

Attention Bias Modification and Emotion Regulation in Depression Recurrence

Jenny Tveit Kristoffersen



Innlevert som hovedoppgave ved psykologisk institutt

Department of psychology

UNIVERSITY OF OSLO

Autumn 2016

Attention Bias Modification and Emotion Regulation in Depression Recurrence

Jenny Tveit Kristoffersen

Innlevert som hovedoppgave ved psykologisk institutt

Department of psychology

UNIVERSITY OF OSLO

Autumn 2016

© Jenny Tveit Kristoffersen

2016

Attention Bias Modification and Emotion Regulation in Depression Recurrence

Jenny Tveit Kristoffersen

<http://www.duo.uio.no/>

Trykk: Reprosentralen, Universitetet i Oslo

Abstract

Title: Attention Bias Modification and Emotion Regulation in Depression Recurrence

Author: Jenny Tveit Kristoffersen

Supervisor: Nils Inge Landrø

Background and research aims: Depression is one of the most common mental disorders today. Depression has a high recurrence rate, and may have a large negative impact on life-quality for the individual. This implies that treatment of depression should not only focus on treating a current depression, but should also focus on preventing new episodes. Biased attention toward negative information has been found in individuals with a current depression, but also in individuals in remission. Modifying this attentional bias with Attention Bias Modification (ABM) has been highlighted as a promising intervention to prevent depression relapse. The goal of ABM is to redirect attention toward a positive attentional bias, making the individual more attentive to positive stimuli. This study test the hypothesis that modifying attentional bias with ABM will lead to a decrease in depressive symptoms and enhance the functional emotion regulation skill, reappraisal. The study also predicts that emotion regulation mediates the relationship between ABM and changes in depressive symptoms.

Method: The current paper is based on a sub-sample from an ongoing study by PI Landrø, N.I. and co-PI Harmer, C.J. In this randomized controlled trial (RCT-study), 123 individuals with a history of depression executed ABM twice a day for two weeks (28 sessions). The participants were randomly assigned to an active or a placebo condition of ABM. Clinical, demographic and cognitive variables were assessed before ABM. Depressive symptoms were assessed both before and after ABM. An emotion regulation paradigm provided a measure of the emotion regulation strategy reappraisal within 3 weeks after ABM.

Results: Individuals in the active ABM condition showed a trend toward a greater decrease in depressive symptoms than individuals in the placebo condition after two weeks of ABM. There was no difference between the active ABM and placebo group in their ability to use reappraisal after ABM. The hypothesis that reappraisal mediate the relationship between ABM and depressive symptoms was not supported.

Conclusions: The study carries indications that ABM may have a therapeutic effect on depressive symptoms for individuals at high risk of depression recurrence, but more research is needed to understand the mechanisms by how ABM works. There were no indications that ABM works through the emotion regulation strategy reappraisal.

Acknowledgements

I would like to give a special thanks to my supervisor, Nils Inge Landrø, for guidance and help along the way. I would also like to thank Rune Jonassen for help with the statistical analyses. Thanks to Adrian for reading my paper and your helpful comments.

Thanks to all of the participants in the study. Your interest and participation made in the study all of this possible.

I would also like to thank everyone in the research group at the project «Secondary prevention of depression applying an experimental Attentional Bias Modification procedure». It was a great learning experience working with all of you as a research assistant.

Table of Contents

Introduction	1
1 Theoretical Background	2
1.1 Negative Attentional Bias.....	2
1.1.1 Modifying Negative Attentional Bias	4
1.1.2 Attention Bias Modification to Prevent Depression Recurrence	5
1.2 Emotion Regulation.....	6
1.2.1 Reappraisal and Suppression.....	8
1.2.2 Emotion Regulation and Depression.....	9
1.3 Attention Bias Modification and Emotion Regulation	11
1.3.1 The Role of Attention in Emotion Regulation	11
1.3.2 Modifying Attention to Enhance Emotion Regulation	11
1.4 Emotion Regulation as a Possible Mediator Between ABM and Depression.....	13
1.5 Aim of Study and Hypotheses	13
2 Method	15
2.1 Sample	15
2.2 Design.....	16
2.3 Procedures	18
2.3.1 The ABM-procedure	18
2.3.2 Clinical and Cognitive Measures	19
Emotion Regulation Questionnaire	19
Color-word Interference Task	20
2.3.3 Outcome Variables.....	20
Beck Depression Inventory-II	20
The Emotion Regulation Paradigm	21
2.4 Statistical Analyses.....	23
3 Results	24
3.1 Sample Demographic, Psychometric and Clinical Characteristics	24
3.2 The Effect of ABM on Depressive Symptoms	26
3.3 The Effect of ABM on Emotion Regulation	27
3.3.1 Validating the Emotion Regulation Paradigm	27

3.3.2	The Effect of ABM on Reappraisal.....	28
3.4	Mediation Analysis.....	29
4	Discussion	31
4.1	Main Findings.....	31
4.1.1	Decrease in Depressive Symptoms after ABM.....	31
4.1.2	ABM had no Direct Effect on Reappraisal	34
4.1.3	Reappraisal did not Mediate the Relationship Between ABM and Depressive Symptoms.....	35
4.2	Clinical Implications.....	37
4.3	Strengths and Limitations	40
4.4	Suggestions for Future Research	41
4.5	Conclusions	42
Literature	43
Figure 1:	Cognitive model of depression.	2
Figure 2:	The dot-probe paradigm	3
Figure 3:	Gross' (2001) model of emotion regulation	7
Figure 4:	Overview of the design.	17
Figure 5:	The Attention Bias Modification procedure	18
Figure 6:	The emotion regulation paradigm.....	22
Figure 7:	A visual analogue scale	22
Figure 8:	The effect of ABM on depressive symptoms.	26
Figure 9:	Validating the emotional regulation paradigm	27
Figure 10:	The effect of ABM on reappraisal	28
Figure 11:	Mediation model.....	30
<i>Table 1:</i>	<i>T-tests of demographic, psychometric and clinical characteristics</i>	25

Introduction

Depression is one of the most common mental disorders in adults today. Statistics from USA showed that the lifetime prevalence for depression is 16.2 %, and that each year 6.6 % of the population suffers from an episode of depression (Kessler et al., 2003). Depression is known for its emotional, cognitive and physiological symptoms. The emotional symptoms include dysfunctional emotion regulation, absence of joy or happiness, and sustained negative affect. The cognitive symptoms include a cognitive bias for negative emotional information, rumination, and negative beliefs about oneself, the world and the future. The physiological symptoms include fatigue, and disturbances in sleep and appetite. Depression has a large impact on life-quality for those who suffer from it. Many patients experience negative consequences for interpersonal relationships and the ability to work or do daily life chores.

Depression is also known for its high recurrence rate. The risk of suffering from a new episode of depression is heightened if the patient has a history of one or more previous episodes. Gotlib and Joormann (2010) found that about 75 % of patients diagnosed with a current depression had a history of more than one previous episode. Other studies have found that patients with a history of one previous episode of depression had a 36.7 % chance of suffering from a new episode, and patients with a history of two previous episodes had a 48 % chance of suffering from a third episode. For each new episode, the risk of suffering from another episode of depression increased with 15 % (Feliciano, Renn & Areán, 2012). The high recurrence rate indicate that it is also necessary to focus on preventing recurrent episodes when treating depression.

Cognitive theories of depression argue that the way people think, the way they interpret the environment and what part of a situation they attend to plays a crucial role in the development, maintenance and recurrence of depression (Gotlib & Joormann, 2010). The goal of previous research has been to gain understanding about how dysfunctions in cognition may lead to emotion dysregulation and other symptoms of depression. Research has started to explore the effect of targeting attentional biases in cognition with computer-based attention bias modification procedures. The earliest studies on this topic were conducted with healthy students, individuals with anxiety or currently depressed patients. Recent studies have now started to explore the effect of Attention Bias Modification (ABM) in individuals at high risk of depression relapse (Browning, Holmes, Charles, Cowen & Harmer, 2012), and suggests that ABM may represent a promising new intervention with the goal to prevent depression.

1 Theoretical Background

1.1 Negative Attentional Bias

Cognitive theories of depression suggest that depression is associated with a negative attentional bias in the processing of emotional information (Beck, 2008). Negative attentional bias is the individual's tendency to attend to negative emotional information rather than neutral or positive emotional information. Patients with depression have also been found to remember or interpret information in a more negative way than never-depressed individuals (Gotlib & Joormann, 2010). They also showed difficulties with redirecting attention away from negative information. Cognitive models of depression suggest that attentional biases are an important factor in the onset and maintenance of depression (Beck, 2008) (See figure 1). The model posits that adverse life-experiences form dysfunctional attitudes (or schemas), which reflect a cognitive vulnerability in the individual. Activation of these dysfunctional schemas by daily-life events over time will result in attentional biases in attention, negatively biased interpretations and mild depressive symptoms (Beck, 2008).

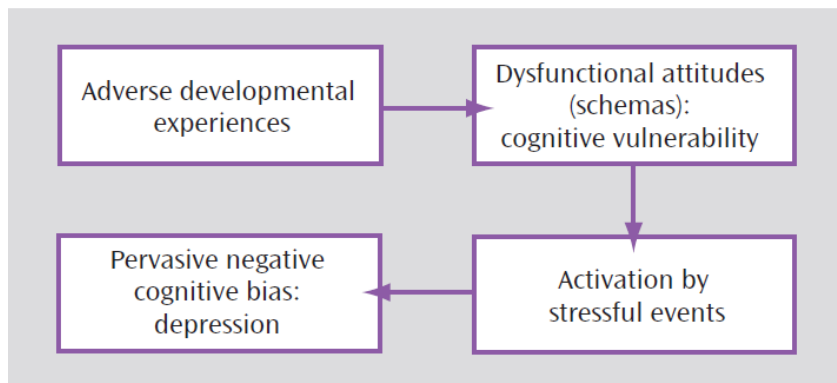


Figure 1: Cognitive model of depression (Beck, 2008). The figure illustrates the role of attentional bias in the onset and maintenance of depression.

Research on attentional bias in depression have mixed findings. Some studies have supported the existence of attentional bias towards negative information in depressed individuals (Donaldson, Lam & Mathews, 2007; Mathews, Ridgeway & Williamson, 1996), whereas other studies have not (Mogg, Bradley, Williams & Mathews, 1993; MacLeod, Mathews & Tata, 1986). A more recent review of Peckham, McHugh and Otto (2010) compared 29 empirical studies on negative attentional bias in depression. Their meta-analysis supported the existence of attentional bias toward negative information in depression. They

found that the association between attentional bias and depression was more robust when a dot-probe paradigm was used. The dot-probe procedure was developed to serve as a more direct measure of attentional bias (See figure 2). In a dot-probe paradigm developed by MacLeod, Rutherford, Campbell, Ebsworthy and Holker (2002), individuals responded to a cue (reported number of dots) after two visual stimuli (words or faces) with different emotional valiance were presented. The cue appeared at the same location as a stimulus with a positive, negative or neutral valiance. Faster reaction time to the cue behind negative stimuli was believed to reflect a negative attentional bias; the individual was more ready to attend to the negative stimuli.

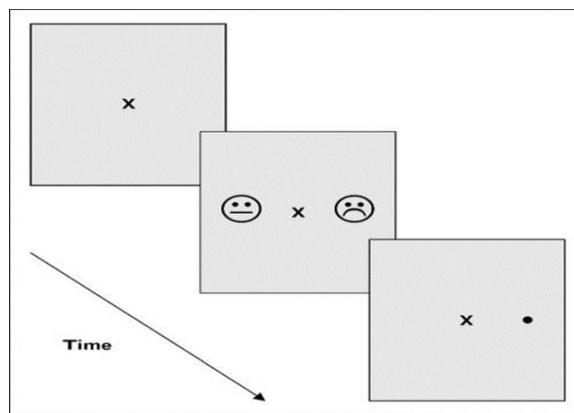


Figure 2: The dot-probe paradigm. Individuals responded to a cue that appeared at the same location of an emotional stimulus. Shorter reaction time to cues behind negative emotional stimuli was believed to reflect negative attentional bias.

Other research has also found evidence for negative attentional bias in both currently depressed patients and patients in remission at high risk of depression relapse, but not in healthy controls (Joormann & Gotlib, 2007). This supports the idea that attentional bias may also be an important characteristic for individuals at high risk of recurrent episodes of depression. This finding may further support the theory that negative attentional bias may be important in the understanding of depression onset and maintenance. Although studies have shown a promising association between depression and negative attentional bias, are they not sufficient to make conclusions about a possible causal link between negative attentional bias and depression. An aim of more recent research has been to gain more knowledge about the possible causal relationship between negative attentional bias and depression.

1.1.1 Modifying Negative Attentional Bias

The next step in the attention bias modification research was to develop procedures to actually modify negative attentional bias. Newer research developed a variation of the computer-based dot-probe paradigm used to describe the attentional bias by MacLeod et al., 2002, called attention bias modification (ABM). This procedure visually train attention from a negative attentional bias to a positive attentional bias. The rationale behind this procedure is based on the knowledge that attention is plastic and can be trained (Wadlinger & Isaacowitz, 2011). The quality of the stimuli used in an attention bias modification procedure may differ from study to study, but what they all have in common is the goal of redirecting attention towards more functional biases (biases toward positive and neutral information). Attentional bias modification procedures aim to modify negative attentional bias through more implicit processes compared to more explicit and verbal processes like in cognitive therapy. Attention bias modification involves modifying attentional processes that are not under voluntary control (Beard, Sawyer & Hofmann, 2012).

Studies have revealed mixed findings on whether negative attentional bias in individuals with depression can be altered by attention bias modification. Some studies were successful in modifying negative attentional bias (Beevers, Clasen, Schnyer & Enok, 2015; MacLeod & Bridle, 2009) and some were not (Baert, Raedt, Schacht & Koster, 2010; Kruijt, Putman & van der Does, 2013). Caution should be taken when comparing the results of these different studies because they used different attention bias modification procedures, and because of differences in how changes in attentional bias was measured and operationalized. The different findings may suggest that some characteristics in the attention bias modification procedure may affect its success. The studies that managed to modify negative attentional bias had a longer implementation period (Beevers et al., 2015; Yang, Ding, Dai, Peng & Zhang, 2015; Browning et al, 2012). The studies that did not manage to modify negative attentional bias had shorter or less frequent implementation of attention bias modification (Kruijt et al., 2013; Everaert, Mogoase, David & Koster, 2015). The quality of the stimuli may also matter. Browning et al. (2012) found a significant increase in positive attentional bias for patients with vulnerability of depression when the stimuli were pictures of faces rather than words. Therefore, the success of attentional bias modification on attentional bias may depend on characteristics of the attention bias modification procedure.

1.1.2 Attention Bias Modification to Prevent Depression Recurrence

Attention bias modification (ABM) has been suggested to represent a novel treatment to prevent depression recurrence because it targets negative attentional bias, an important factor in the onset and maintenance of depression (Browning et al., 2012). The casual relationship between negative attentional bias and depression has been explored by studies trying to modify attention and observing its effects on depressive symptoms. These studies compare individuals with depression, randomly assigned to an active or placebo condition of ABM.

Studies have revealed mixed findings on this issue too. Some studies found a decrease in depressive symptoms in individuals receiving ABM. (Yang et al., 2015; Baert et al., 2010). Yang et al. (2015) found a significant reduction in depressive symptoms in college students with mild to severe symptoms of depression immediately after 8 sessions of ABM completed during a 2-week period. This reduction of depressive symptoms was not found in the placebo group. Baert et al., (2010) found a mild improvement in the severity of depressive symptoms in college student with mild to severe depressive symptoms after 10 sessions of ABM. The decrease of depressive symptoms was not found in individuals with severe depressive symptoms, indicating that the therapeutic effect of the ABM was better at a mild to moderate symptom-level. The authors suggested that the therapeutic effects of ABM may depend on depression severity. In contrast, other studies found no differences in depressive symptoms between depressed individuals that received an active ABM and those who received a placebo ABM (Beevers et al., 2015; Kruijt et al., 2013). It should be noted that Beevers et al. (2015) found an approximately 40 % decrease in depressive symptoms after 8 sessions of ABM during a 4-week period, but this was not exclusive for the group that received an active ABM. A decrease in depressive symptoms was also found in individuals receiving a placebo ABM. Kruijt et al. (2013) found no immediate effect on depressive symptoms after a single-session ABM. This negative finding is in line with the research saying that ABM-procedures with longer implementation duration may have a better effect on negative attentional bias (Browning et al., 2012).

The long-term effect of ABM has also been studied. Yang et al. (2015) found a significant reduction in depressive symptoms immediately after ABM in the active ABM group, and this effect maintained at the 3-month follow up. There were also more participants in the active ABM group that remained asymptomatic at the 7 month follow up compared to the placebo group. Beevers et al. (2015) found that symptoms of depression decreased after ABM and were relatively stable to a 1-month follow up. Browning et al. (2012) found a

delayed response to the ABM-intervention with a significant difference between the active ABM group and the placebo group in depressive symptoms at the 1-month follow-up.

Studies have also tried to explain the mechanisms by which ABM works. It has been suggested that there might exist variables that mediate the relationship between ABM and decreases in depressive symptoms. Yang et al. (2015) found that rumination mediated the effect of ABM on the reduction of depressive symptoms. Browning et al. (2012) suggested that the effect of ABM on depressive symptoms is mediated by changes in Cortisol Awakening Response (CAR) (ABM reduces CAR, which in turn reduces depressive symptoms). The sample size in the Browning et al. (2012) study was not large enough to do the actual mediation analysis, so this remains only a suggestion for now. It is beyond the scope of this paper to go into detail about rumination and CAR as possible mediators, but it illustrates the fact that the mechanisms behind ABM are not yet fully understood. Emotion regulation has also been suggested as a possible mediator between ABM and decreases in depressive symptoms. It may be a variable worth exploring as to gain more knowledge about the mechanisms behind ABM.

1.2 Emotion Regulation

Emotions may be defined as “feeling (or affect) states that involve a pattern of cognitive, psychological and behavioral reactions to events” (Keller, 2009, p. 502). Emotions are important for social communication and in guiding behavior. Negative emotions are associated with both decreased well-being and mental disorders (Keller, 2009). According to Keller (2009), does all emotional states share four common characteristics: 1) Emotions are triggered by inner or outer stimuli. 2) Emotional responses are a result of appraisals given to the situation. 3) The body has a physiological response to the appraisal of a situation or a stimulus. 4) Emotions include behavioral tendencies – how we express our emotions or how we act on them. E.g. imagine that a dog is running towards you. This situation may trigger the emotion fear if you believe the dog is dangerous. A physiological response may follow e.g. sweating and elevated heart rate. This impacts how you act in the situation, and you might run away from the dog. James J. Gross (2001) views emotions as a process where a person in a specific situation may attend to a specific aspect of the situation, and ascribe that situation a specific appraisal or meaning before an emotional response is fully activated. Behavioral, experiential and physiological tendencies will then follow.

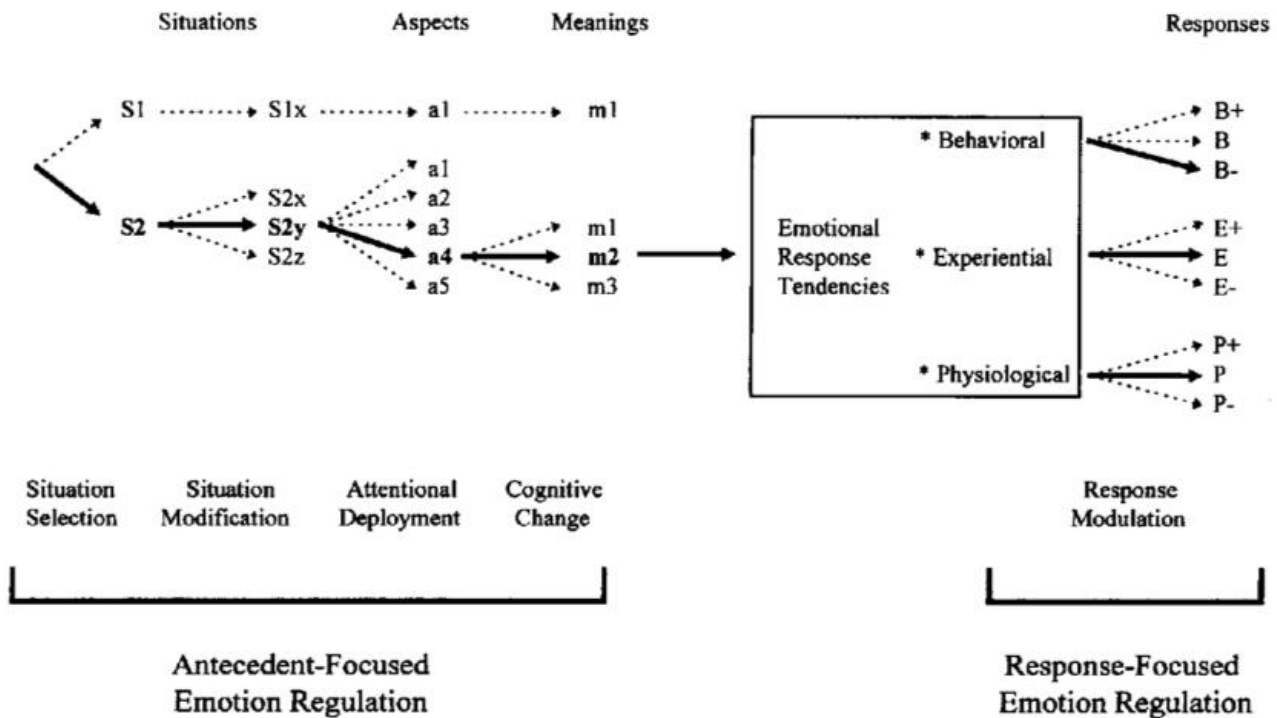


Figure 3: Gross' (2001) model of emotion regulation. Different emotion regulation strategies find place at different stages in the emotion eliciting process. Reappraisal finds place before the emotional response is fully elicited and suppression finds place after the emotional response is fully activated.

There are also ways to influence - or regulate - the quality of the emotion experienced or the expression of that emotion. Emotion regulation is something that at some point in the emotion eliciting process could affect which emotion is experienced or expressed in the end. Emotion regulation may be defined as, “The processes by which individuals influence which emotions they have, when they have them and how they experience and express these emotions” (Gross, 1998, p. 275). James J. Gross’ (2001) process-model of emotion regulation (see figure 3) suggests that how we regulate emotions has a great impact on the subjective experience of an emotion or the expression of it. Gross (2001) suggest that different strategies to regulate emotions may occur at different time points in this process, both before and after the emotional response is fully activated. Gross (2001) called the strategies finding place before the emotion is fully activated for antecedent-focused emotion regulation. These strategies work by changing what aspects of the situation you attend to or how you appraise the situation. E.g. instead of thinking that the dog running towards you is going to bite you, a positive appraisal would be that the dog is excited to see you. This could make you happy to

see the dog rather than scared, and even make you run toward the dog in excitement. Emotion regulation strategies occurring after the emotional response is fully activated, did Gross (2001) call response-focused emotion regulation. Behavioral, experiential and physiological tendencies that follow the emotional response are modified rather than the emotional experience itself. E.g. you are still filled with fear that the running dog will bite you, but you change your behavior and expression of that fear, so that no one can tell that you are scared.

1.2.1 Reappraisal and Suppression

There are several different emotion regulation strategies described in the literature, but a great deal of the literature has focused on the two emotion regulation strategies reappraisal and suppression. These strategies are frequently used by most people in daily-life, and are easily measured and manipulated in research settings. Reappraisal and suppression are also examples of two different superior emotion regulation strategies: antecedent-focused emotion regulation and response-focused emotion regulation. Studies has also found evidence that individual difference in the use of reappraisal and suppression may be related to depression (Garnefski & Kraaij, 2007; John & Gross, 2004; Zare & Solgi, 2010). It is therefore interesting to focus on these two strategies in the work of preventing depression recurrence.

Reappraisal is an emotion regulation strategy where the individual modifies how he/she appraise the situation. What meaning the individual ascribes to the situation will determine the quality of the emotion experienced in the end. E.g.: A job interview is a situation that trigger nervousness for many people. You could appraise the situation as a situation where the interviewers assess whether you are a good candidate for the job or not, or you could appraise the job interview as a situation where you need to get to know the workplace to see if it could be a good job for you. These two different appraisals of the situation may elicit different emotions. The first appraisal might elicit nervousness, and the second appraisal might elicit curiosity. In this situation, the persons' appraisal early in the emotion eliciting process determine the emotion experienced in the end. There is also scientific evidence that voluntary changes in the appraisal of a situation may change the intensity of the emotional response (Joormann & Gotlib, 2009). Suppression, on the other hand, will not impact the quality of the emotion, but rather the expression of it. E.g. putting on a poker face to look calm during a job interview even though you're still feeling very nervous. Though you might look calm, you haven't regulated your emotions to the point where you actually feel calm.

The main difference between reappraisal and suppression is the point in the emotional eliciting process they occur. Reappraisal finds place before the emotional response is fully elicited, and suppression finds place later in the emotion elicit process, after the emotion is fully activated. Using reappraisal changes the emotional experience, while in suppression, the individual does not regulate the quality or intensity of the emotion. The different emotional consequences of reappraisal and suppression also imply that individual differences in using these strategies might be important to understand individual differences in experience and expression of negative emotions. In the depression literature has reappraisal been viewed as a functional emotion regulation strategy because of its ability to regulate a negative emotion to a positive or neutral emotional response. In contrast, suppression has been viewed as a dysfunctional emotion regulation strategy because of its inability to regulate negative emotions elicited by an event, leading to a lasting negative emotional state. This might be important in understanding why individuals with depression and at high risk of depression often experience a lasting state of negative affect.

1.2.2 Emotion Regulation and Depression

Dysfunctional emotion regulation strategies were found to be related to mental disorders like mood disorders, anxiety disorders and eating disorders (Mehrabi, Mohammadkhani, Dolatshahi, Pourshahbaz & Mohammadi, 2014; Aldao, Nolen-Hoeksema & Schweizer, 2010). Dysfunctional emotion regulation can be defined as response patterns of emotion regulation strategies that have a negative affective consequence for the individual, with a greater degree of experienced negative affect across situations (more than expected when negative emotions sometimes are the appropriate emotional response to an event). In contrast, functional emotion regulation strategies were found to be related to better emotional adjustment and psychological health (Mehrabi et al., 2014). Recent research has started to study emotion regulation in clinical groups, and there is evidence that dysfunctions in emotion regulation may be crucial in understanding depression (Garnefski & Kraaij, 2007; John & Gross, 2004; Zare & Solgi, 2010). The importance of dysfunctional emotion regulation in depression is also reflected in the fact that one of the main criteria in diagnosing major depressive disorder is lasting negative affect and the absence of positive affect.

The importance of emotion regulation in depression is further implicated when reappraisal and suppression has been found to have different affective consequences. Individuals who use suppression more habitually reported more depressive symptoms than

individuals who use reappraisal more habitually (John & Gross, 2004). Previously depressed individuals have also been found to more often respond to negative emotions with rumination and suppression, and more rarely with reappraisal (Aker, Harmer & Landrø, 2014). John and Gross (2004) found that a habitual use of suppression was associated with lower self-esteem, less optimism and less satisfaction with social relationships and life in general. Those who suppressed more also reported feeling inauthentic (because they do not show people around them how they really feel), ruminate more, and were less good at repairing negative mood. Those who suppress more also reported experiencing less positive emotions. On the other hand, higher use of reappraisal was associated with fewer symptoms of depression (John & Gross, 2004). Those who habitually used reappraisal were more satisfied with their life, were more optimistic, had higher self-esteem and handled their environment better.

Ehring, Tuschen-Caffier, Schnülle, Fischer and Gross (2010) studied emotion regulation strategies with experimental interventions to compare individuals at high risk of depression and healthy controls. The participants viewed different film clips with neutral or negative emotional content. During the film the participants were instructed to “just watch” (spontaneous condition), “try not to show how you are feeling” (suppression) or to “watch the movie with an objective eye, like pretending to be the director” (reappraisal). This study found that individuals at high risk of depression used suppression more than healthy controls in the spontaneous condition. Interestingly, both groups were successfully able to use reappraisal when instructed to. An explanation for this might be that individuals at high risk of depression might have difficulties with spontaneously choosing a functional emotion regulation strategy. They might be able to use reappraisal, but have trouble choosing it.

Reappraisal and suppression has also been viewed as a protective factor and a vulnerability factor for depression respectively. John and Gross (2004) suggested that reappraisal could be a protective factor against depression because of the strategy’s ability to change the emotional quality from negative to neutral or positive. Reappraisal may reflect a functional emotion regulation strategy, and be a more well-adapted way to repair negative mood than suppression. They also argued that suppression might reflect a vulnerability to depression because of its association with experiencing more negative emotions. Suppression, but not reappraisal, was found to predict previous depressive episodes (Aker et al., 2014).

1.3 Attention Bias Modification and Emotion Regulation

1.3.1 The Role of Attention in Emotion Regulation

Attention is an important component of emotion regulation (Wadlinger & Isaacowitz, 2011).

Automatic biases in attention and difficulties in redirecting attention may be important attentional mechanisms in emotion regulation (Joormann & D'Avanzato, 2010).

Dysfunctional attentional biases (e.g. attentional bias toward negative emotional information) may lead to inflexible, automatic and unconscious negative appraisals of a situation (Siemer & Reisenzein, 2007). Individuals with depression and at high risk of depression were found to have difficulties redirecting attention away from negative emotional information (Joormann & D'Avanzato, 2010). The negative attentional bias may prompt an individual to attend to a negative aspect of a situation and make a negative appraisal of that situation, which in turn elicit a negative emotional response. This makes it harder to activate positive or neutral appraisals of the situation and regulate negative emotions to a neutral or positive emotional response. The ability to use the reappraisal may depend on the person's ability to override automatic attentional and appraisal biases (Gotlib & Joormann, 2010). A greater flexibility in attention may enhance the possibility to disengage attention from negative aspects of the situation and attend to more positive or neutral aspects of a situation, or it may enhance the ability to have positive appraisals more available (Wadlinger & Isaacowitz, 2011). This in turn could lead to more positive emotional experiences.

1.3.2 Modifying Attention to Enhance Emotion Regulation

Another goal of the ABM-research has been to study its ability to prompt functional emotion regulation. Because of the role of attention in emotion regulation, training attention may be a valuable tool to prompt functional emotion regulation (Wadlinger & Isaacowitz, 2011). A review of Wadlinger and Isaacowitz (2011) concluded that modifying attention could directly modify attentional processes that were important in emotion regulation. Several studies have investigated the effect of attention bias modification on emotion regulation (Dandeneau, Baldwin, Baccus, Sakellaropoulou & Pruessner, 2007; Johnson, 2009; Wadlinger & Isaacowitz, 2008). These studies used different attention bias modification procedures, but the common goal of the procedures was to reorientate attention away from negative information

and towards positive information. Dandeneau et al. (2007) found that undergraduate students who completed a visual probe task over five days reported feeling less stressed about an upcoming exam. The students also reported feeling less anxious and more competent in their academic abilities after completing the exam. The attention bias modification procedure used in this study involved ignoring information associated with social threat, and rather search for information associated with social acceptance. For these students, the attention bias modification helped them regulate their emotions in daily-life tasks, but it was unknown whether these regulation improvements were a result of attentional preferences towards positive stimuli or something else. A study by Wadlinger and Isaacowitz (2008) found that the participants who received attention bias modification viewed negative images significantly less than those who did not receive attention bias modification. This implicated that the participants had learned a strategy of attentional avoidance toward negative stimuli, because this difference did not exist before the attention bias modification procedure was implemented. A third study also found that modifying attention towards positive stimuli facilitated effective emotion regulation (Johnson, 2009). Participants who redirected attention towards positive faces and away from angry faces with attention modification, almost had lower state frustration scores on a stress task as compared to those who didn't receive the attention bias modification. The participants that were better at attending to happy faces also persevered longer on the stress task. Individual differences in the ability to attend to positive faces predicted how long the participants attempted to complete the stress task. Taken together, these studies provide evidence that attention bias modification using dot-probe or visual search procedures may effect emotion regulation outcomes. It should be noted that these studies were not carried out in a clinical sample with individuals with depression or at high risk of depression, so conclusions about how this is in that particular sample can not be drawn from these studies. Therefore, more research is needed to understand the effect of attention bias modification on emotion regulation in individuals suffering from depression or at high risk of depression recurrence.

1.4 Emotion Regulation as a Possible Mediator Between ABM and Depression

The effect of modifying attentional biases on symptoms of depression has been studied empirically, but the mechanisms behind this relationship are not yet fully understood. Questions can be raised about whether modifying attention has a direct effect on depressive symptoms or whether modifying attention affect other important processes in depression, which in turn leads to a decrease in depressive symptoms. Biases in attention, dysfunctional emotion regulation and lasting negative affect are all known markers of depression, and therefore interesting targets of ABM when the goal is a decrease in depressive symptoms. Emotion regulation may therefore be an interesting variable to further explore as ways to understand the relationship between ABM and depression. A link between attention and emotion regulation has been illustrated in the current chapter, where biases in attention may prompt dysfunctional emotion regulation strategies and lasting negative affect.

On the other hand, could a redirection of attention toward positive information enhance functional emotion regulation skills (reappraisal) which in turn could lead to a reduction in the experience of lasting negative affect and a reduction of depressive symptoms? Could reappraisal represent a mediator between ABM and changes in depressive symptoms? No studies were found on reappraisal as a possible mediator between ABM and depression, but a couple of studies on anxiety implicated that modifying attention may have an impact on emotional processes, which in turn affected anxiety vulnerability (Mathews & MacLeod, 2002; MacLeod et al., 2002).

1.5 Aim of Study and Hypotheses

The aim of the study is to explore the relationship between ABM, depression and emotion regulation. The emotion regulation process studied here is the functional emotion regulation strategy, reappraisal. The current study holds three hypotheses about the relationship between these three variables based on the literature reviewed in this chapter.

The first hypothesis is that ABM will have a direct effect on depressive symptoms after two weeks of ABM. It is predicted that individuals in the active ABM condition will have a greater decrease in depressive symptoms than individuals in the placebo condition.

The second hypothesis is that ABM will enhance the individual's ability to use the functional emotion regulation strategy, reappraisal. It is predicted that individuals in the active

ABM condition will report less negative emotions in response to aversive emotional stimuli than individuals in the placebo condition, representing a better ability to use reappraisal.

As the relationship between ABM and depression is not fully understood yet, a secondary aim of this study is to explore emotion regulation as a possible mechanism by how ABM works. The third hypothesis is that reappraisal mediate the relationship between ABM and changes in depressive symptoms.

2 Method

The current paper is based on a subsample from the research project “Secondary Prevention of Depression Applying an Experimental Attentional Bias Modification Procedure” by principal investigator (PI) Nils Inge Landrø at the department of psychology at the University of Oslo, Norway, and co-PI Catherine J. Harmer, director of Psychopharmacology and Emotional Research Lab (PERL) at the Department of Psychiatry in Oxford, England. I worked as a research assistant on this project for a year during my education at the clinical psychology program at the University of Oslo. This research project study the effect of Attention Bias Modification (ABM) on three different important markers for depression: dysfunctional emotion regulation, cortisol and residual symptoms in individuals at high risk of depression recurrence. The aim of this study is to establish a simple non-medical procedure to prevent depression recurrence. The study is a Randomized Controlled Trial (RCT) with the ABM-procedure as the clinical intervention being tested. It is also a longitudinal study that follows the participants for a year, with assessments at 2 weeks, 1 month, 2 months, 6 months and 12 months after the first assessment. A subsample also conducted an emotion regulation paradigm in a MRI part of the study to assess emotion regulation skills. The collection of data to this project started spring 2014 and is planned to end fall 2017. The data used in the current paper were collected from fall 2014 to spring 2016. The research project was approved by the Norwegian regional ethical committee (REK).

2.1 Sample

A total of 318 participants with a history of depression were recruited to participate in the study “Secondary Prevention of Depression Applying an Experimental Attentional Bias Modification Procedure”. The current study is based on the subsample of 129 participants that also conducted the emotion regulation paradigm. Two of these participants withdrew from the study, and four participants were excluded from the study because they did not have a history of depression. 123 participants were included in the final sample. This sample consisted of 69% ($n = 85$) women and 31 % ($n = 38$) men. The age of the participants ranged from 21–71, with a mean age at 43.5 ($n = 123$, $SD = 12.9$). 103 participants met the criteria for recurrent depression in the structured diagnostic interview Mini International Neuropsychiatric Interview (M.I.N.I.) 6.0.0 (Sheehan et al., 1998) (Norwegian version by Leiknes & Malt,

2009). 18 participants met the criteria in M.I.N.I. for one previous episode of depression and 2 participants met the criteria for dysthymia. 14 participants met the criteria for current depression at the first assessment. 40 participants met the criteria for a current comorbid diagnosis. Comorbid diagnosis included panic disorder, anxiety disorders, OCD, dysthymia, hypomania and PTSD. Participants with comorbid diagnosis were included because research has shown that a high degree of comorbidity is expected in patients with depression (Hasin, Goodwin, Stinson & Grant, 2005).

Participants were recruited from Diakonhjemmet hospital, Vinderen in Oslo, the Coperio center in Trondheim, Lovisenberg hospital in Oslo and Unicare in Oslo. Enrollment to the study was based on the individuals having a history of depression. A short letter with information about the study was sent out to potential participants before a research assistant contacted each individual shortly after by phone. All of the participants signed a consent form after they received information about the study. Exclusion criteria were never-depressed individuals, and individuals with a former neurological disorder or psychosis.

2.2 Design

An overview of this study's design is given in figure 4. The first assessment was conducted at the neuropsychological lab at the psychology department, University of Oslo. At the first assessment, demographic variables were registered and an assessment of depression and other psychological disorders were conducted by a trained clinician or a clinical psychology student with M.I.N.I. (the modules of alcohol and drug abuse and antisocial personality disorder were excluded). The color-word interference task (Delis, Kaplan & Kramer, 2001) was included as a measure of executive functions at the first assessment. The participants also conducted a placebo condition of ABM to set a baseline. The participants then received instructions about the procedures for the following two weeks at the end of the meeting.

Symptoms of depression were also assessed with Beck Depression Inventory-II (BDI-II; Beck, Steer & Garbin, 1988). The habitual use of the emotion regulation strategies cognitive reappraisal and suppression were assessed with the Emotion Regulation Questionnaire (ERQ; Gross & John, 2003). The participants received BDI-II, ERQ and a written consent form with information about the study by mail or e-mail before the first assessment. The participants filled out these questionnaires at home 2-3 days before the first assessment.

The participants conducted ABM at home on a computer provided by the research project twice a day for 14 days: once in the morning and once in the evening (28 sessions). The participants were not told the rationale behind ABM, but were told that the study was about attention and mood. The participants were randomly assigned to the ABM active condition or the placebo condition. Another research assistant did this before the first assessment, creating a double blind.

A second assessment was conducted immediately after the 2-week training period. The placebo condition of ABM was executed again to compare to the baseline set at the first assessment. The participants also filled out BDI-II again a couple of days before the second assessment. The participants conducted an emotion regulation paradigm within three weeks after the second assessment. The emotion regulation paradigm was conducted in a MRI-scanner at the Intervention Centre at the National Hospital (Rikshospitalet) in Oslo. Participants with claustrophobia or with a specific surgery metal in their body did not conduct the emotion regulation paradigm in the MRI-scanner for of safety reasons. They were offered to execute the emotion regulation paradigm in the neuropsychological lab at the University of Oslo. 111 participants executed the emotion regulation paradigm in the MRI-scanner, and 12 participants executed the paradigm in the neuropsychological lab.

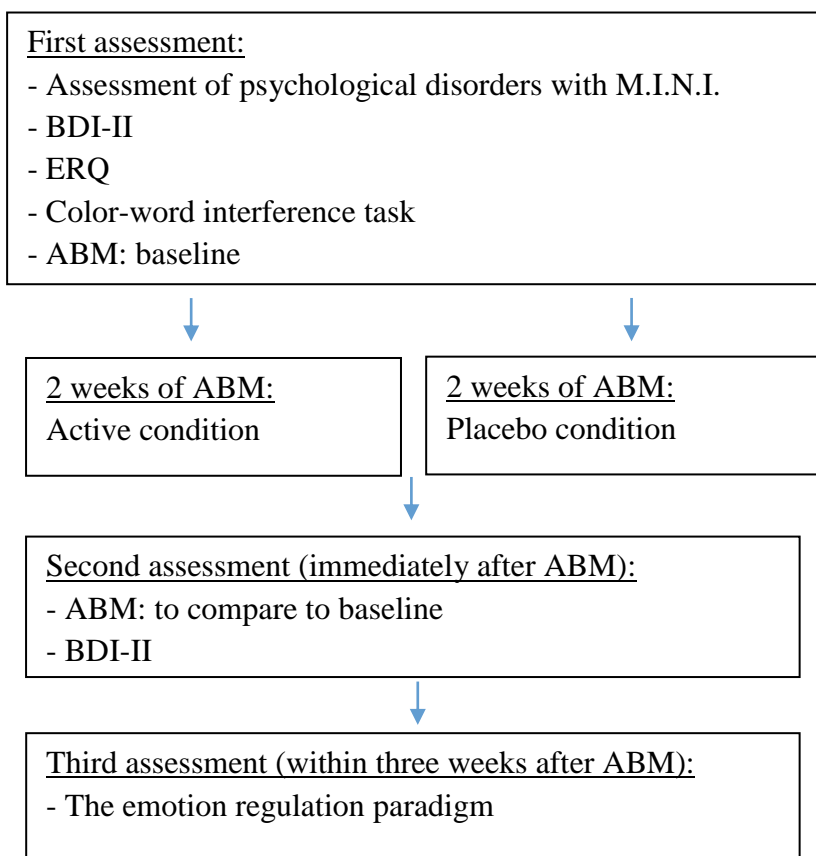


Figure 4: Overview of the design.

2.3 Procedures

2.3.1 The ABM-procedure

The Attention Bias Modification (ABM)-procedure used in this study was identical to the ABM-procedure with pictures in the study by Browning et al. (2012) (See figure 5). This is a variant of the dot-probe paradigm developed by MacLeod et al. (2002) used to describe the negative attentional bias in depression and anxiety. Each trial in ABM begins with a fixation mark presented for 1000 ms on a black screen. Then, two pictures of faces with different facial expressions are presented for 500 or 1000 ms above and below the fixation mark. The pictures appear in pairs of two with the facial expressions being positive and neutral, positive and negative or negative and neutral. A cue (one or two dots) then appears at the same location as one of the pictures. The participant responds to the cue by pressing one of two buttons indicating how many dots they see. There are 96 trials in each session, and the procedure takes about 5-7 minutes to complete.

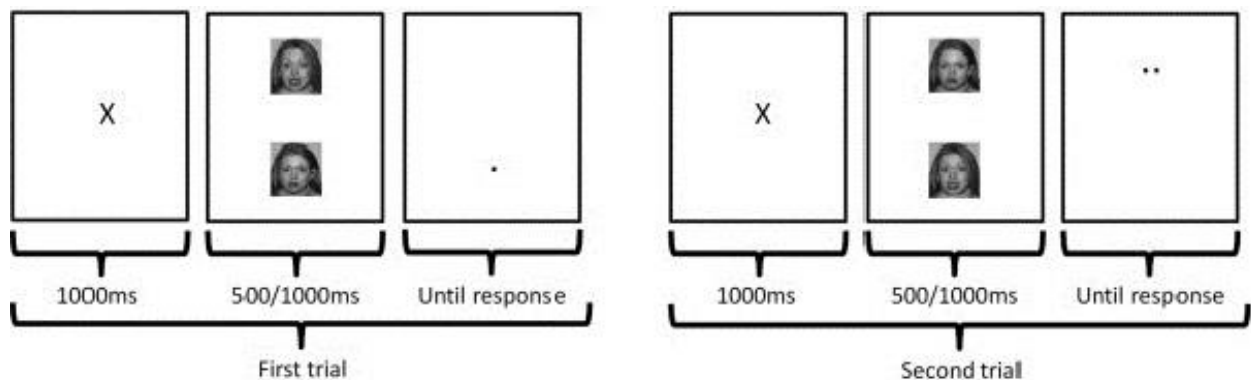


Figure 5: The Attention Bias Modification procedure: A cue will appear at the location of a visual stimuli, and the participant respond to the number of dots. When the cue systematically appears behind the face with a positive emotional expression, attention was believed to be modified toward positive stimuli.

The participants were randomly assigned to one of two different conditions of the ABM-procedure: An active and a placebo condition. The difference between the two conditions was that the cues in the active condition appeared more systematically at the location of a positive facial expression (80 % of the trials), while the cues in the placebo condition did not appear in any systematically pattern. It is believed that the active condition is unconsciously encouraging the individual to automatically direct attention toward positive

stimuli due to the systematic appearance of the cues behind positive stimuli. This is believed to enhance a positive attentional bias. In contrast, when the cues do not appear in any systematic pattern, it is believed that attention is not redirected (Browning et al., 2012).

The ABM sessions at the first and second assessment were identical to the placebo condition described above. They were administered to assess the effect of the ABM itself on attentional bias. It is believed that faster reaction time to cues behind negative emotional stimuli reflects a negative attentional bias, and that the individual is more ready to attend to negative stimuli. In contrast, faster reaction time to cues behind positive emotional stimuli is believed to reflect a positive attentional bias, and that the individual is more ready to attend to positive stimuli.

2.3.2 Clinical and Cognitive Measures

Emotion Regulation Questionnaire

Emotion Regulation Questionnaire (ERQ) (Gross & John, 2003) (Norwegian translation by Engen, Friberg, & Aker) measures the participant's habitual use of the emotion regulation strategies reappraisal and suppression. ERQ is a self-report measure where the participant rates different statements of how they regulate or express emotions on a scale from 1 (strongly disagree) to 7 (strongly agree). ERQ consists of items asking about emotions in general and items asking about negative or positive emotions specifically. The reappraisal scale is made up by items 1, 3, 5, 7, 8 and 10. An example of an item on the reappraisal scale is: "I control my emotions by changing the way I think about the situation I'm in". The suppression scale is made up by items 2, 4, 6 and 9. An example of an item on the suppression scale is: «I control my emotions by not expressing them». The two scales are scored separately, and are not mutually exclusive. Previous studies have demonstrated a good reliability of the two scales (Gross & John, 2003). Both the reappraisal and suppression scale demonstrated a high internal consistency (coefficient alphas ranging from .75 to .80 and .68 to .76 respectively). The test-retest reliability across two months was also good (Cronbach alpha = .69 for both scales).

Color-word Interference Task

The color-word interference task, also known as the Stroop task, from the Delis-Kaplan Executive Function System (D-KEFS) neuropsychological test battery (Delis et al., 2001) was used as a measure of cognitive control. The color-word interference task consists of four templates: One template with colored squares (red, green and blue), one template with words (“red”, “green” and “blue”) printed in black, and two templates with words (red, green and blue) printed in non-matching colors. The color-word interference task has four subsections: 1) Color naming (naming the color of colored squares). This serves as a measure of a fundamental linguistic skill: speed of naming. 2) Word reading (reading words printed in black). This is a measure of another fundamental linguistic skill: speed of reading. 3) Inhibition (naming the color of the ink that words were printed in, when the word and the color do not match). This measures the ability to inhibit the more automatic response of reading the word itself. 4) Inhibition and switching (switching between naming the color of the ink the words were printed in and reading the word itself). This measures both inhibition and cognitive flexibility. The researcher gave instructions to the participants before each trial and timed the participant’s performance. The participants were instructed to do the task as quickly and correctly as he/she could, and to correct the answer and keep on going if he/she made an error. The researcher noted number of seconds to complete the task and number of errors. The raw score of seconds to complete the task was used in the analyses of executive functions. Lower completion time were believed to reflect better performance on the task. A review of MacLeod (1991) on the Stroop task argues that overall, previous studies have demonstrated a reasonably good reliability and validity of the Stroop task.

2.3.3 Outcome Variables

Beck Depression Inventory-II

Beck Depression Inventory-II (BDI-II; Beck et al., 1988) is a widely used self-report questionnaire to assess the intensity of depression or to detect depression in normal populations. A Norwegian version of BDI-II was used in this study. BDI-II consists of 21 items about emotional, physiological and psychological symptoms of depression. Each item provides a score from 0 to 3. The BDI-II score is achieved by adding up the scores on all items. The center for cognitive therapy has proposed cut-off scores for BDI-II with scores

lower than 10 reflecting none or minimal depression, scores ranging from 10-18 reflecting mild to moderate depression, scores ranging from 19-29 as reflecting moderate to severe depression, and scores ranging from 30-63 as reflecting severe depression (Beck et al., 1988). A review of the psychometric properties of BDI found a high internal consistency (Cronbach alpha = .86 for psychiatric patients) (Beck et al., 1988). A high concurrent validity was also found to other clinical ratings of depression (Cronbach alpha of .72 for psychiatric patients) and to Hamilton (Cronbach alpha of .73 for psychiatric patients).

The variable BDI-differential was constructed to reflect changes in depressive symptoms from the first to the second assessment. BDI-differential was obtained by subtracting the BDI-post measure (second assessment) from the BDI-pre measure (first assessment). A positive BDI-differential score was believed to reflect a decrease of depressive symptoms, and a negative score was believed to reflect an increase of depressive symptoms.

$$\text{BDI-differential} = \text{BDI-pre} - \text{BDI-post}$$

The Emotion Regulation Paradigm

A validated emotion regulation paradigm (Phan et al., 2005) (see figure 6) was used as a behavioral measure to answer the research question about the effect of ABM on emotion regulation. The emotion regulation paradigm was administered on a screen in the MRI-scanner at the intervention center at Rikshospitalet or on a computer in the neuropsychological lab. The participants received instructions to the task and practiced different reappraisal strategies with the research assistant outside the MRI-scanner before the task began.

In the emotion regulation paradigm, an instruction first appeared on the screen for 2000 ms instructing the participant to either “look at the picture” or “create distance”. The “look at the picture”-instruction held that the participants should respond in the way that feels natural for them. The “create distance” instruction held that the participant should regulate negative emotions by using reappraisal. Distancing is a reappraisal strategy that involves viewing a stimulus in a detached, objective and impartial manner (Denny, Ochsner, Weber & Wager, 2014). It involves creating a mental space between oneself and the stimuli. Examples of reappraisals could be “It is just a movie” or “It happened a long time ago”. Then, a picture was presented for 6000 ms that either elicited a negative or neutral emotional response.

Examples of pictures believed to elicit negative emotions were pictures of children exposed to violence or war, people crying or people with severe injuries. The emotion regulation paradigm used pictures from two different validated picture sets: The International Affective Picture Schedule (IAPS) (Lang, Bradley & Cubert, 1997) and EmoPicS (Wessa et al., 2010).

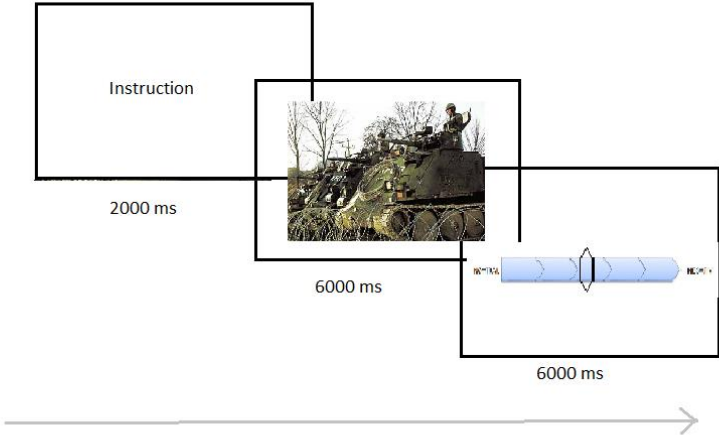


Figure 6: The emotion regulation paradigm. The participants were instructed to «look at the picture» or «create distance» (reappraisal) while looking at an aversive or neutral picture. The participants then reported the degree of negative emotions experienced during the presentation of the picture.

The participants then rated their emotional response to the picture after the picture was presented on a visual analogue scale. The scale was a continuum from neutral to negative (see figure 7), and the participant moved the marker toward neutral or negative by pressing one of two buttons with their right hand. The scale was presented for 6000 ms and the marker started in the middle of the scale.

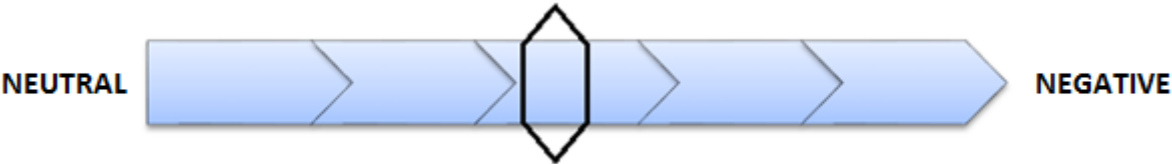


Figure 7: A visual analogue scale. The participants indicated the degree of negative or neutral emotions experienced during the presentation of aversive or neutral pictures.

The emotion regulation paradigm was conducted in two sessions with 36 trials in each session. There were also three training trials in the beginning of each session. The instructions and pictures were presented in pairs: 12 pairs of “look – neutral”, 12 pairs of “look – negative” and 12 pairs of “reappraise – negative”. Approximately 29 of the stimuli pictures were from IAPS and 10 pictures were from EmoPicS in each session. Each session took about 11 minutes to complete. There was a 15-minute break between each session.

The variable reappraisal was constructed from the emotion regulation paradigm. The reappraisal score take individual differences into account by comparing each individual to themselves. Reappraisal was calculated by subtracting the score on the “look-negative” condition from the score on the “reappraise-negative” condition. This score was believed to reflect the ability to successfully use reappraisal. A higher score on this scale was believed to indicate a greater success in using reappraisal.

$$\text{Reappraisal} = \text{“reappraise-negative”} - \text{“look-negative”}$$

2.4 Statistical Analyses

All of the statistical analyses were conducted using the statistical program SPSS 22.0.0.2 for Windows. Descriptive frequency statistics were used to retrieve sample demographics and diagnosis frequency in the sample. Independent samples t-tests were conducted to compare psychometric and clinical characteristics between the two groups before ABM. The effect of ABM on changes in depression and emotion regulation was explored with two-way between subject ANOVA. The mediation hypothesis was tested using linear regression analysis following the four steps of Baron and Kenny (Baron & Kenny, 1986).

The statistical significant-level was set at $p < .05$. The current study is part of a larger and ongoing study, so results at trend level ($p < .1$) were also considered interesting and believed to reflect tendencies in the sample.

3 Results

3.1 Sample Demographic, Psychometric and Clinical Characteristics

The active and the placebo group were compared on demographic, psychometric and clinical characteristics collected before the ABM intervention was implemented to rule out that any preexisting differences between the two groups could explain the outcome on depressive symptoms or emotion regulation. Descriptive statistics revealed that there was a notable difference in the number of participants in the active ($n = 53$) and the placebo ($n = 70$) group. Descriptive statistics also revealed that there were approximately the same percentage of men and women in the active ABM group (28 % men, $n = 15$ and 72 % women, $n = 38$) and placebo group (33 % men, $n = 23$, and 67 % women, $n = 47$).

Separate independent-samples t-tests were run with age, education, reappraisal, suppression, level of depression and measures of executive functions as dependent variables, and with ABM as the independent variable (see table 1). There was no significant difference in age ($t(121) = .124$, $p = .90$) between the active and the placebo group. Education level was measured using the International Standard Classification of Education (ISCED). Here, education levels are divided into 9 categories (Level 1-4 = upper secondary education, level 5-6 = tertiary education 1-4 years, level 7-8 = tertiary education for more than 4 years and level 9 = not else classified) (UNESCO, 2012). There was no significant difference in education level ($t(121) = -.653$, $p = .51$) between the two groups. There was also no difference in the habitual use of reappraisal ($t(121) = -.102$, $p = .92$) or suppression ($t(121) = 1.167$, $p = .25$) between the two groups. A factor analysis of ERQ in this sample showed that the items fitted perfectly in two separate factors. This confirmed the distribution of items into the specific ERQ reappraisal and suppression scales. Reliability analyses revealed that the internal consistency of the reappraisal scale in the current sample was good (Cronbach alpha = .82). The internal consistency of the suppression scale was acceptable (Cronbach alpha = .76). There was no significant difference in levels of depression measured with BDI-II at the first assessment between the active ABM and placebo group ($t(121) = -1.742$, $p = .08$), but a comparison of the means shows a notable, but not significant, difference at trend level ($p < .10$) in levels of depression between the two groups. The effect size was small-medium. Reliability analysis showed that the BDI-II scale had a good internal

consistency (Cronbach alpha = .92) in the current sample. There was no significant difference between the two groups on the color-word interference task on the subscales Stroop color naming scale ($t(121) = .532, p = .60$) and the Stroop word reading scale ($t(121) = .627, p = .53$). There was also no significant difference between the two groups on the Stroop inhibition scale ($t(121) = -.708, p = .48$), or the Stroop inhibition and switching scale ($t(120) = -.372, p = .71$). These independent samples t-tests showed that the randomization into the active and placebo group had the effect as expected of an RCT study, where the participants in the two groups are matched in demographic, psychometric and clinical characteristics.

Table 1: T-tests of demographic, psychometric and clinical characteristics

Outcomes	Group						t	Cohen's d
	Active ABM			Placebo				
	M	SD	n	M	SD	n		
Age	43.4	13.6	53	43.7	12.5	70	.124	.0
Education level	5.8	1.4	53	6.0	1.3	70	-.653	.2
ERQ reappraisal	25.5	6.1	53	25.4	7.6	70	-.102	.0
ERQ suppression	13.3	5.2	53	14.5	5.5	70	1.167	.2
BDI-II pre	16.5	11.1	53	13.1	10.0	70	-1.742	.3
Stroop color naming	31.1	8.0	53	32.0	9.2	70	.532	.1
Stroop word reading	23.2	7.2	53	24.2	9.1	70	.627	.1
Stroop inhibition	53.6	16.1	53	51.7	13.8	70	-.708	.1
Stroop inhibition and switching	60.2	16.8	53	60.1	15.6	70	-.372	.0

* $p < .05$.

3.2 The Effect of ABM on Depressive Symptoms

To test the hypothesis that ABM had an effect on depressive symptoms after a 2-week training period, a two-way between subject ANOVA was conducted. The categorical variable ABM was set as the fixed factor and was made up by the two conditions: an active ABM condition and a placebo condition. The calculated BDI-differential score was used as the dependent variable. The analysis revealed a trend toward a significant main effect of ABM on changes in depressive symptoms ($F(1, 118) = 3.37, p = .07$). The effect size was small (partial eta squared = .03). Explained variance was low ($R^2 = .028$), which means that ABM explains 2.8% of the variance in the decrease in depressive symptoms. This result is a promising indication that individuals in the active ABM condition may have experienced a greater decrease of depressive symptoms than individuals in the placebo condition (see figure 8).

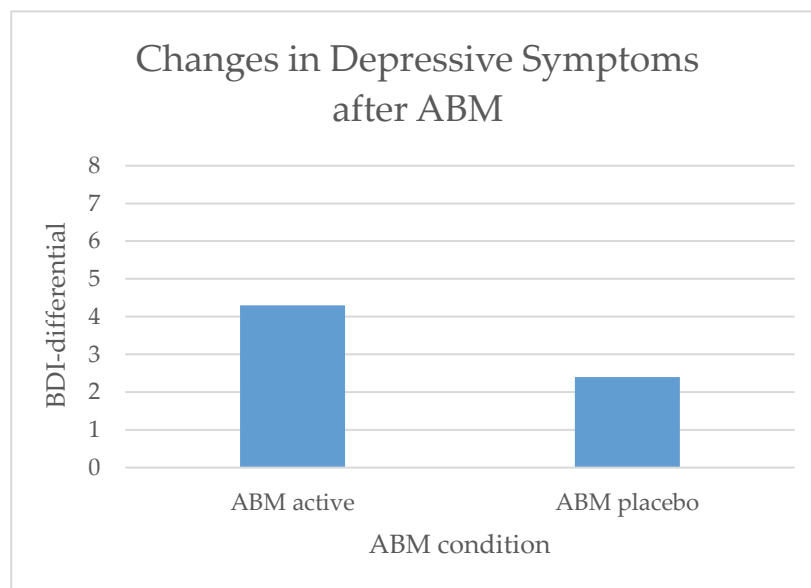


Figure 8: The effect of ABM on depressive symptoms. The active ABM group showed a trend toward a greater decrease in depressive symptoms than the placebo group.

3.3 The Effect of ABM on Emotion Regulation

3.3.1 Validating the Emotion Regulation Paradigm

A one-way repeated measure ANOVA was carried out in the whole sample as a manipulation check by comparing affective ratings on the three different conditions on the emotion regulation paradigm (“look-neutral”, “look-negative” and “reappraise-negative”), (see figure 9). There was a significant difference in affective ratings between the three conditions: “look-neutral” (M = 7.2, SD = 7.0), “look-negative” (M = 60.7, SD = 14.5) and “reappraise-negative” (M = 42.9, SD = 17.0), (Wilk’s Lambda = .06, F(2, 121) = 925.8, p=.00). The effect size was large (multivariate partial eta squared = .94). The same was true within both the active ABM group (Wilk’s Lambda = .04, F(2, 51) = 568.2, p = .00, multivariate partial eta squared = .96) and the placebo group (Wilk’s Lambda = .074, F(2, 68) = 425.3, p = .00, multivariate partial eta squared = .93). This finding indicates that the emotion regulation manipulation worked as expected with the three different conditions eliciting different emotional responses (degree of reported negative affect).

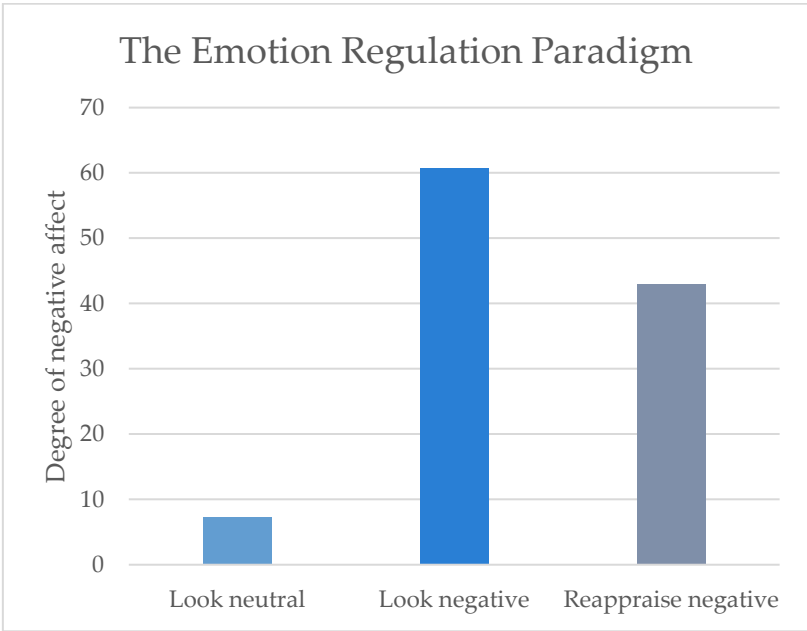


Figure 9: Validating the emotional regulation paradigm. There was a significant difference in the degree of negative affect reported on the three different conditions in the emotion regulation paradigm.

3.3.2 The Effect of ABM on Reappraisal

A two-way between groups ANOVA was conducted to explore the impact of the predictor variable ABM on the outcome variable reappraisal. The categorical variable, ABM, was set as the predictor variable. The outcome variable, reappraisal, was a continuous variable ranging from 0 – 100 calculated from the emotion regulation paradigm as described earlier. There was no statistically significant main effect of ABM on reappraisal ($F(1, 121) = 1.12, p = .29$). This result indicates that ABM had no direct effect on the functional emotion regulation strategy reappraisal, and that the active ABM group was not better than the placebo group to use reappraisal after two weeks of ABM (see figure 10).

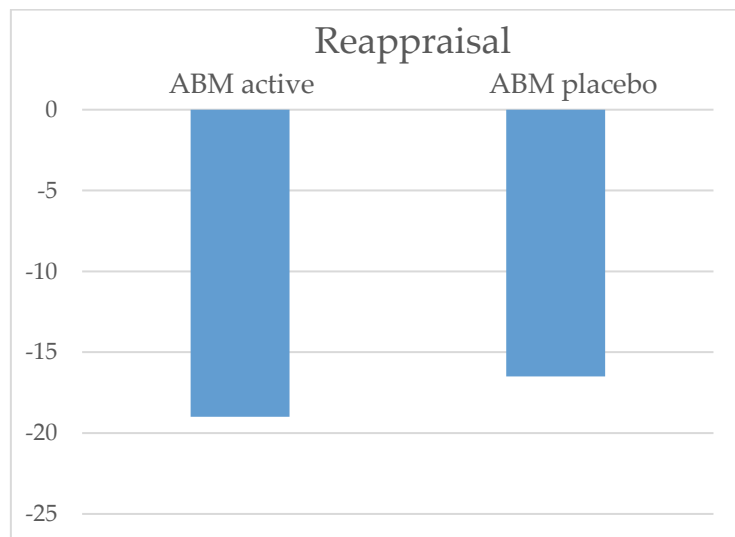


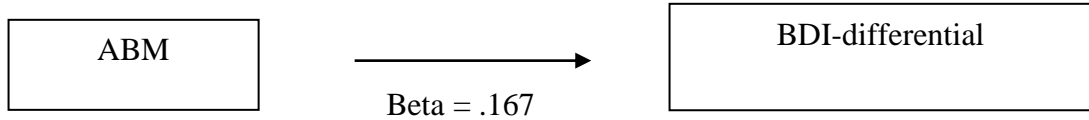
Figure 10: The effect of ABM on reappraisal. There was no significant difference between the active ABM group and the placebo group.

3.4 Mediation Analysis

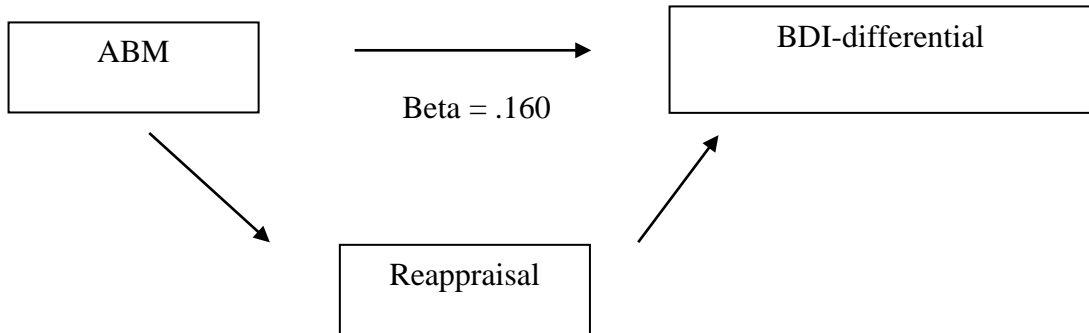
The third hypothesis was that emotion regulation (here: reappraisal) mediates the relationship between ABM and depressive symptoms. Linear regression in SPSS was used to test this mediation model by following Baron and Kenny's four steps (Baron & Kenny, 1986):

- 1) Determining the relationship between the predictor variable and the outcome variable.
- 2) Determining the relationship between the predictor variable and the mediation variable.
- 3) Determining the relationship between the mediation variable and the outcome variable.
- 4) Using a multiple regression to determine the relationship between the predictor variable and the outcome variable when controlling for the mediation variable. If the strength of the relationship between the predictor variable and the outcome variable is reduced, yet still significant when controlling for the mediation variable, this would indicate that the mediation variable is partially mediating the relationship between the predictor variable and the outcome variable.

A mediation model was tested with reappraisal as the mediation variable between ABM and depressive symptoms. The calculated BDI-differential score was set as the outcome variable, and ABM was set as the predictor variable. The hypothesis that reappraisal mediates the relationship between ABM and depressive symptoms was not supported (see figure 11). The predictor variable, ABM, was related to the outcome variable, BDI-differential at trend level ($F(1, 118) = 3.37, p = .07$). The predictor variable, ABM, was not significantly related to the mediating variable, reappraisal ($F(1,121) = 1.12, p = .29$). Reappraisal was not significantly related to the outcome variable BDI-differential ($F(1, 118) = .82, p = .37$). Then, a multiple regression was conducted with both ABM and reappraisal as predictor variables and BDI-differential as the outcome variable. The overall equation was not significant; ($F(2,117) = 1.97, p = .14$). Most importantly, there was no reduction in the strength of the relationship between ABM and BDI-differential in the multiple regression (Beta = .160; $t = 1.76, p = .08$) compared to the direct relationship in the linear regression (Beta = .167). These results suggest that reappraisal does not mediate the relationship between ABM and changes in depressive symptoms.



a) Direct relationship



b) Mediated relationship

* $p < .05$

Figure 11: Mediation model. Reappraisal did not mediate the relationship between ABM and changes in depressive symptoms.

4 Discussion

4.1 Main Findings

4.1.1 Decrease in Depressive Symptoms after ABM

Results from the current study yielded promising support to the hypothesis that ABM lead to a decrease in depressive symptoms. Results showed that individuals in the active ABM group had trend toward a greater decrease in depressive symptoms than individuals in the placebo group immediately after the implementation period had ended. In other words, ABM led directly to a decrease in symptoms of depression. This finding was not statistically significant at a 95% significant level, but represent an interesting and promising finding with a trend toward a significant result ($p < .10$). The result of the current study is in line with previous findings that ABM over multiple sessions promotes a decrease in depressive symptoms (Yang, et al., 2015; Baert et al., 2009). What separates the current study from the studies above is that the current sample mostly included individuals who were not currently depressed, and consisted of individuals at high risk of depression recurrence. The current sample is also not only based on college students, but has a sample with a larger age range (21-71 years) than the other studies. It was interesting that a decrease in depressive symptoms after ABM was also found in this sample, and that the effect might not be limited to currently depressed individuals.

The current study stands in contrast to studies that found no difference in depressive symptoms immediately after the implementation of ABM (Beevers et al., 2015; Kruijt et al., 2013). A possible explanation for this is the intensity and number of ABM sessions the participants conducted in these studies. In the Beevers et al. (2015) study, participants conducted 8 session of ABM in a 4-week period (low intensity), and the participants in the Kruijt et al. (2013) study conducted a single-session ABM. The current study has a longer and more frequent ABM implementation (28 sessions during 2 weeks). The result of the current study is therefore in line with other studies with a longer and more intense ABM implementation. E.g. Yang et al. (2015) found a significant decrease in depressive symptoms right after a two-week period where 8 sessions of ABM were completed. Baert et al. (2009) had 10 sessions of ABM, and found a decrease in depressive symptoms for individuals with moderate to severe symptoms. This argument is in line with the study by Browning et al.

(2012) that compared different versions of ABM, and that found that ABM with longer implementation duration had better effect than single session ABM.

The current study used the same ABM-procedure as Browning et al. (2012). The Browning et al. (2012) study found a decrease in depressive symptoms first one month after ABM had ended, and not immediately after the implementation period. This stand in contrast to the current study which found a trend toward a decrease in depressive symptoms immediately after ABM and other studies that found an effect of ABM immediately after implementation. This also raises a question about whether it is possible that a statistically significant difference in depressive symptoms first will appear a month after ABM. One possible explanation for this is the clinical characteristics of the sample. The study by Browning et al. (2012) consisted of previously depressed individuals, currently in remission. The other studies consisted of currently depressed individuals. The current study represents a sample of individuals with a history of depression where some were currently depressed and others were not. A possible explanation for the delayed effect found in the Browning et al. (2012) study could be that the therapeutic effect of ABM on depressive symptoms is delayed in individuals who are not currently depressed. Another possible way to understand this delayed effect is that the enhancement of emotional outcomes continues beyond the completion of an attentional bias modification-intervention (MacLeod, Koster & fox, 2009). It could be that the induced cognitive change remains stable, or even decline, with time and that any detected increases in emotional benefits reflects an interaction between the participant, their altered emotional dispositions and the environment. The individual could encounter situations that reveal their altered emotional dispositions to them which then becomes available for consciousness and self-reflection, and are therefore available to report self-reports questionnaires after a month.

Another possible explanation for the trend level effect of ABM on depressive symptoms could be that depressive symptoms were measured with a self-report questionnaire. Self-report questionnaires are based on the individual's own perception and judgement of its current state. Therefore, BDI-II does not include clinical ratings of e.g. agitation or psychomotor retardation that could only be observed by another person or clinician. Another well-established depression rating scale, Hamilton (1960), is conducted as an interview between a clinician and the patient and include such behavioral measures. Here, the clinician's assessment of the patient's psychomotor symptoms of depression also plays a crucial role in the assessment of depression. Another difference between BDI-II and Hamilton

is that while BDI-II focus on the individual's subjective experience of depression, does Hamilton focus on behavioral and somatic symptoms of depression (Steer, Beck, Riskind & Brown, 1987). Even though there is a difference between which aspect of depression these two depression rating scales tap, did a review of psychometric properties of BDI (Beck et al., 1988) find a high concurrent validity with Hamilton (Cronbach alpha of .73 for psychiatric patients and .74 in a non-psychiatric sample). Steer et al. (1987) found in their study that the Pearson product-moment correlation between the BDI and Hamilton was .54. Their study also supported the idea that BDI taps more cognitive aspects of depression and that Hamilton taps more somatic aspects of depression. Steer et al. (1987) then posits that a combination of these two measures would give a comprehensive picture of depression (unless the clinician is more interested in one or the other mentioned aspects of depression). In a commentary on the Cognitive Bias Modification (CBM)-research, MacLeod et al. (2009) did encourage fellow researchers in the CBM field to carry on using well-established assessment instruments like BDI-II and Hamilton. At the same time, they were concerned about the fact that many of these instruments relies heavily on self-report, and that this may elicit demand effects where participants report symptom change because they feel like they are expected to do so. MacLeod et al. (2009) encouraged future researchers to routinely interrogate participants' expectancies to assess the likelihood that demand effects contribute to their report of symptom change. The authors also encouraged future researchers to include behavioral and somatic measures, due to the possibility that CBM may also influence other interesting emotional or decision processes even when the symptoms themselves are unaffected.

It should be noted that an assumption was made in the current study about the ability of ABM to actually modify negative attentional bias. The commentary by MacLeod et al. (2009) also addressed this issue, and stressed the necessary for CBM researcher to first demonstrate that the cognitive bias modification procedure at hand succeeded in modifying what it aimed to modify. By doing this, one could with more confidence determine that the symptom change observed after CBM actually were influenced by changes in cognition and not something else. MacLeod et al. (2009) also stressed that changes in cognition should be assessed with a cognitive experimental task and not by self-report questionnaires. Self-report measures have been revealed to yield inaccurate measures of cognitive processes (Nisbett & Wilson, 1977), so an experimental task that objectively and reliably measure the cognitive processes targeted should be included. An analysis of the effect of ABM on the negative attentional bias in the active ABM group should be included in future research so that the

changes in depressive symptoms observed at trend level with confidence could be attributed to the ABM-procedure. This should be done by comparing the ABM session at the second assessment to the baseline set in the first assessment.

Even though the current study supported the hypothesis that ABM has a direct effect on depression, the ABM-procedure itself only explained a small portion of the decrease in depressive symptoms. This raises questions about the mechanisms behind ABM, and whether there are other variables that the ABM might work through that could contribute to explain the variance in the outcome variable. Post hoc analyses revealed that individuals with more depressive symptoms before implementation of ABM had a significantly greater decrease in depressive symptoms than individuals with less symptoms before ABM. However, this was not exclusive to the active ABM group, but is still an interesting finding for future research. Post hoc analyses also revealed that executive functions might be another interesting third variable to further explore as possible way that ABM work. Post hoc analyses found a trend toward a significant effect of Stroop inhibition on decrease of depressive symptoms. Individuals with a greater ability to inhibit more automatic responses had a greater decrease in depressive symptoms after ABM than individuals with a lower ability to inhibit more automatic responses. This trend was not found in the placebo group. These post hoc analyses may reveal interesting variables to further explore to gain a better understanding of the ABM-procedure.

4.1.2 ABM had no Direct Effect on Reappraisal

The second hypothesis that ABM would enhance the use of the functional emotion regulation strategy, reappraisal, was not supported. The active ABM group was not better than the placebo group to successfully use reappraisal when instructed to in the emotion regulation paradigm. This study stands in contrast to other studies that have found an effect of ABM on emotion regulation (Wadlinger & Isaacowitz, 2008; Dandeneau et al., 2007; Johnson, 2009).

One possible explanation for this difference include how reappraisal was operationalized and measured. The current study relied on a visual analogue scale where the participants rated their current emotional experience from neutral to negative. This way of operationalizing reappraisal only reflected the self-reported emotional valence of an experienced emotion. It did not include measures of emotional responses that are not available for consciousness. Other studies have also included other measures like eye gaze, state frustration or measures of real life function as indications of successful emotion regulation.

Questions could be raised about whether including physiological measures, e.g. heart rate and respiration, could have represented a more appropriate operationalization of reappraisal.

Another possible explanation for the negative finding in the current study could be that demand effects was induced by the instructions in the “reappraise-negative” condition. Both groups were successfully able to follow the different instructions on the emotion regulation paradigm and an expected pattern of self-reported degree of negative affect emerged on the three different conditions. Participants in both groups reported the highest degree of negative affect in the “look-negative” condition, a somewhat lower degree of negative affect in the “reappraise-negative” condition and the lowest degree of negative affect in the “look-neutral” condition. The instructions in the “reappraise-negative” condition told the participants to create distance, and the participants might have felt that they were expected to report a lower degree of negative effect after this instruction, creating demand effects. A study by Ehring et al. (2010) found that depressed individuals were able to use reappraisal just as well as never depressed individuals when instructed to. The difference between depressed and never-depressed first emerged in a spontaneous condition where they found that depressed individuals used suppression more than never depressed individuals. The authors of this study suggested that depressed individuals had the same ability to use reappraisal as never depressed individuals when instructed to, but had a harder time choosing this more functional emotion regulation strategy spontaneously. This study may help explain why no difference was found between the individuals in the active ABM group and the placebo group in their ability to use reappraisal. The participants could have done just as well because they were instructed to use reappraisal, and felt like they were expected to report a lower degree of negative affect when looking at the aversive pictures.

4.1.3 Reappraisal did not Mediate the Relationship Between ABM and Depressive Symptoms

The second goal of the current study was to gain further understanding about the mechanisms behind the ABM-procedure and its effect on depressive symptoms. The third hypothesis that a functional emotion regulation strategy mediates the relationship between ABM and depressive symptoms was not supported. Reappraisal did not mediate the relationship between ABM and the decrease in depressive symptoms. This implies that ABM does not lead to a decrease in depressive symptoms by enhancing the individual’s ability to use reappraisal. This result could be expected when ABM was not found to have a direct effect on

reappraisal. Based on this result should it also be expected that being better at using reappraisal is not the mechanism that the ABM works through when it leads to a decrease in depressive symptoms. No other studies were found that had ran similar mediator analyses in the same kind of clinical sample with individuals at high risk of depressive recurrence. Other studies on individuals at high risk of anxiety found that ABM had an effect on how information was processed, which in turn caused changes in state anxiety (MacLeod et al., 2002).

It should be noted that reappraisal is only one example of emotion regulation. The current study implied that ABM does not enhance the successful use of the functional emotion regulation strategy reappraisal, but conclusions about the role of other emotion regulation processes or skills, like suppression, rumination etc. cannot be drawn from the current study. A study by Nolen-Hoeksema and Aldao (2011) found that dysfunctional emotion regulation strategies was significantly related to depression, and that functional emotion regulation strategies was not related to lower levels of depression. This finding could help us to understand why reappraisal was not found to have the decreasing effect on depressive symptoms as hypothesized. Dysfunctional emotion regulation strategies might be a more interesting target of ABM than functional emotion regulation strategies. Criticism about the operationalization of reappraisal has already been made, and if this is true, would this also help explain why reappraisal was not found to mediate the relationship. The current study did also not include a spontaneous condition where the individual's ability to choose different emotion regulation strategies could have been observed. Like in the study by Ehring et al. (2010), the different use of emotion regulation strategies between depressed individuals and never depressed individuals appeared in a spontaneous condition where the depressed individuals used a dysfunctional emotion regulation strategy, and not when the individuals were instructed to use a functional emotion regulation strategy. Therefore, it would have been interesting to see whether ABM might affect how individuals spontaneously choose an emotion regulation strategy, or whether ABM could affect the use of dysfunctional emotion regulation strategies. This might represent other possible ways to explore the role of emotion regulation in depression in the ABM-research.

4.2 Clinical Implications

A goal of recent ABM-research has been to explore the possibility that ABM could be an effective intervention to prevent depression recurrence. As previously mentioned, clinicians should not only be concerned about treating the current depression at hand, but should also be concerned about preventing depression recurrence. The current study contributes to a growing body of research on this issue and yield promising support to ABM as a novel intervention to prevent depression recurrence. The ABM-procedure is a cost-effective intervention and can easily be administrated by patients themselves at home on a computer. ABM could therefore represent an intervention that is easy to administrate to out-patients in remission who have suffered from multiple episodes of depression to prevent subsequent episodes.

In the work of preventing depression recurrence, one could argue that targeting important risk factors of depression could be effective. Residual symptoms are named as an important marker of depression, and many individuals that has suffered from depression were found to have residual symptoms in remission (Paykel, 2008). Residual symptoms include the typical symptoms of depression (except those typical of severe depression), but the individual does not meet the diagnostic criteria for a depressive episode (Paykel, 2008). Studies have found that residual symptoms are an important predictor of depression recurrence. In a study by Paykel et al. (1995), 76 % of the participants *with* residual symptoms at remission relapsed within the next 10 months. In comparison, only 25 % of the participants *without* residual symptoms at remission relapsed within the same period. Patients in remission with residual symptoms continued to have more depressive symptoms and impaired social functioning long term (Kennedy & Paykel, 2004). This implies that patients in remission may need treatment tailored for their specific psychological characteristics to prevent depression recurrence. The decrease in depressive symptoms found in the active ABM group is a promising indication that the ABM-procedure may affect this important marker of depression recurrence.

In the current study, symptoms of depression were measured with BDI-II as in the study by Browning et al. (2012). BDI-scores above 9 were believed to indicate the existence of residual symptoms as long as the individual did not meet the diagnostic criteria for current depression. Both the active ABM and placebo group were found to have BDI-scores above 9 in the current study, indicating the existence of residual symptoms in both groups. It should be noted that the sample also included individuals that were currently depressed. This would increase the mean BDI-score before implementation of ABM and make it harder to interpret whether the heightened BDI in the sample truly reflect the existence of residual symptoms or

whether it reflects the inclusion of currently depressed individuals. It should also be noted that the mean BDI-score (degree of depressive symptoms) was slightly higher in the active ABM group than in the placebo group before ABM was implemented. A possible explanation for the heightened BDI in the active ABM group could be due to the fact that some of the participants were currently depressed at the first assessment (9 currently depressed in the active ABM group and 5 currently depressed in the placebo group). Currently depressed individuals are expected to have a higher BDI-score, so more currently depressed individuals might lead to a higher mean BDI in the active ABM group. Another possible explanation for the difference in BDI-scores at the first assessment could be the difference in number of participants in the two groups (53 participants in the active ABM group and 73 participants in the placebo group). The difference in BDI at the first assessment was not significant, yet worth commenting.

ABM may also represent a promising alternative to medical treatment of depression recurrence. Medication has been shown to prevent relapses of depression as long as the individual continues to take the medication (Dobson et al., 2008). Less is known about the preventing effects of antidepressants when they are discontinued. Medical treatment may also have undesired side effects. Dobson et al. (2008) compared the effect of cognitive therapy, antidepressant medication and behavioral activation on preventing depression recurrence. They found that individuals receiving cognitive therapy had as good effect of the treatment as individuals on continued medical treatment. On the other hand, when studying the long term effects of previous treatment, individuals on medication that withdrew onto a placebo pill during the study had more relapses compared to individuals who had previously received cognitive therapy. When preventing depression recurrence, medication might not be the intervention of choice. The Norwegian health department (Helsedirektoratet, 2015) has stated that there should be a medication free treatment alternative available for individuals to choose among. This calls for alternative treatments that prevents depression recurrence long term. Previous studies have showed that the effect of ABM still persist after implementation, or that it actually occurs 1 month after implementation. It then seems worthwhile to further explore the long term effects of the ABM-procedure as a promising medical free treatment alternative to prevent depression recurrence.

A commentary on CBM-research by MacLeod et al. (2009) argued that even though ABM or other CBM-procedures are cost-effective and home based interventions that are easy to implement, should they not be understood as alternatives to *replace* other traditional and

evidence based treatments (e.g. cognitive behavioral therapy (CBT) or medical treatment). Rather, they suggested that CBM could represent interventions to boost the therapeutic effects of therapy. While the CBM-procedure works by targeting automatic cognitions that are unavailable for consciousness and is believed to modify attention at an unconscious level, does traditional therapy aim to modify attention or other cognitive processes explicitly with therapeutic interventions. The question is whether this combination of implicit and explicit interventions to modify attention have a better effect than explicit or implicit interventions alone? The rationale behind this is that CBM could make the individual more available for interventions of additional therapy. One could ask hypothetically if changes in cognition induced by CBM could enhance the effect of e.g. CBT? One of the goals of CBT is to change the way the individual think about or perceive a situation to change unhelpful thinking. One could argue hypothetically that implicitly enhancing an attentional bias toward positive information could make the individual more available for cognitive interventions with the goal of enhancing helpful thinking. Another ongoing part of the study “Secondary prevention of depression applying an experimental Attentional Bias Modification procedure” by Landrø and Harmer is currently being conducted by PhD-candidate Tom Østergaard in Arendal, Norway called “Secondary prevention of depression through group-based Acceptance and Commitment Therapy preceded by an experimental Attentional Bias Modification procedure”. Østergaard studies the combined effect of ABM and Acceptance and Commitment Therapy (ACT) on depression. He hypothesizes that ABM will boost the effect of ACT by inducing a positive attentional bias to make the individual more available for the ACT-interventions. The goal of ACT is to enhance psychological flexibility which will reduce experiential avoidance (The tendency to engage in behaviors to avoid, alter or control unpleasant thoughts, feelings or physiological sensations). Experiential avoidance has been suggested to enhance the pain and suffering of depressive symptoms (Yovel & Bigman, 2012). Inducing a positive attentional bias could hypothetically help the individual to increase cognitive flexibility by not being stuck in a negative attentional bias. These are only two examples of how, theoretically, the ABM procedure could boost the effect of additional therapy. More research is needed to test these hypotheses.

4.3 Strengths and Limitations

One of the strengths of the current study is its RCT-design. RCT studies are known for being the gold standard within clinical trials and are often used when testing new treatments within the field of medicine. The RCT design is based on randomization of participants into an active and a placebo treatment condition. The randomization keeps everything but the clinical intervention constant and is the same in the two groups that are compared, so that the observed effects may be easily attributed to the treatment itself. It should then be expected that any observed difference in depressive symptoms or emotion regulation after the ABM intervention, could be ascribed to the ABM-procedure itself and not something else. In previous studies on cognitive bias modification (CBM) has the CBM-intervention been implemented in a lab. A commentary on the CBM research by MacLeod et al. (2009) suggests the intervention has to be implemented in a real-life setting, not in a lab, if one want to be able to generalize the effect of the intervention into real-life settings. In the current study was ABM conducted by the individuals at home on a laptop. This home-delivered component may enhance the generalization from an experimental to a real-life environment.

A second strength of the study is that it studies the effect of ABM within a clinical sample of individuals at high risk of depression recurrence. This group is useful to study when the goal is to prevent depression recurrence. Previous studies on attention bias modification has often been executed with individuals with anxiety or anxiety vulnerability, so the results on these studies cannot be directly translated to individuals with depression. On the other hand, a limitation of the sample itself is that it could have been more homogeneous to represent individuals at high risk of depression recurrence that are not currently depressed. Some individuals in this study had a history of multiple episodes of depression, others a history of only one previous episode, and some participants were also currently depressed. If the currently depressed individuals and the not currently depressed individuals actually are two different clinical groups with different psychological or psychometric characteristics, could this make the sample less homogeneous. The same could be said about individuals with a history of only one previous episode of depression and individuals with a history of two or more episodes of depression. It would be interesting to study a more homogeneous sample with individuals at risk of depression recurrence defined as individuals with a history of two or more episodes of depression who are not currently depressed.

Another limitation to the study is the power of the statistical analyses. The power of the analyses conducted were small (the effect of ABM on decrease of depressive symptoms =

.45, the effect of ABM on reappraisal = .18). The statistical power should be .80 or greater. With the sample size in this study ($n = 123$) is the probability of detecting a possible effect of the intervention low. It is a heightened probability of making type II errors by accepting the null hypothesis (that there is no difference between the two groups) and rejecting the alternative hypothesis (that there is a difference between the two groups), when the alternative hypothesis is actually true. The chance to make false conclusions about the existence of any differences between the two groups is heightened. The probability of finding effects of ABM on depressive symptoms and reappraisal is therefore small in the current sample. Due to the low power of the analyses, results on trend level were also considered interesting.

4.4 Suggestions for Future Research

The current study is one of the first studies on ABM and emotion regulation in individuals at high risk of depression recurrence and raises several questions for future research. The finding that ABM did not enhance reappraisal and the results from the mediator analyses still leaves unanswered questions about the mechanisms behind ABM. The current study did not include measures of dysfunctional emotion regulation, and it would be interesting to further explore the role of emotion regulation by studying the role of other emotion regulation processes.

Another suggestion for future research is to gain more knowledge about individual differences in the therapeutic effects of ABM. Could some individuals with specific clinical, psychometric or psychological characteristics have a better effect of ABM than others? E.g. could executive functions or depression level before implementation enhance the individual's effect of ABM? Clinicians today are concerned about tailoring treatment to the individual. By gaining knowledge about who benefits more from ABM, could clinicians more easily know which patients to assign and recommend the ABM-procedure to.

ABM has been suggested to represent an intervention that can be administrated in the combination with other traditional depression treatments. The rationale behind this is that ABM could make the individual more available to additional treatment by enhancing a positive attentional bias. Whether ABM could boost the effect of additional treatment could therefore be an interesting question for future research.

4.5 Conclusions

Conclusions that can be drawn from the current study are that the ABM-procedure may enhance a decrease in depressive symptoms, but does not enhance the ability to use reappraisal on a subsequent emotion regulation task. The ABM-procedure did not decrease residual symptoms of depression by enhancing the ability to use reappraisal. This leaves open the possibility to further explore the mechanisms of ABM. Excluding reappraisal as a possible mediator between ABM and depressive symptoms may also be a notable contribution to this issue. More research is also needed to gain a better understanding of who could benefit more from ABM to individualize treatment of depression. Overall, the current study carries implications that the ABM-procedure could represent a promising intervention to prevent depression recurrence due to its effects on residual symptom of depression.

Literature

- Aker, M., Harmer, C., & Landrø, N.I. (2014). More Rumination and Less Effective Emotion Regulation in Previously Depressed Women with Preserved Executive Functions. *BMC Psychiatry, 14*(334), 1-10.
- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation Strategies Across Psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*(2), 217-237.
- Baert, S., Raedt, R.D., Schacht, R., & Koster, E.H.W. (2010) Attentional Bias Training in Depression: Therapeutic effects depend on depression severity. *Journal of Behavior Therapy and Experimental Psychology, 41*(3), 265-274.
- Baron, R.M., & Kenny, D.A. (1986). The Moderator-Mediator Variable Distinction in Social Psychological Research: Conceptual, strategic and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173-1182.
- Beard, C., Sawyer, A.T., & Hofmann, S.G. (2012). Efficacy of Attention Bias Modification Using Threat and Appetitive Stimuli: A meta-analytic review. *Behaviour Therapy, 43*(4), 724-740.
- Beck, A.T. (2008). The Evolution of the Cognitive Model of Depression and its Neurobiological Correlates. *The Journal of American Psychiatry, 165*(8), 969-977.
- Beck, A.S., Steer, R.A., & Garbin, M.G. (1988). Psychometric Properties of the Beck Depression Inventory: Twenty years of evaluation. *Clinical Psychology Review, 8*(1), 77-100.
- Beevers, C.G., Clasen, P.C., Schnyer, D.M., & Enok, P.M. (2015). Attention Bias Modification for Major Depressive Disorder: Effects on attention bias, resting state, and symptom change. *Journal of Abnormal Psychology, 124*(3), 463-475.
- 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.

- Dandeneau, S.D., Baldwin, M.W., Baccus, J.R., Sakellaropoulou, M., & Pruessner, J.C. (2007). Cutting Stress off at the Pass: Reducing vigilance and responsiveness to social threat by manipulating attention. *Journal of Personality and Social Psychology*, 93(4), 651-666.
- Delis, D.C., Kaplan, E., & Kramer, J.H. (2001). *Delis-Kaplan Executive Function System (D-KEFS) examiner's manual*. San Antonio, TX: The Psychological Corporation.
- Denny, B.T., Ochsner, K.N., Weber, J., & Wager, T.D. (2014). Anticipatory Brain Activity Predicts the Success or Failure of Subsequent Emotion Regulation. *Social, Cognitive and Affective Neuroscience*, 9(4), 403-411.
- Dobson, K.S., Dimidjian, S., Kohlenberg, R.J., Rizvi, S.L., Hollon, S.D., Schmaling, K.B., Gallop, R.J., Gollan, J.K., Dunner, D.L., & Jacobson, N.D. (2008). Randomized Trial of Behavioral Activation, Cognitive Therapy and Antidepressant Medication in the Prevention of Relapse and Recurrence in Major Depression. *Journal of consulting and clinical psychology*, 76(3), 468-477.
- Donaldson, C., Lam, D., & Mathews, S. (2007) Rumination and Attention in Major Depression. *Behavior Research and Therapy*, 45(11), 2664-2678.
- Ehring, T., Tuschen-Caffier, B., Schnülle, J., Fischer, S., & Gross, J.J. (2010). Emotion Regulation and Vulnerability to Depression: Spontaneous versus instructed use of emotion regulation and reappraisal. *Emotion*, 10(4), 563-572.
- Everaert, J., Mogoase, C., David, D., & Koster, E.H.W. (2015) Attention Bias Modification via Single-sessions Dot-probe Training: Failures to replicate. *Journal of Behavior Therapy and Experimental Psychology*, 49(part A), 5-12.
- Feliciano, L., Renn, B.N., & Areán, P.A. (2012). Mood Disorders: Depressive disorders. In Hersen, M. & Beidel, D.C. (Red.), *Adult psychopathology and diagnosis* (p. 317-356). Hoboken: John Wiley & sons, Inc.
- Garnefski, N., & Kraaij, V. (2007). The Cognitive Emotion Regulation Questionnaire; psychometric features and prospective relationships with depression and anxiety in adults. *European Journal of Psychological Assessment*, 23(3), 141-149.

- Gotlib, I.H., & Joormann, J. (2010). Cognition and Depression: Current status and future directions. *Annual Review of Clinical Psychology*, 6, 285-312.
- Gross, J.J. (1998). The Emerging field of Emotion Regulation: An integrative review. *Review of General Psychology*, 2(3), 271-299.
- Gross, J.J. (2001). Emotion Regulation in Adulthood: Timing is everything. *Current Directions in Psychological Science*, 10(6), 214-219.
- Gross, J.J., & John, O.P. (2003). Individual Differences in two Emotion Regulation Processes: Implications for affect, relationships and well-being. *Journal of Personality and Social Psychology*, 85(2), 348-362.
- Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, 23, 56-62.
- Hasin, D.S., Goodwin, R.D., Stinson, F.S., & Grant, B.F. (2005). Epidemiology of Major Depressive Disorder: Results from the national epidemiologic survey on alcoholism and related conditions. *Archives of General Psychiatry*, 62(10), 1097-1106.
- Helsedirektoratet. (2015). *Medikamentfrie tilbud i psykisk helsevern - oppfølging av oppdrag 2015*. Hentet 2.8.16 fra <https://www.regjeringen.no/no/dokumenter/medikamentfrie-tilbud-i-psykisk-helsevern---oppfolging-av-oppdrag-2015/id2464239/>
- John, O.P., & Gross, J.J. (2004). Healthy and Unhealthy Emotion Regulation: Personality process, individual differences and life span development. *Journal of Personality*, 72(6), 1301-1333.
- Johnson, D.R. (2009). Goal-directed Attentional Deployment to Emotional Faces and Individual Differences in Emotion Regulation. *Journal of Research in Personality*, 43(1), 8-13.
- Joorman, J., & D'Avanzato, C. (2010). Emotion Regulation in Depression: Examining the role of cognitive processes. *Cognition and Emotion*, 24(6), 913-939.
- Joormann, J., & Gotlib, I.H. (2007). Selective Attention to Emotional Faces Following Recovery From Depression. *Journal of Abnormal Psychology*, 116(1), 80-85.

- Joormann, J., & Gotlib, I.H. (2009). Emotion Regulation in Depression: Relation to cognitive inhibition. *Cognition and Emotion*, *24*(2), 281-298.
- Keller, J. (2009) Motivation and Emotion. In Passer, M., Smith, R., Holt, N., Bremner, A., Sutherland, E., & Vlieg, M (Red.). *Psychology: the Science of Mind and Behaviour*. (p. 474-526) New York: McGraw-Hill higher education.
- Kennedy, N., & Paykel, E.S. (2004). Residual Symptoms at Remission from Depression: Impact on long-term outcome. *Journal of Affective Disorders*, *80*(2), 135-144.
- Kessler, R.B., Berglund, P., Demler, O., Jin, R., Korte, D., Merikangas, K.R., Rush, J., Walters, E.E., & Wang, P.S. (2003). The Epidemiology of Major Depressive Disorder. *The Journal of the American Medical Association*, *289*(23), 3095-3105.
- Kruijt, A.W., Putman, P., & Van der Does, W. (2013). The Effects of a Visual Search Attentional Bias Modification Paradigm on Attentional bias in Dysphoric Individuals. *Journal of Behavior Therapy and Experimental Psychiatry*, *44*(2), 248-254.
- Lang, P.B., Bradley, M.M., & Cuthbert, B.N. (1997). *International Affective Picture System (IAPS): technical and affective ratings*. Gainsville, Florida: NIMH center for the study of emotion and attention, University of Florida.
- MacLeod, C.M. (1991). Half a Century of Research on the Stroop Effect: An integrative review. *Psychological Bulletin*, *109*(2), 163-203.
- MacLeod, C., & Bridle, R. (2009). The Reduction of Anxiety Vulnerability through the Modification of Attentional Bias: A real-world study using home-based cognitive bias modification procedure. *Journal of Abnormal Psychology*, *118*(1), 65-75.
- MacLeod, C., Koster, E.H.W., & Fox, E. (2009). Whither Cognitive Bias Modification Research? Commentary on the special Section Articles. *Journal of Abnormal Psychology*, *118*(1), 89-99.
- MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional Bias in Emotional Disorders. *Journal of Abnormal Psychology*, *95*(1), 15-20.
- 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), 107-123.
- Mathews, A., & MacLeod, C. (2002). Induced Processing Biases have Causal Effects on Anxiety. *Cognition and Emotion*, *16*(3), 331-354.
- Mathews, A., Ridgeway, V., & Williamson, D.A. (1996). Evidence for Attention to Threatening Stimuli in Depression. *Behavioural Research and Therapy*, *34*(9), 695-705.
- Mehrabi, A., Mohammadkhani, P., Dolatshahi, B., Pourshahbaz, A., & Mohammadi, A. (2014). Emotion Regulation in Depression: An integrative review. *PCP*, *2*(3), 181-94.
- Mogg, K., Bradley, B.P., Williams, R., & Mathews, A. (1993). Subliminal Processing of Emotional Information in Anxiety and Depression. *Journal of Abnormal Psychology*, *102*(2), 304-311.
- Nisbett, R.E., & Wilson, T.D. (1977). Telling More Than We Can Know: Verbal reports on mental processes. *Psychological Review*, *84*(3), 231-259
- Nolen-Hoeksema, S., & Aldao, A. (2011). Gender and Age Differences in Emotion Regulation Strategies and their Relationship to Depressive Symptoms. *Personality and Individual Differences*, *51*(6), 704-708.
- Paykel, E.S. (2008). Partial Remission, Residual Symptoms and Relapse in Depression. *Dialogues in Clinical Neuroscience*, *10*(4), 431-437.
- Paykel, E.S., Ramana, R., Cooper, Z., Hayhurst, H., Kerr, J., & Barocka, A. (1995). Residual Symptoms After Partial Remission: An important outcome in depression. *Psychological Medicine*, *25*(6), 1171-1180.
- 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.
- Phan, K L., Fitzgerald, D.A., Nathan, P.J., Moore, G.J., Uhde, T.W., & Tancer, M.E. (2005). Neural Substrates for Voluntary Suppression of Negative Affect: A functional magnetic resonance imaging study. *Biological Psychiatry*, *57*(3), 210-219.

- 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(20), 22-33.
- Siemer, M., & Reisenzein, R. (2007). Appraisals and Emotions: Can you have one without the other? *Emotion*, 7(1), 26-29.
- Steer, R.A., Beck, A.T., Riskind, J.H. & Brown, G. (1987). Relationships Between the Beck Depression Inventory and the Hamilton Psychiatric Rating Scale for Depression in Depressed Outpatients. *Journal of Psychopathology and Behavioral Assessment*, 9(3), 327-339.
- UNESCO. (2012). *International standard classification of education ISCED 2011*. Montreal, Quebec: UNESCO institute for statistics.
- Wadlinger, H.A., & Isaacowitz, D.M. (2011). Fixing our Focus: Training attention to regulate emotion. *Personality and Social Psychology Review*, 15(1), 72-102.
- Wadlinger, H.A., & Isaacowitz, D.M. (2008). Looking Happy: The experimental manipulation of a positive visual attention bias. *Emotion*, 8(1), 121-126.
- Wessa M., Kanske P., Neumeister P., Bode K., Heissler J., & Schönfelder S. (2010). EmoPics: Subjektive und psychophysiologische evaluation neuen bildmaterials für die klinisch-biopsychologische Forschung. *Zeitschrift für Klinische Psychologie und Psychotherapie*, 1(11), 77.
- Yang, W., Ding, Z., Dai, T., Peng, F., & Zhang, J. (2015) Attention Bias Modification Training in Individuals with Depressive Symptoms: A randomized controlled trial. *Journal of Behavior Therapy and Experimental Psychiatry*, 49(part A), 101-111.
- Yovel, I., & Bigman, N. (2012). *Acceptance and Commitment to Chosen Values in Cognitive Behavioral Therapy*. In Mikulincer, M., & Shaver, P.R. (Eds.). *The Social Psychology of Meaning, Morality and Choice*. Washington, DC: American Psychological Association.