational level (ISCED), <i>M</i> ( <i>SD</i> )	5.9 (1.2)	6.0 (1.1)
Use of SSRI/SNRI medication, <i>n</i>	43	38
Rumination score (RRS), <i>M</i> ( <i>SD</i> )	49.5 (12.2)	51.4 (12.1)
Stroop inhibition-switching, seconds to complete, <i>M</i> ( <i>SD</i> )	59.9 (15.5)	60.1 (14.4)
Number of depressive episodes, <i>M</i> ( <i>SD</i> )	4.1 (4.6)	4.1 (4.9)
Comorbidity rate	61%	62%
HRSD total score, <i>M</i> ( <i>SD</i> )		
baseline	8.3 (5.1)	9.2 (6.0)
post	8.8 (5.9)	8.3 (6.0)
pre-post change	-0.5 (4.6)	1.1 (5.3) *

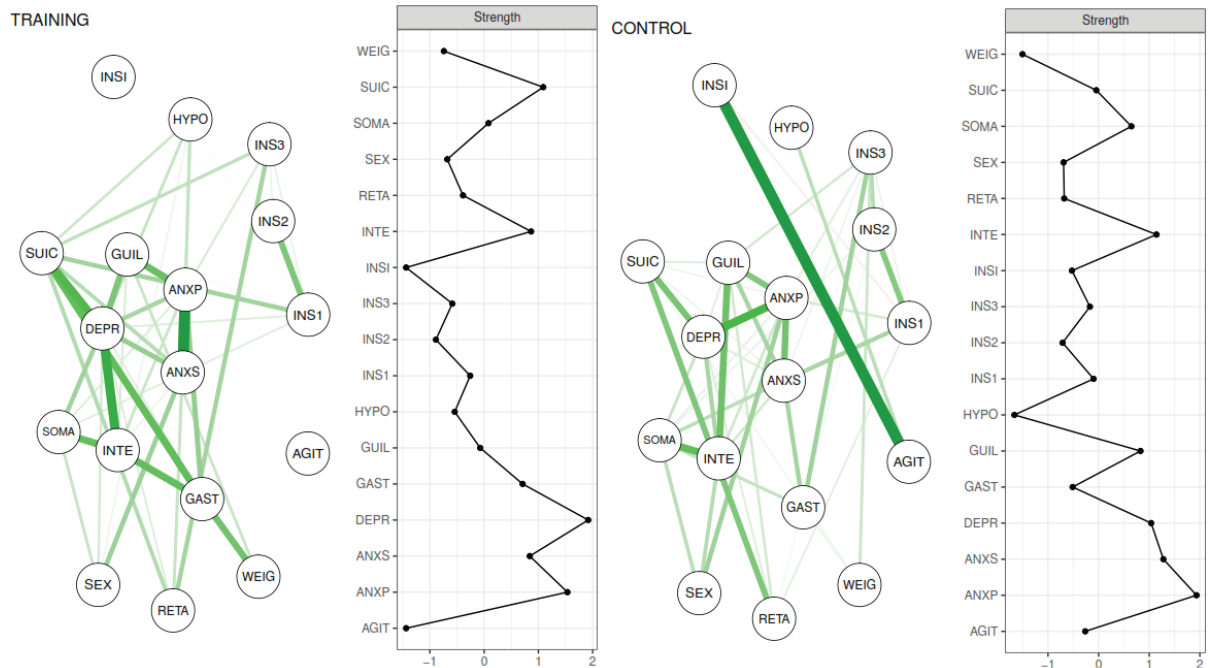
**Note.** SSRI/SNRI = serotonin specific/serotonin-norepinephrine reuptake inhibitor; HRSD = Hamilton Rating Scale for Depression; MDD = major depressive disorder; ISCED = International Standard Classification of Education; RRS = Ruminative Responses Scale; \* statistical significant difference ( $p < 0.01$ ) versus control group. All other differences (t-tests and chi-square tests) were not statistical significant.



**Figure 1.** Symptom change network and strength centrality for the whole sample. ABM = ABM condition (control = 0, training = 1). Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).



**Figure 2.** Symptom change networks and strength centrality for the training group (left) and the control group (right). Nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).



**Figure 3.** Post ABM symptom networks and strength centrality for the training group (left) and the control group (right). Nodes represent HRSD item scores post ABM (post minus baseline).

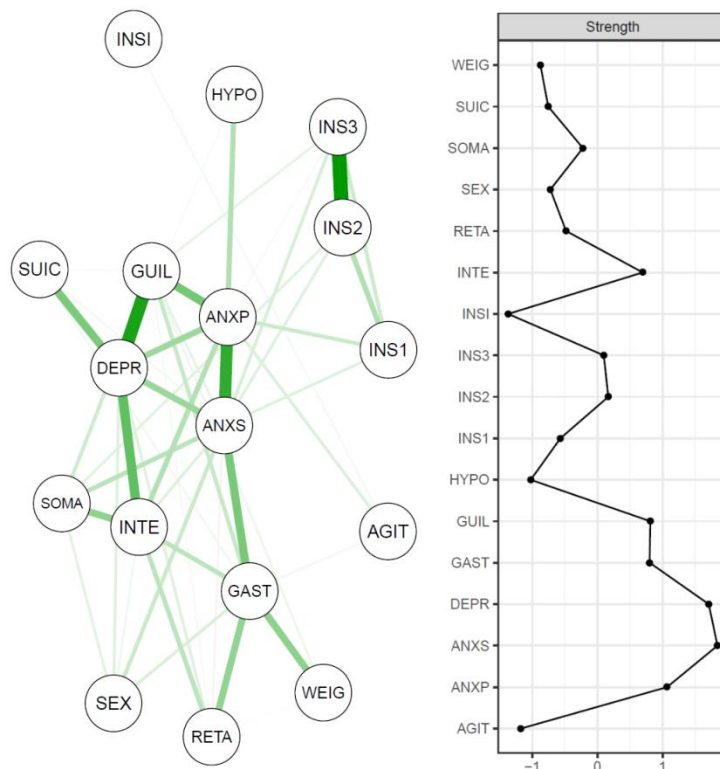
Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).

## Supplementary Materials

**Stability of centrality indices.** We evaluated the stability of the centrality indices by using the *R* package *bootnet* (Epskamp et al., 2017) by implementing a subset bootstrap procedure (Costenbader & Valente, 2003). To do so, we repeatedly correlated centrality metrics of the original dataset with centrality metrics calculated from a subsample of participants missing via person-dropping bootstraps as implemented. If correlation values decline substantially as participants are removed, then this centrality index would be considered as less stable. We set the bootstraps to 1,000 and plotted the centrality stability correlation coefficient (CS-coefficient) to quantify the effects of this person-dropping procedure. The CS-coefficient represents the maximum proportion of participants that can be dropped while maintaining 95% probability that the correlation between centrality metrics from the full data set and the subset data are at least .70. A minimum CS-coefficient of .25 is recommended for interpreting centrality indices (Epskamp et al., 2017).

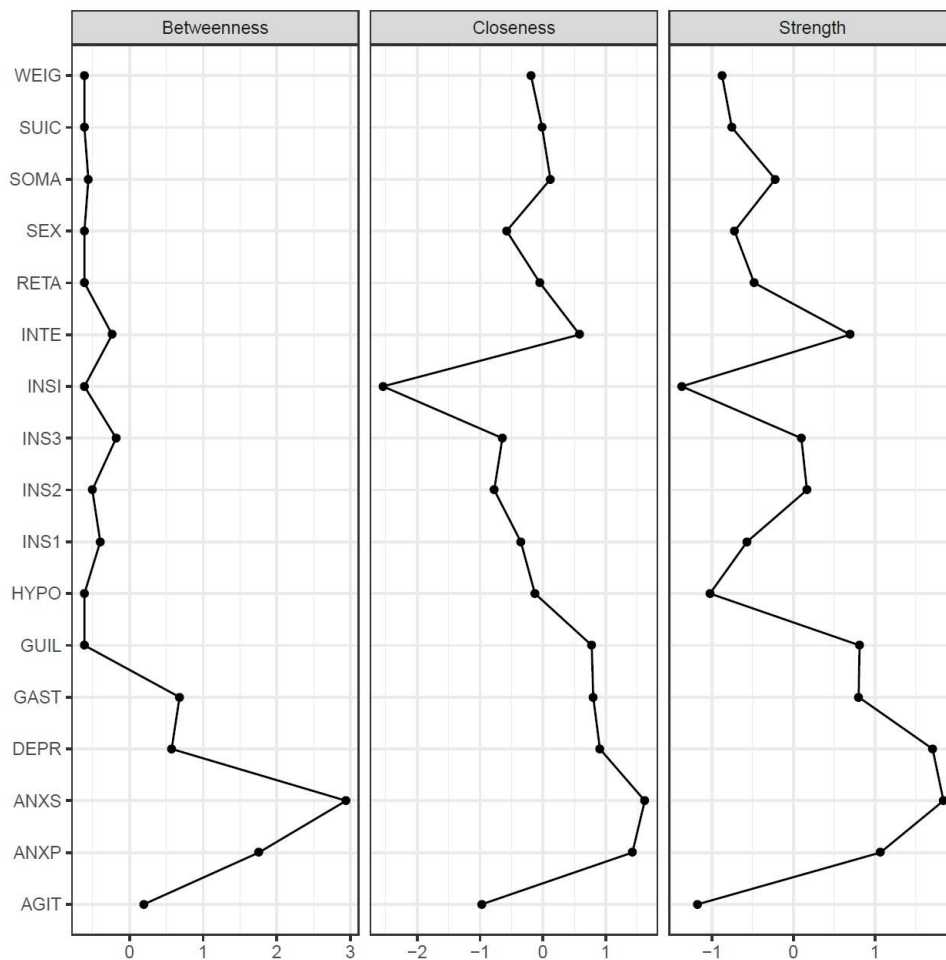
Costenbader, E., & Valente, T. W. (2003). The stability of centrality measures when networks are sampled. *Social Networks*, 25(4), 283-307. doi:10.1016/S0378-8733(03)00012-1

Epskamp, S., Borsboom, D., & Fried, E. I. (2017). Estimating psychological networks and their accuracy: A tutorial paper. *Behavior Research Methods*. doi:10.3758/s13428-017-0862-1

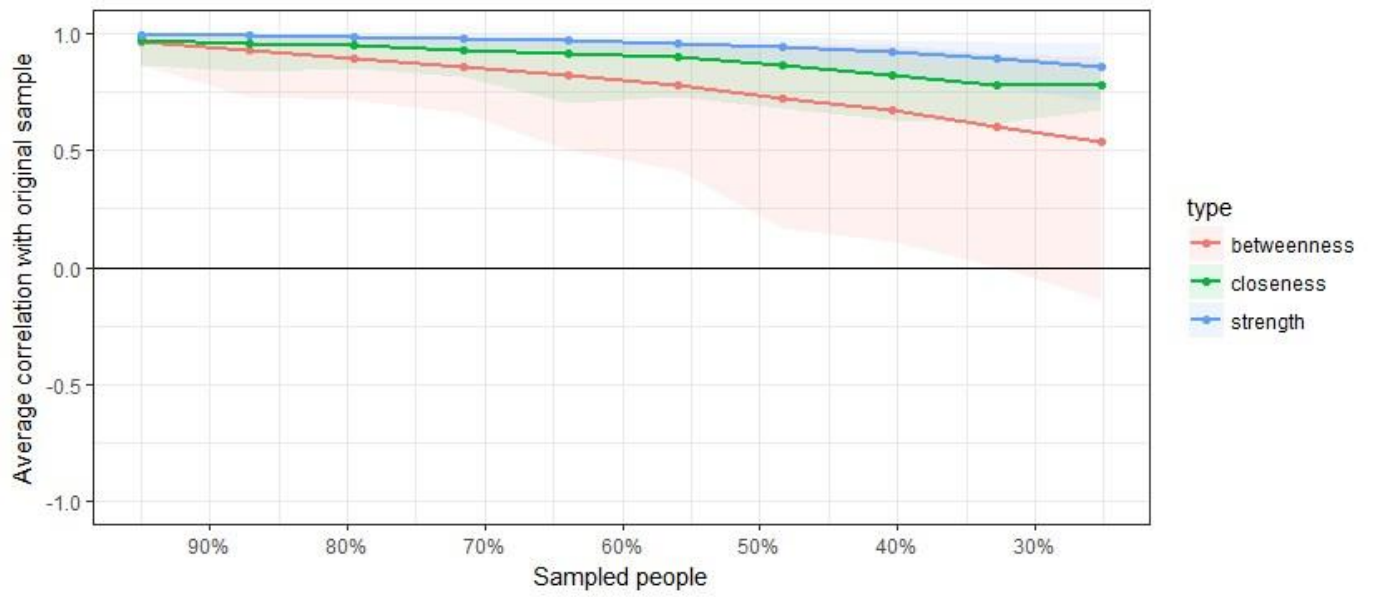


**Figure S1.** Baseline symptom network and strength centrality indices for the whole sample.

Nodes represent HRSD scores at baseline. Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).

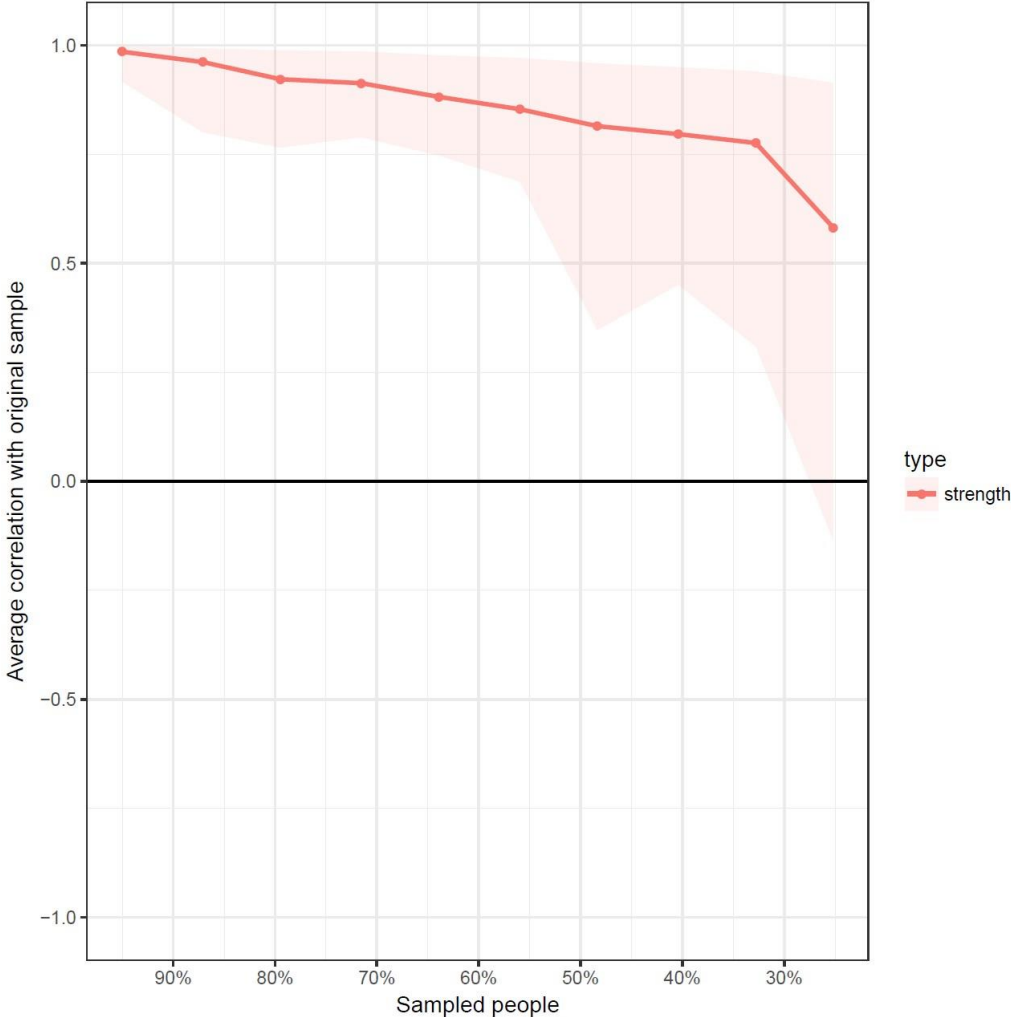


**Figure S2.** Centrality indices for baseline symptom network. Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss.

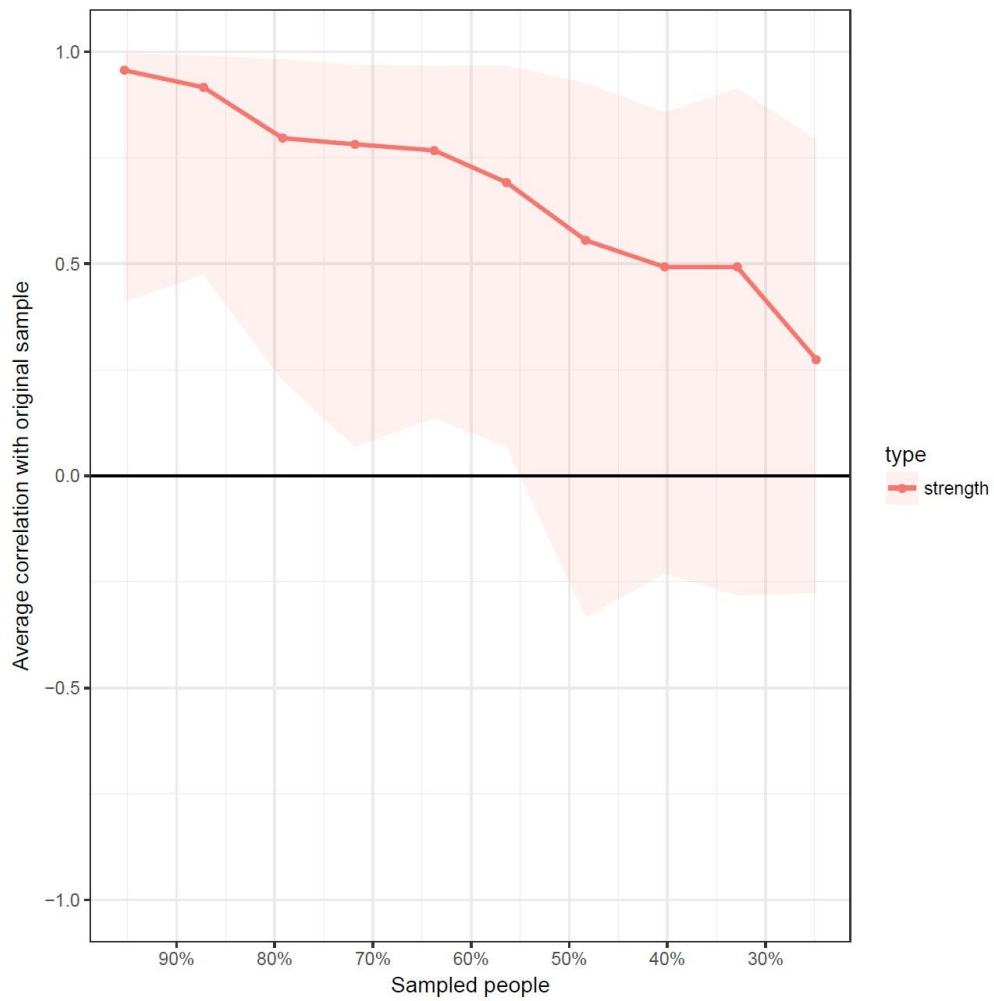


**Figure S3.** Stability analysis (person-drop) for the baseline symptom network.

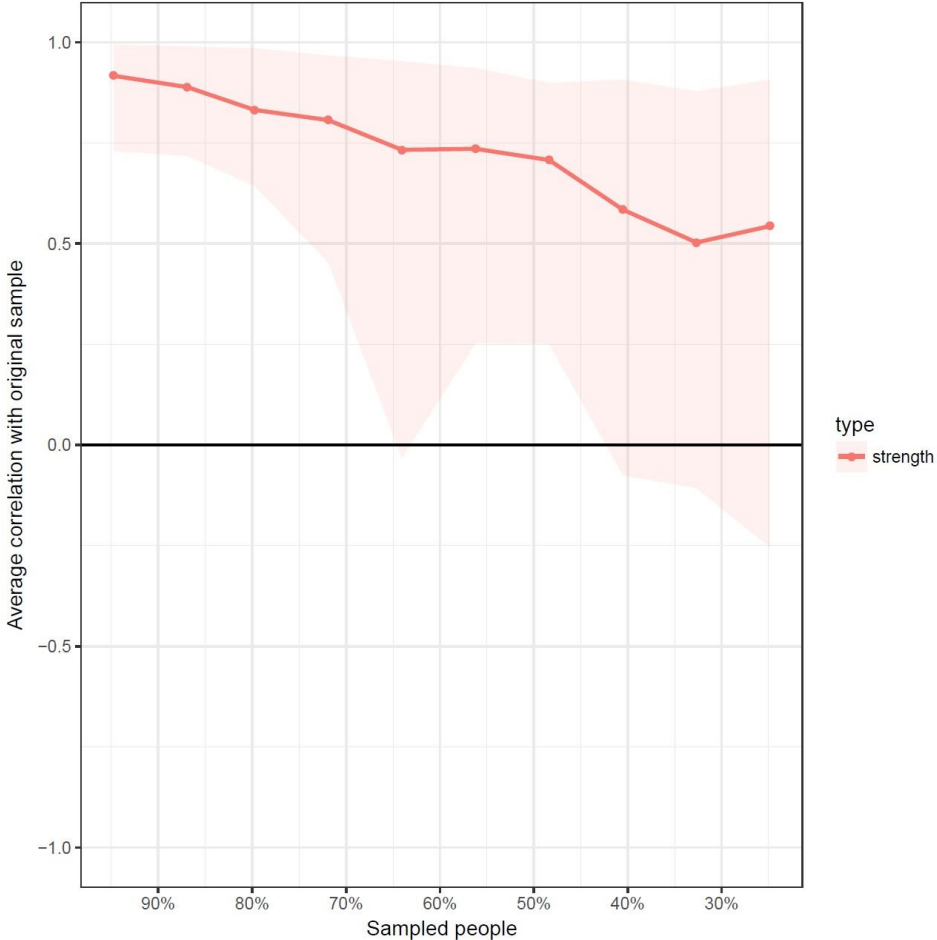




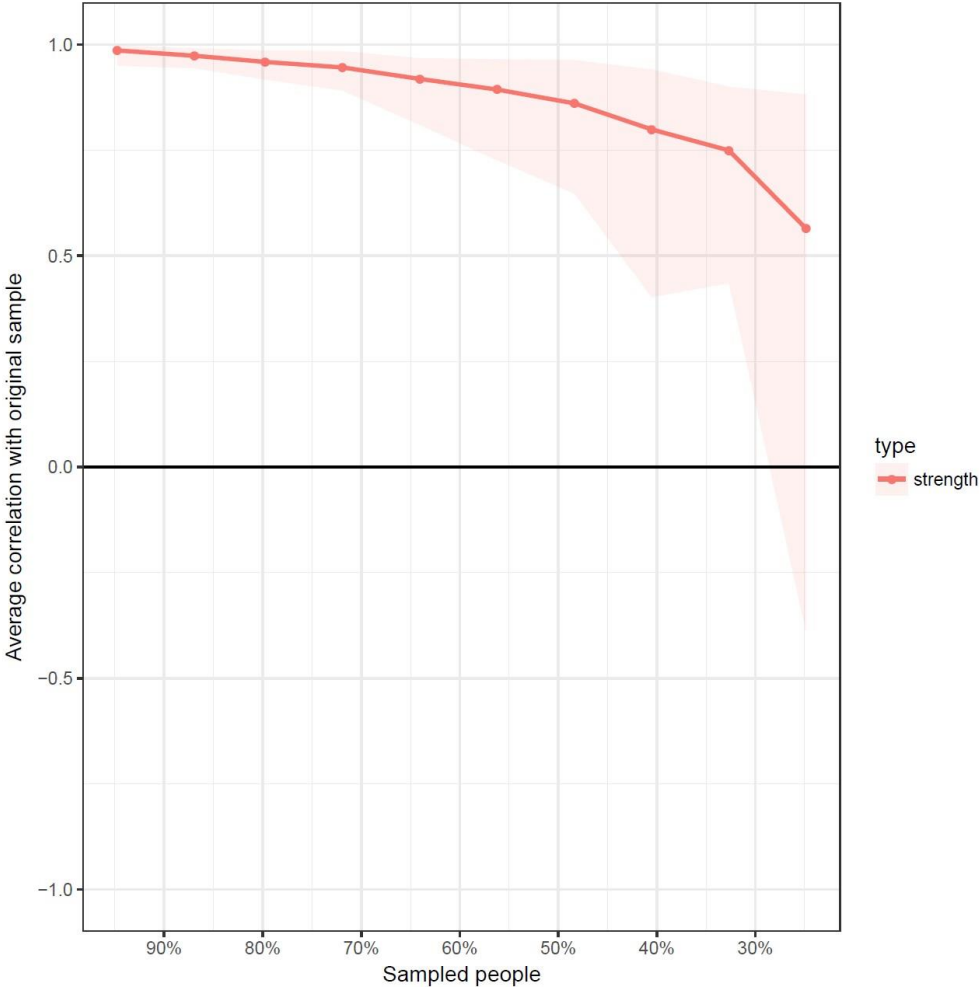
**Figure S4.** Stability analysis (person-drop) for the symptom change network.



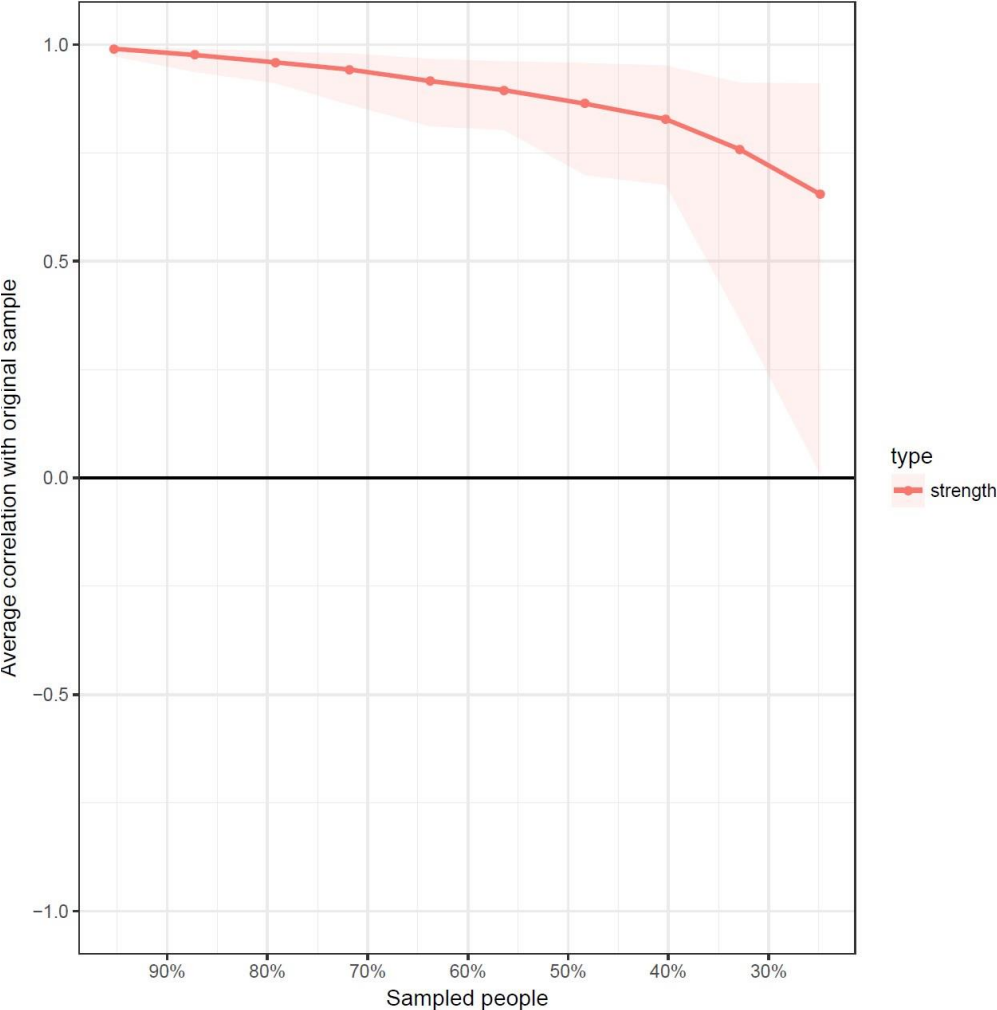
**Figure S5.** Stability analysis (person-drop) for the symptom change network in the control group.



**Figure S6.** Stability analysis (person-drop) for the symptom change network in the training group.



**Figure S7.** Stability analysis (person-drop) for the post ABM network in the training group.



**Figure S8.** Stability analysis (person-drop) for the post ABM network in the control group.

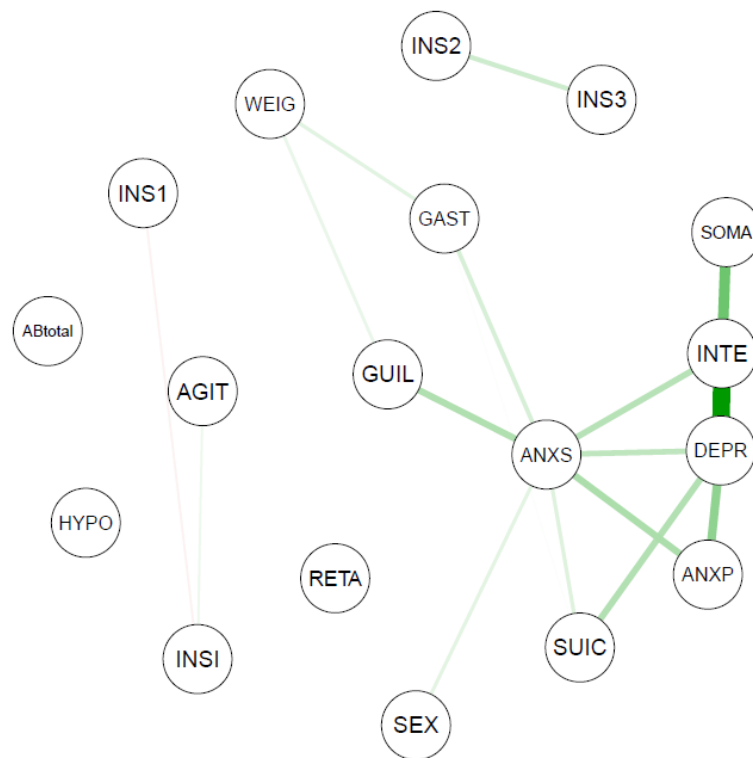
**Measurement of attentional bias**

Attentional bias was measured at baseline and after two weeks of ABM using a standard visual probe procedure (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002) consisting of 96 trials, with the same trial types as used in the ABM procedure. Novel facial stimuli were used in the assessment tasks. We calculated a total attentional bias score, and three valence-specific attentional bias scores based on the difference in reaction between trials in which the probe replaced the relatively more 1) negative face vs. the more positive face, 2) neutral face vs. the more positive face, 3) negative face vs. the more neutral face.

MacLeod, C., Rutherford, E., Campbell, L., Ebsworthy, G., & Holker, L. (2002). Selective attention and emotional vulnerability: Assessing the causal basis of their association through the experimental manipulation of attentional bias. *Journal of abnormal psychology, 111*(1). doi:10.1037/0021-843X.111.1.107 11866165

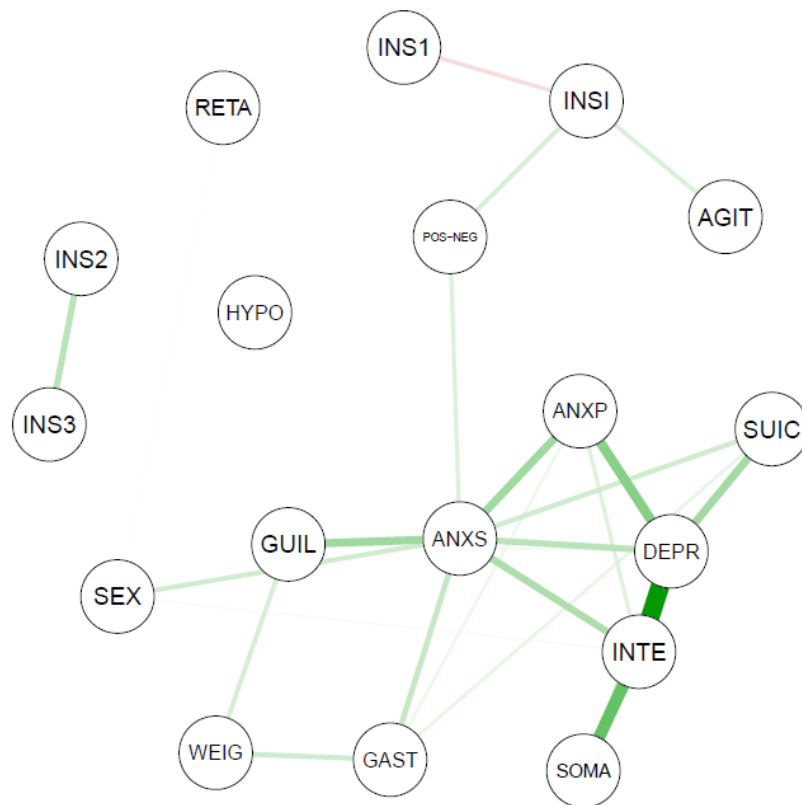
### **Associations between changes in specific attentional bias measures and symptom changes**

We conducted additional analyses to explore the specific contribution of attentional bias (AB) changes to symptom changes. First, we examined the contribution of total AB change on symptom changes (*Figure S9*). Second, we examined each valence-specific AB index separately (*Figure S10-12*). Third, to control for the general covariance among AB measures (i.e., general reaction time pre-post changes), we included all three AB change indices together (*Figure S13*). Finally, in an effort to delineate to what extent AB changes specifically resulting from the ABM intervention interplay with symptom changes, we also examined the role of valence-specific AB changes in the ABM training group only (*Figure S14*). Overall, the results suggest that only attentional bias for positive vs. negative was related to changes in symptom from pre to post ABM. However, when considering the ABM group only (*Figure S14*), there were no edges appearing between attentional bias changes and symptoms. However, the small sample size ( $n = 153$ ) greatly reduces sensitivity to detect edges.

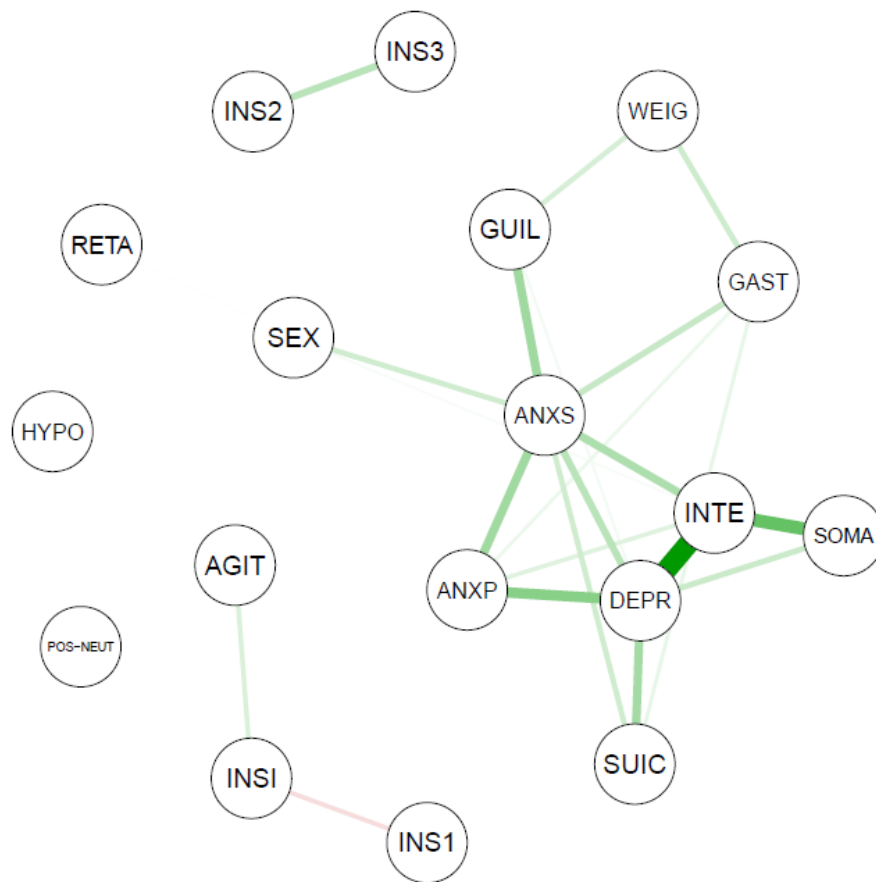


**Figure S9.** Symptom change network for the whole sample. ABtotal = changes in attentional bias (total) from pre to post ABM. Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).

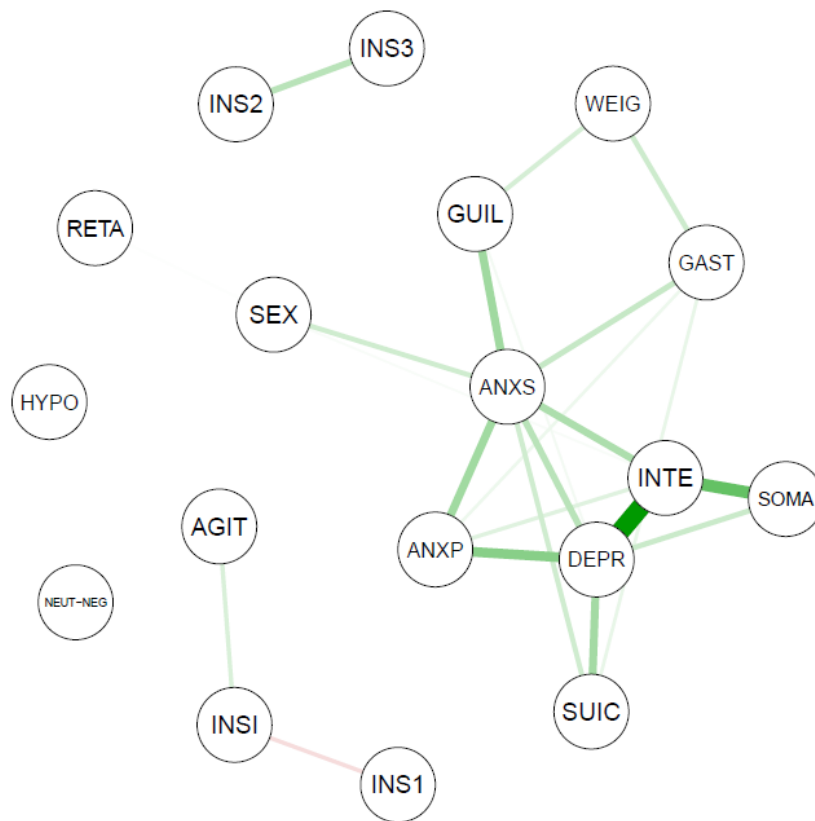




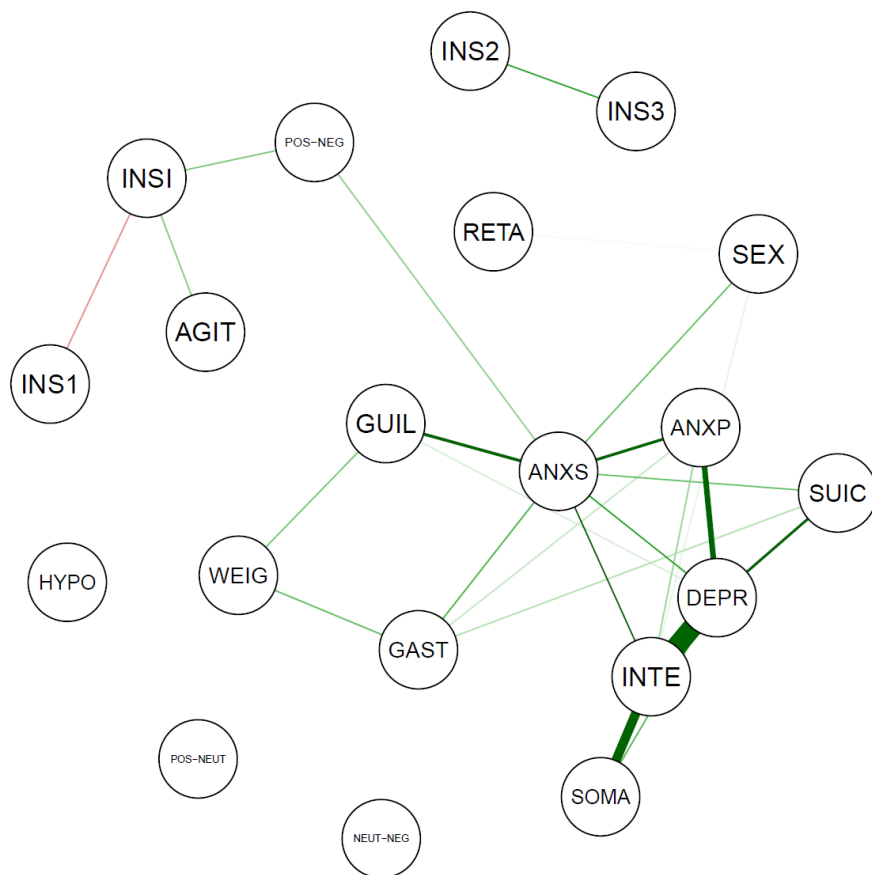
**Figure S10.** Symptom change network for the whole sample. pos-neg = changes in attentional bias for positive vs. negative stimuli from pre to post ABM. Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).



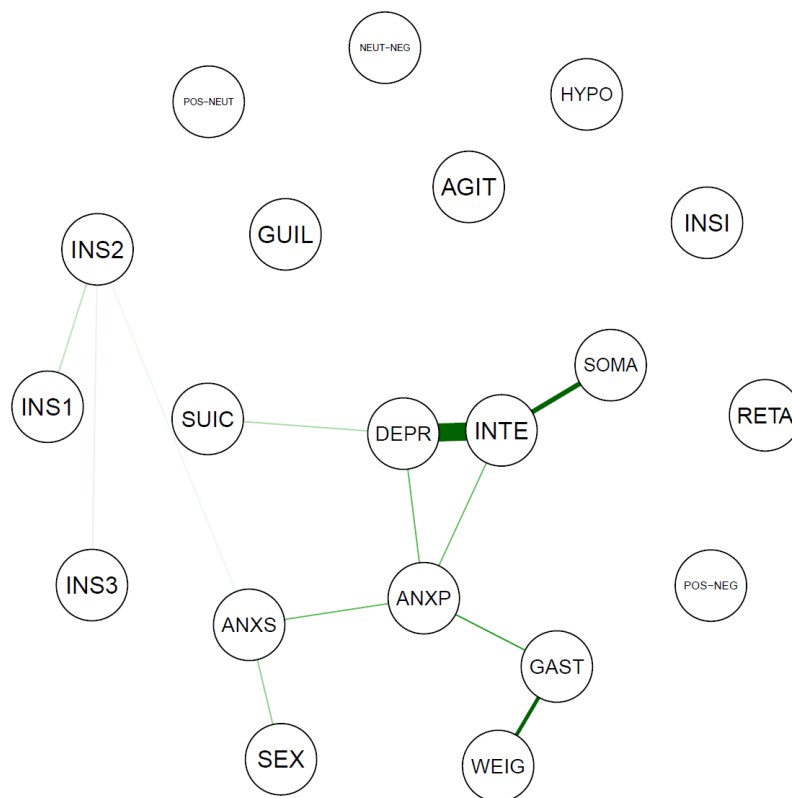
**Figure S11.** Symptom change network for the whole sample. pos-neut = changes in attentional bias for positive vs. neutral stimuli from pre to post ABM. Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).



**Figure S12.** Symptom change network for the whole sample. neut-neg = changes in attentional bias for neutral vs. negative stimuli from pre to post ABM. Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).



**Figure S13.** Symptom change network for the whole sample with three attentional bias change measures. pos-neg/pos-neut/neut-neg = changes in attentional bias for positive vs. negative/neutral or neutral vs. negative, stimuli from pre to post ABM. Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).



**Figure S14.** Symptom change network in the ABM group ( $n = 153$ ) with three attentional bias change measures. pos-neg/pos-neut/neut-neg = changes in attentional bias for positive vs. negative/neutral or neutral vs. negative, stimuli from pre to post ABM. Symptom nodes represent symptom severity changes on each HRSD item from baseline to post ABM (post minus baseline). Label descriptions: DEPR = depressed mood; GUIL = self-depreciation and guilt feelings; SUIC = suicidal impulses; INS1 = early insomnia; INS2 = middle insomnia; INS3 = late insomnia; INTE = work and interests; RETA = psychomotor retardation; AGIT = psychomotor agitation; ANXP = anxiety (psychic); ANXS = anxiety (somatic); GAST = gastro-intestinal symptoms; SOMA = somatic symptoms; SEX = sexual interest; HYPO = hypochondriasis; INSI = loss of insight; WEIG = weight loss. Associations are indicated by edge thickness between nodes (thicker edge = stronger association).















# Dysfunctional Metacognitive Beliefs Are Associated with Decreased Executive Control

Brage Kraft\*, Rune Jonassen, Tore C. Stiles and Nils. I. Landrø

Clinical Neuroscience Research Group, Department of Psychology, University of Oslo, Oslo, Norway

## OPEN ACCESS

### Edited by:

Nuno Barbosa Rocha,  
Health School—P. Porto, Portugal

### Reviewed by:

Amitai Abramovitch,  
Texas State University, USA  
Susana Ochoa,  
Parc Sanitari Sant Joan de Déu, Spain

### \*Correspondence:

Brage Kraft  
brage.kraft@psykologi.uio.no;  
bragekb@gmail.com

### Specialty section:

This article was submitted to  
Psychology for Clinical Settings,  
a section of the journal  
Frontiers in Psychology

**Received:** 10 February 2017

**Accepted:** 30 March 2017

**Published:** 19 April 2017

### Citation:

Kraft B, Jonassen R, Stiles TC and  
Landrø NI (2017) Dysfunctional  
Metacognitive Beliefs Are Associated  
with Decreased Executive Control.  
Front. Psychol. 8:593.  
doi: 10.3389/fpsyg.2017.00593

Dysfunctional metacognitive beliefs (“metacognitions”) and executive control are important factors in mental disorders such as depression and anxiety, but the relationship between these concepts has not been studied systematically. We examined whether there is an association between metacognitions and executive control and hypothesized that decreased executive control statistically predicts increased levels of metacognitions. Two hundred and ninety-nine individuals recruited from the general population and outpatient psychiatric clinics completed the Metacognitions Questionnaire-30 and three subtests from the Cambridge Neuropsychological Test Automated Battery corresponding to the three-component model of executive functions. Controlling for current depression and anxiety symptoms, decreased ability to shift between mental sets was associated with increased negative beliefs about the uncontrollability and danger of worry and beliefs about the need to control thoughts. The results suggest a basic association between metacognitions and executive control. Individual differences in executive control could prove important in the personalization of metacognitive therapy.

**Keywords:** metacognitions, metacognitive beliefs, executive control, executive functions, rumination, worry, depression

## INTRODUCTION

Metacognition refers to the control, modification, and interpretation of thoughts (Wells and Cartwright-Hatton, 2004) and is conceptually closely related to executive control (Fernandez-Duque et al., 2000). Executive functions control and coordinate low-level cognitive processes to guide behavior toward a goal (Banich, 2009). Both executive control mechanisms and metacognition have been highlighted in recent models of mental disorders such as major depression and generalized anxiety disorder (Wells, 1995; Matthews and Wells, 2004; Eysenck et al., 2007; Joormann et al., 2007). However, whether metacognitive beliefs and executive control are empirically related has not been studied systematically.

Impaired executive control has been consistently linked to depression (Snyder, 2013) and rumination (Yang et al., 2016). Rumination is a repetitive and passive focus on one’s depressive symptoms and is an important factor in depression (Nolen-Hoeksema et al., 2008). Having certain dysfunctional metacognitive beliefs (“metacognitions”) predicts rumination and depression (Papageorgiou and Wells, 2009). Common metacognitions in depression include beliefs that rumination will help find answers about the causes of one’s depression and that depressive thoughts are uncontrollable and damaging (Papageorgiou and Wells, 2001). Such metacognitions lock the individual in a perseverative cycle of negative thinking (Matthews and Wells, 2004).

Likewise, an inability to disengage attention from negative thoughts has been proposed as a central neurocognitive mechanism in the consolidation of depressive symptoms (Koster et al., 2011).

Individuals with a history of depression tend to worry about relapsing (Wells and Carter, 2002; Spada et al., 2008b; Sarisoy et al., 2014). Rumination and worry may be conceived as similar cognitive processes described in different research contexts (Watkins et al., 2005) and can be conceptualized collectively as persistent negative thinking (Beckwé et al., 2014). Metacognitions about worry have also been proposed as a promising vulnerability marker of depressive relapse (Halvorsen et al., 2015).

The self-regulatory executive function (S-REF) model of emotional disorders integrates information processing research with Beck's schema theory (Wells and Matthews, 1996). Central to this model is how metacognitions reinforce inflexible and maladaptive coping responses. Metacognitions provide top-down generic procedures for coping ("rumination is helpful"). Input from low-level networks (negative thought intrusions) activate a coping response (rumination), and an online process termed the supervisory executive controls and evaluates the effectiveness of the coping response. The supervisory executive appears to overlap with the concept of executive control, and Wells and Matthews (1996) has proposed attentional capacity and reduced cognitive flexibility as important factors in the modification of metacognitions.

The executive functions can be conceived as three distinct but related components: "shifting" (between tasks or mental sets), "updating" (and monitoring of working memory representations) and "inhibition" (of prepotent responses; Miyake et al., 2000). Depression is associated with reduced performance in all three components (Snyder, 2013); a recent meta-analysis indicated that reduced shifting and inhibition (but not updating) contributes to a ruminative thinking style (Yang et al., 2016).

Very little is known about the neurocognitive correlates of metacognitions. Spada et al. (2010) found that metacognitions about worry were related to self-report of decreased ability to shift and focus attention. However, it is important to note that self-reports of executive control and cognitive test performance have weak or no correlation, and self-reports of executive control are actually better explained by emotional symptom load (Løvstad et al., 2016). Using an emotional Stroop task, Kaur et al. (2011) found a positive correlation between metacognitions and attentional bias for health words after health anxiety induction. However, this study only provided attention bias scores, and no measures of executive functions corresponding to the three-component model by Miyake et al. (2000). Thus, whether metacognitions are related to executive control remains unclear.

**Abbreviations:** MCQ-PBW, positive beliefs about worry; MCQ-NBW, negative beliefs about worry; MCQ-CC, cognitive confidence; MCQ-NCT, beliefs about the need to control thoughts; MCQ-CSC, cognitive self-consciousness; ID/ED, Intra-extra dimensional task; SWM, Spatial working memory task; SST, Stop-signal task.

The main aim of the present study is to examine whether there is a basic association between metacognitions and executive control as measured by objective, well-standardized neuropsychological tests. We hypothesize that: (1) metacognitions and executive control are associated, and (2) decreased executive control statistically predicts increased levels of metacognitions after controlling for current depression and anxiety symptoms.

## METHODS

### Participants

Participants were recruited by newspaper advertisements and posters, and from outpatient psychiatric clinics in Norway. They received oral and written information about the main aim of the study, which was to examine cognitive control and brain function in subjects with major depression and in healthy subjects. The inclusion criteria were age 18–65 years and fluency in Norwegian. All participants provided written informed consent in accordance with the Declaration of Helsinki, and the study was carried out in accordance with the recommendations of the Regional Committee for Medical and Health Research Ethics in Norway and the Norwegian Social Science Data Services.

### Measures and Procedure

Trained psychologists performed evaluations for mental disorders using the Structured Clinical Interviews for DSM-IV criteria I and II (First and Gibbon, 2004). We excluded participants with a history of neurological disorders, bipolar disorder, or psychosis. Education level was classified according to the International Standard Classification of Education (ISCED; UNESCO, 1997). Participants completed Beck's Depression Inventory II (BDI; Beck et al., 1996) and Beck's Anxiety Inventory (BAI; Beck et al., 1988) to measure current symptoms of depression and anxiety, respectively. General cognitive function was examined using Picture Completion (PC) and Similarities (SI) subtests from the Wechsler Adult Intelligence Scale Third Edition (WAIS-III; Wechsler, 1997).

### Metacognitions

To measure metacognitions, participants completed the Metacognitions Questionnaire-30 (MCQ-30; Wells and Cartwright-Hatton, 2004). The MCQ-30 is a 30-item questionnaire where the respondent is asked to rate how much they agree with a specific statement on a 4-point Likert response scale: 1 (*do not agree*), 2 (*agree slightly*), 3 (*agree moderately*), and 4 (*agree very much*). The items generate five subscales: positive beliefs about worry (MCQ-PBW), which measures the extent to which a person believes that perseverative thinking is useful; negative beliefs about the uncontrollability and danger of worry (MCQ-NBW), which assesses the extent to which a person thinks that perseverative thinking is uncontrollable and dangerous; cognitive confidence (MCQ-CC), which assesses confidence in attention and memory; beliefs about the need to control thoughts (MCQ-NCT), which assesses the extent to which a person believes that certain thoughts should be suppressed; and cognitive self-consciousness

(MCQ-CSC), which measures the tendency to monitor one's own thoughts and focus attention inwards. Internal consistency for the MCQ-30 is good (Cronbach's alphas from 0.72 to 0.93) and test-retest reliability is acceptable ( $r$  from 0.59 to 0.87; Wells and Cartwright-Hatton, 2004; Spada et al., 2008a).

## Neuropsychological Assessment of Executive Control

Executive control was assessed using three subtests from the Cambridge Neuropsychological Test Automated Battery (CANTAB, 2009). Subtests corresponded to the three-component model by Miyake et al. (2000): the Intra-Extra Dimensional Task (ID/ED) measures shifting, the Spatial Working Memory Task (SWM) measures updating, and the Stop-Signal Task (SST) measures inhibition. The ID/ED requires participants to pay attention to different examples within a stimulus dimension and shift attention from one set of stimuli to a new, formerly unimportant set of stimuli across nine stages. Shifting ability is operationalized as total errors adjusted for whether the entire task is completed. A high total error adjusted score represents reduced shifting ability (Kaplan et al., 2006). The SWM requires participants to search through several boxes to locate tokens. After a token is located, it will not reappear in the same box during that same trial. Accuracy of working memory is operationalized as the between-trial errors score, which is calculated when the subject searches for a token in a box where a token had been found in a previous trial. High between-trial error scores represent working memory failures (Owen et al., 1990). The SST requires the subject to override a prepotent go response when presented with an infrequent stop signal (a beep). Inhibitory efficiency is operationalized as a stop-signal reaction time, which is estimated through automatic adjustment of the delay between the go stimulus and the stop signal. A higher stop-signal reaction time represents reduced inhibition ability (Logan et al., 1997).

## Statistical Analyses

First, we explored the bivariate correlations between the five MCQ-30 subscales and the three CANTAB subtests. Any statistically significant correlations (two-tailed tests) were then examined further using hierarchical multiple linear regression analyses, with the MCQ-30 subscale as the dependent variable and the CANTAB subtest as the predictor variable. Bonferroni corrections for multiple comparisons were applied. Control variables were age, education level (ISCED), general cognitive functioning (WAIS PC and SI), and current symptoms of depression (BDI), and anxiety (BAI). Control variables were entered in step 1, and the CANTAB variable in step 2. All statistical analyses were performed in IBM SPSS 22.

## RESULTS

### Sample Characteristics

Six participants were excluded from the analyses because of incomplete assessment, and one participant was excluded because of an extremely high score on SWM. A total 299 participants were included in the study. **Table 1** presents the

**TABLE 1 | Participant characteristics (N = 299).**

Characteristic	M (SD)
Age, years	37.4 (13.1)
ISCED	4.6 (1.0)
BDI	8.8 (11.3)
BAI	5.6 (7.1)
WAIS PC, scaled score	12.6 (3.2)
WAIS SI, scaled score	10.8 (3.0)

ISCED, International Standard Classification of Education; BDI, Beck's depression inventory II; BAI, Beck's anxiety inventory; WAIS PC, Wechsler Adult Intelligence Scale Picture Completion; WAIS SI, Wechsler Adult Intelligence Scale Similarities.

demographic and clinical information. There were 201 female participants (67%) and 98 male participants (33%). Fifty-nine participants (20%) were diagnosed with an ongoing episode of major depression, and 54 (18%) had a history of depression. Forty-six participants (16%) had an ongoing anxiety disorder. Fourteen participants (5%) had other mental disorders, and 18 (6%) had personality disorders. Cronbach's alpha for the BDI was 0.96. Computing Cronbach's alpha for the BAI was not possible because only sum scores were available in the data set.

## Correlation Analyses

**Table 2** presents the MCQ-30 subscale scores and CANTAB performance scores. Cronbach's alpha on the MCQ-30 was 0.90 (MCQ-PBW  $\alpha = 0.79$ ; MCQ-NBW  $\alpha = 0.83$ ; MCQ-CC  $\alpha = 0.87$ ; MCQ-NCT  $\alpha = 0.76$ ; MCQ-CSC  $\alpha = 0.79$ ). **Table 3** presents the bivariate Pearson's correlations between the five MCQ-30 subscales and three CANTAB subtests. Bonferroni correction was applied, indicating that only correlations with significance levels below 0.003 were considered significant at a 0.05 level. Shifting errors correlated positively with negative beliefs about worry [ $r_{(297)} = 0.22$ ,  $R^2 = 0.05$ ,  $p < 0.001$ ] and beliefs about the need to control thoughts [ $r_{(297)} = 0.21$ ,  $R^2 = 0.04$ ,  $p < 0.001$ ]. There was a positive correlation between updating errors and negative beliefs about worry [ $r_{(297)} = 0.18$ ,  $R^2 = 0.03$ ,  $p = 0.002$ ]. There was a positive correlation between updating errors and cognitive confidence at borderline significance level [ $r_{(297)} = 0.17$ ,  $R^2 = 0.03$ ,  $p = 0.003$ ].

## Regression Analyses

To control for the possible effect of age, education, general cognitive ability, and current symptoms, we analyzed the statistical significant relationships in four regression analyses. Negative beliefs about worry was predicted by depression and anxiety symptoms (**Table 4**, step 1). Taking control variables into account, shifting errors predicted negative beliefs about worry (**Table 4**, step 2). After control variables, updating errors predicted negative beliefs about worry, but the effect was not significant after Bonferroni correction [ $\beta = 0.11$ ,  $t_{(297)} = 2.11$ ,  $p = 0.04$ ]. Age, education level, and depression and anxiety symptoms predicted beliefs about the need to control thoughts (**Table 5**, step 1). After control variables, shifting errors predicted beliefs about the need to control thoughts at a borderline statistical significance level (**Table 5**, step 2). Cognitive

**TABLE 2 | Metacognitions and executive control functions.**

Variable	M (SD)
MCQ-PBW	8.5 (2.6)
MCQ-NBW	10.2 (3.9)
MCQ-CC	10.4 (4.2)
MCQ-NCT	9.0 (3.2)
MCQ-CSC	12.4 (4.0)
ID/ED, total errors adjusted	24.8 (21.7)
SWM, between-trial errors	20.0 (17.7)
SST, stop-signal reaction time	189.9 (55.6)

Twenty MCQ item scores (0.22%) were missing and replaced by the mean of the subscale for each subject. MCQ-PBW, positive beliefs about worry; MCQ-NBW, negative beliefs about worry; MCQ-CC, cognitive confidence; MCQ-NCT, beliefs about the need to control thoughts; MCQ-CSC, cognitive self-consciousness; ID/ED, Intra-extra dimensional task; SWM, Spatial working memory task; SST, Stop-signal task.

**TABLE 3 | Bivariate correlations between metacognitions and executive functions.**

Variable	MCQ-PBW	MCQ-NBW	MCQ-CC	MCQ-NCT	MCQ-CSC
ID/ED	-0.01	0.22**	0.10	0.21**	0.05
SWM	0.01	0.18**	0.17*	0.09	0.03
SST	0.03	0.04	0.09	0.09	-0.03

\* $p < 0.003$ ; \*\* $p < 0.003$ . MCQ-PBW, positive beliefs about worry; MCQ-NBW, negative beliefs about worry; MCQ-CC, cognitive confidence; MCQ-NCT, beliefs about the need to control thoughts; MCQ-CSC, cognitive self-consciousness; ID/ED, Intra-extra dimensional task; SWM, Spatial working memory task; SST, Stop-signal task.

confidence was predicted by age [ $\beta = 0.11$ ,  $t_{(297)} = 2.22$ ,  $p < 0.05$ ] and depressive symptoms [ $\beta = 0.46$ ,  $t_{(297)} = 5.88$ ,  $p < 0.01$ ], but not by updating errors [ $\beta = 0.01$ ,  $t_{(297)} = 0.15$ ,  $p = 0.88$ ].

## DISCUSSION

The present study makes an important first contribution in bridging metacognitions to executive control based on solid cognitive paradigms. We found that negative beliefs about the uncontrollability and danger of worry and beliefs about the need to control thoughts was related to a decreased ability to shift between mental sets. Shifting ability was also associated with metacognitions after controlling for age, education level, general cognitive function, and depression and anxiety symptoms, suggesting that there is a basic association between metacognitions and shifting ability.

Spada et al. (2008b) have emphasized how negative beliefs about the uncontrollability and danger of worry and beliefs about the need to control thoughts create a cognitive gridlock, which produces even more worry. Our results suggest that this cognitive gridlock is associated with decreased ability to shift between mental sets. This may clarify why individuals with high levels of metacognitions experience difficulties with stopping rumination and worry and switching to more adaptive means of coping.

Interpreting the results according to the S-REF model (Matthews and Wells, 2004) could indicate why and how metacognitions are related to shifting ability. Individuals with

**TABLE 4 | Hierarchical multiple regression with negative beliefs about worry as dependent variable.**

Predictor	B	SE	$\beta$	t	p	95% CI
<b>STEP 1 (CONTROL VARIABLES)</b>						
Age	-0.02	0.01	-0.05	-1.17	0.24	-0.04 to 0.01
ISCED	-0.25	0.19	-0.06	-1.36	0.18	-0.62 to 0.11
WAIS PC	0.00	0.06	0.00	0.08	0.94	-0.11 to 0.11
WAIS SI	0.02	0.06	0.02	0.40	0.69	-0.10 to 0.14
BDI	0.17	0.02	0.50	7.08	<0.01	0.12 to 0.22
BAI	0.12	0.04	0.21	2.95	<0.01	0.04 to 0.19
<b>STEP 2 (CANTAB VARIABLE)</b>						
ID/ED	0.03	0.01	0.14	3.15	0.002	0.01 to 0.04

ISCED, International Standard Classification of Education; WAIS PC, Wechsler Adult Intelligence Scale Picture Completion; WAIS SI, Wechsler Adult Intelligence Scale Similarities; BDI, Beck's depression inventory II; BAI, Beck's anxiety inventory; ID/ED, Intra-extra dimensional task, total errors adjusted.

**TABLE 5 | Hierarchical multiple regression with beliefs about the need to control thoughts as dependent variable.**

Predictor	B	SE	$\beta$	t	p	95% CI
<b>STEP 1 (CONTROL VARIABLES)</b>						
Age	-0.04	0.01	-0.15	-2.70	<0.01	-0.06 to -0.01
ISCED	-0.41	0.18	-0.12	-2.82	0.02	-0.76 to -0.06
WAIS PC	-0.02	0.05	-0.02	-0.37	0.72	-0.13 to 0.09
WAIS SI	-0.04	0.06	-0.04	-0.68	0.50	-0.16 to 0.08
BDI	0.07	0.02	0.24	2.94	<0.01	0.02 to 0.12
BAI	0.10	0.04	0.22	2.57	0.01	0.02 to 0.17
<b>STEP 2 (CANTAB VARIABLE)</b>						
ID/ED	0.02	0.01	0.16	3.02	0.003	0.01 to 0.04

ISCED, International Standard Classification of Education; WAIS PC, Wechsler Adult Intelligence Scale Picture Completion; WAIS SI, Wechsler Adult Intelligence Scale Similarities; BDI, Beck's depression inventory II; BAI, Beck's anxiety inventory; ID/ED, Intra-extra dimensional task, total errors adjusted.

low executive control fail more often when attempting to stop rumination (Koster et al., 2011). A person who often fails at stopping rumination may conclude that they are not very good at it, which strengthens metacognitions (Fernandez-Duque et al., 2000). Wells and Matthews (1996) refer to this as "belief elaboration." The association between metacognitions and executive control may be ascribed to this mechanism. However, the association could also be explained by the fact that metacognitions predict rumination, which in turn decreases executive control. Given the cross-sectional design of the study, we cannot draw conclusions on the direction of the relationship.

Reduced inhibitory control has been proposed to be a gateway for negative thought intrusions, and is more likely to trigger rumination (De Raedt and Koster, 2010; Zetsche and Joormann, 2011; Daches and Mor, 2014; Hoorelbeke and Koster, 2017; Koster et al., 2017). Thus, inhibition probably comes into play in the S-REF model when intrusions from low-level networks activate the supervisory executive. There is a close relationship between metacognitions and rumination (Papageorgiou and



Wells, 2009; Solem et al., 2016) and between rumination and inhibition (Yang et al., 2016), but metacognitions and intrusions are not directly linked in the S-REF model (Matthews and Wells, 2004). This may explain why metacognitions and inhibition were not related.

Whether low cognitive confidence is an accurate reflection of actual cognitive performance has been uncertain (Wells, 2000). Our results revealed an association between cognitive confidence and updating, but this disappeared after controlling for current depression and anxiety symptoms. This is probably because only currently depressed individuals have lower cognitive confidence (Halvorsen et al., 2015). Thus, the present results suggest that the relationship between low cognitive confidence and executive control is limited and is probably restricted to acute depression and anxiety.

Executive control has been implicated in efficacy of psychotherapy, where symptom reduction after cognitive behavioral therapy (CBT) is primarily produced by its impact on higher-order executive functions (Clark and Beck, 2010). Better executive control predicts the use of adaptive emotion regulation strategies (Schmeichel and Tang, 2015), and the ease with which such strategies are utilized (Gotlib and Joormann, 2010). Compared to CBT, metacognitive therapy (MCT) appears to lead to better improvement of executive control, and it has been speculated that this could be ascribed to the attention training technique (Groves et al., 2015). Attention training in MCT involves listening to different sounds in different spatial locations, then switching attention between these sounds. Attention training may therefore potentially increase shifting ability. However, weakening metacognitions and learning to stop rumination are also important interventions in MCT (Wells, 2011), and appear to focus on increasing shifting ability: stopping rumination involves shifting focus away from one's depressive symptoms, and weakening metacognitions involves switching from rumination as the default mental set of coping to a more flexible mental set (e.g., problem solving). Thus, increased executive control after MCT could also be explained by the weakening of metacognitions.

Individual differences in executive control could be important in the personalization of treatment. Cognitive training which

targets shifting ability might increase effectiveness of MCT by increasing the ability to stop rumination and weaken metacognitions. Individuals with low executive control might benefit from training shifting ability before they attempt to stop rumination. On the other hand, individuals with better executive control might not need explicit training. It is also possible that MCT requires a minimum of executive control and that other treatment methods might be more appropriate for individuals with low executive control. Future research should examine how executive control and metacognitions interact during the course of depression and if this is related to treatment.

There are some limitations to the present study, which future research should address. The generalizability of the results is questionable, as the sample was chosen by convenience. Participants recruited via newspaper advertisements and posters may have been influenced by desirability bias when reporting metacognitions. The subtleties of the relationship between metacognitions and executive control could have been elucidated by analyzing the data with individuals separated into sub-groups based on diagnosis, but this was not possible due to low statistical power. After taking into account control variables, the effects of executive control on metacognitions were small to medium in size. Thus, further studies are needed to assess the theoretical and clinical relevance of the current results.

## AUTHOR CONTRIBUTIONS

BK: concept, data analysis and interpretation, drafting and revising the work, and final approval of the version to be published. RJ: data collection. NL: organizing the study. RJ, TS, NL: interpretation of data, critical revision, and final approval of the version to be published. All authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

## FUNDING

The study was funded by The Research Council of Norway (grant number 175387/V50) awarded to NL.

## REFERENCES

- Banich, M. T. (2009). Executive function: the search for an integrated account. *Curr. Dir. Psychol. Sci.* 18, 89–94. doi: 10.1111/j.1467-8721.2009.01615.x
- Beck, A. T., Epstein, N., Brown, G., and Steer, R. A. (1988). An inventory for measuring clinical anxiety: psychometric properties. *J. Consult. Clin. Psychol.* 56:893. doi: 10.1037/0022-006X.56.6.893
- Beck, A. T., Steer, R. A., and Brown, G. K. (1996). *Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.
- Beckwé, M., Deroost, N., Koster, E. H., De Lissnyder, E., and De Raedt, R. (2014). Worrying and rumination are both associated with reduced cognitive control. *Psychol. Res.* 78, 651–660. doi: 10.1007/s00426-013-0517-5
- CANTAB (2009). *The Cambridge Neuropsychological Test Automated Battery*. Cambridge, UK: Cambridge Cognition Limited.
- Clark, D. A., and Beck, A. T. (2010). Cognitive theory and therapy of anxiety and depression: convergence with neurobiological findings. *Trends Cogn. Sci.* 14, 418–424. doi: 10.1016/j.tics.2010.06.007
- Daches, S., and Mor, N. (2014). Training ruminators to inhibit negative information: a preliminary report. *Cogn. Ther. Res.* 38, 160–171. doi: 10.1007/s10608-013-9585-5
- De Raedt, R., and Koster, E. H. (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. doi: 10.3758/CABN.10.1.50
- Eysenck, M. W., Derakshan, N., Santos, R., and Calvo, M. G. (2007). Anxiety and cognitive performance: attentional control theory. *Emotion* 7, 336–353. doi: 10.1037/1528-3542.7.2.336
- Fernandez-Duque, D., Baird, J. A., and Posner, M. I. (2000). Executive attention and metacognitive regulation. *Conscious. Cogn.* 9, 288–307. doi: 10.1006/ccog.2000.0447

- First, M. B., and Gibbon, M. (2004). "The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I) and the Structured Clinical Interview for DSM-IV Axis II Disorders (SCID-II)," in *Comprehensive Handbook of Psychological Assessment, Vol. 2: Personality Assessment*, eds M. J. Hilsenroth and D. L. Segal (Hoboken, NJ: John Wiley & Sons), 134–143.
- Gotlib, I. H., and Joormann, J. (2010). Cognition and depression: current status and future directions. *Annu. Rev. Clin. Psychol.* 6:285. doi: 10.1146/annurev.clinpsy.121208.131305
- Groves, S. J., Porter, R. J., Jordan, J., Knight, R., Carter, J. D., McIntosh, V. V., et al. (2015). Changes in neuropsychological function after treatment with metacognitive therapy or cognitive behavior therapy for depression. *Depress. Anxiety* 32, 437–444. doi: 10.1002/da.22341
- Halvorsen, M., Hagen, R., Hjemdal, O., Eriksen, M. S., Sørli, Å. J., Waterloo, K., et al. (2015). Metacognitions and thought control strategies in unipolar major depression: a comparison of currently depressed, previously depressed, and never-depressed individuals. *Cogn. Ther. Res.* 39, 31–40. doi: 10.1007/s10608-014-9638-4
- Hoorelbeke, K., and Koster, E. H. W. (2017). Internet-delivered cognitive control training as a preventive intervention for remitted depressed patients: evidence from a double-blind randomized controlled trial study. *J. Consult. Clin. Psychol.* 85, 135–146. doi: 10.1037/ccp0000128
- Joormann, J., Yoon, K. L., and Zetsche, U. (2007). Cognitive inhibition in depression. *Appl. Prevent. Psychol.* 12, 128–139. doi: 10.1016/j.appsy.2007.09.002
- Kaplan, J. S., Erickson, K., Luckenbaugh, D. A., Weiland-Fiedler, P., Geraci, M., Sahakian, B. J., et al. (2006). Differential performance on tasks of affective processing and decision-making in patients with panic disorder and panic disorder with comorbid major depressive disorder. *J. Affect. Disord.* 95, 165–171. doi: 10.1016/j.jad.2006.04.016
- Kaur, A., Butow, P., and Thewes, B. (2011). Do metacognitions predict attentional bias in health anxiety? *Cogn. Ther. Res.* 35, 575–580. doi: 10.1007/s10608-011-9387-6
- Koster, E. H. W., Hoorelbeke, K., Onraedt, T., Owens, M., and Derakshan, N. (2017). Cognitive control interventions for depression: a systematic review of findings from training studies. *Clin. Psychol. Rev.* 53, 79–92. doi: 10.1016/j.cpr.2017.02.002
- Koster, E. H., De Lissnyder, E., Derakshan, N., and De Raedt, R. (2011). Understanding depressive rumination from a cognitive science perspective: the impaired disengagement hypothesis. *Clin. Psychol. Rev.* 31, 138–145. doi: 10.1016/j.cpr.2010.08.005
- Logan, G. D., Schachar, R. J., and Tannock, R. (1997). Impulsivity and inhibitory control. *Psychol. Sci.* 8, 60–64. doi: 10.1111/j.1467-9280.1997.tb00545.x
- Løvstad, M., Sigurdardottir, S., Andersson, S., Grane, V., Moberget, T., Stubberud, J., et al. (2016). Behavior rating inventory of executive function adult version in patients with neurological and neuropsychiatric conditions: symptom levels and relationship to emotional distress. *J. Int. Neuropsychol. Soc.* 22, 682–694. doi: 10.1017/S135561771600031X
- Matthews, G., and Wells, A. (2004). "Rumination, depression, and metacognition: the S-REF model," in *Depressive Rumination*, eds C. Papageorgiou and A. Wells. (West Sussex: John Wiley & Sons Ltd), 125–151.
- Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., and Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: a latent variable analysis. *Cogn. Psychol.* 41, 49–100. doi: 10.1006/cogp.1999.0734
- Nolen-Hoeksema, S., Wisco, B. E., and Lyubomirsky, S. (2008). Rethinking rumination. *Perspect. Psychol. Sci.* 3, 400–424. doi: 10.1111/j.1745-6924.2008.00088.x
- Owen, A. M., Downes, J. J., Sahakian, B. J., Polkey, C. E., and Robbins, T. W. (1990). Planning and spatial working memory following frontal lobe lesions in man. *Neuropsychologia* 28, 1021–1034. doi: 10.1016/0028-3932(90)90137-D
- Papageorgiou, C., and Wells, A. (2001). Metacognitive beliefs about rumination in recurrent major depression. *Cogn. Behav. Pract.* 8, 160–164. doi: 10.1016/S1077-7229(01)80021-3
- Papageorgiou, C., and Wells, A. (2009). A prospective test of the clinical metacognitive model of rumination and depression. *Int. J. Cogn. Ther.* 2, 123–131. doi: 10.1521/ijct.2009.2.2.123
- Sarisoy, G., Pazvantoglu, O., Özturan, D. D., Ay, N. D., Yilman, T., Mor, S., et al. (2014). Metacognitive beliefs in unipolar and bipolar depression: a comparative study. *Nord. J. Psychiatry* 68, 275–281. doi: 10.3109/08039488.2013.814710
- Schmeichel, B. J., and Tang, D. (2015). Individual differences in executive functioning and their relationship to emotional processes and responses. *Curr. Dir. Psychol. Sci.* 24, 93–98. doi: 10.1177/0963721414555178
- Snyder, H. R. (2013). Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: a meta-analysis and review. *Psychol. Bull.* 139:81. doi: 10.1037/a0028727
- Solem, S., Hagen, R., Hoksnes, J. J., and Hjemdal, O. (2016). The metacognitive model of depression: an empirical test in a large Norwegian sample. *Psychiatry Res.* 242, 171–173. doi: 10.1016/j.psychres.2016.05.056
- Spada, M. M., Georgiou, G. A., and Wells, A. (2010). The relationship among metacognitions, attentional control, and state anxiety. *Cogn. Behav. Ther.* 39, 64–71. doi: 10.1080/16506070902991791
- Spada, M. M., Mohiyeddini, C., and Wells, A. (2008a). Measuring metacognitions associated with emotional distress: factor structure and predictive validity of the metacognitions questionnaire 30. *Pers. Individ. Dif.* 45, 238–242. doi: 10.1016/j.paid.2008.04.005
- Spada, M. M., Nikčević, A. V., Moneta, G. B., and Wells, A. (2008b). Metacognition, perceived stress, and negative emotion. *Pers. Individ. Dif.* 44, 1172–1181. doi: 10.1016/j.paid.2007.11.010
- UNESCO (1997). *International Standard Classification of Education (ISCED)*. Available online at: [http://www.unesco.org/education/information/nfsunesco/doc/iscsed\\_1997.htm](http://www.unesco.org/education/information/nfsunesco/doc/iscsed_1997.htm) (Accessed December 12, 2016).
- Watkins, E., Moulds, M., and Mackintosh, B. (2005). Comparisons between rumination and worry in a non-clinical population. *Behav. Res. Ther.* 43, 1577–1585. doi: 10.1016/j.brat.2004.11.008
- Wechsler, D. (1997). *WAIS-III: Administration and Scoring Manual: Wechsler Adult Intelligence Scale*. San Antonio, TX: Psychological Corporation.
- Wells, A. (1995). Meta-cognition and worry: a cognitive model of generalized anxiety disorder. *Behav. Cogn. Psychother.* 23, 301–320. doi: 10.1017/S1352465800015897
- Wells, A. (2000). *Emotional Disorders and Metacognition: Innovative Cognitive Therapy*. New York, NY: John Wiley & Sons Ltd.
- Wells, A. (2011). *Metacognitive Therapy for Anxiety and Depression*. New York, NY: Guilford press.
- Wells, A., and Carter, K. (2002). Further tests of a cognitive model of generalized anxiety disorder: metacognitions and worry in GAD, panic disorder, social phobia, depression, and nonpatients. *Behav. Ther.* 32, 85–102. doi: 10.1016/S0005-7894(01)80045-9
- Wells, A., and Cartwright-Hatton, S. (2004). A short form of the metacognitions questionnaire: properties of the MCQ-30. *Behav. Res. Ther.* 42, 385–396. doi: 10.1016/S0005-7967(03)00147-5
- Wells, A., and Matthews, G. (1996). Modelling cognition in emotional disorder: the S-REF model. *Behav. Res. Ther.* 34, 881–888. doi: 10.1016/S0005-7967(96)00050-2
- Yang, Y., Cao, S., Shields, G. S., Teng, Z., and Liu, Y. (2016). The relationships between rumination and core executive functions: a meta-analysis. *Depress. Anxiety* 34, 1–14. doi: 10.1002/da.22539
- Zetsche, U., and Joormann, J. (2011). Components of interference control predict depressive symptoms and rumination cross-sectionally and at six months follow-up. *J. Behav. Ther. Exp. Psychiatry* 42, 65–73. doi: 10.1016/j.jbtep.2010.06.001

**Conflict of Interest Statement:** The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Copyright © 2017 Kraft, Jonassen, Stiles and Landrø. This is an open-access article distributed under the terms of the Creative Commons Attribution License (CC BY). The use, distribution or reproduction in other forums is permitted, provided the original author(s) or licensor are credited and that the original publication in this journal is cited, in accordance with accepted academic practice. No use, distribution or reproduction is permitted which does not comply with these terms.