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Specific affect regulation impairments
in major depression

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Dipl.-Psych. Timo Brockmeyer

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Dekan: Prof. Dr. Klaus Fiedler

Berater: Prof. Dr. Matthias Backenstraß, Prof. Dr. Martin Grosse Holtforth

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List of publications for dissertation

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“Lots of food pellets and no foot shocks - this is the goal of life in this giant Skinner box world.” —Randall Larsen (2000, p. 139)

1 Introduction

Major depressive disorder (MDD) is one of the most debilitating mental disorders and is primarily characterized by sustained negative affect and by the absence of positive affect (APA, 1994). Several researchers have proposed that individuals suffering from this disorder may exhibit maladaptive cognitive and behavioural affect regulation strategies (Campbell-Sills & Barlow, 2007; Gross & Muñoz, 1995; Joormann & D'Avanzato, 2010; Kring & Werner, 2004). Only recently, the field has begun to investigate cognitive and behavioural processes and mechanisms underlying deficient affect regulation in detail and to link them among each other (Joormann & D'Avanzato, 2010).

One particular area of research is concerned with the question whether certain emotion regulation difficulties are more strongly associated with certain mental disorders than with others (Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema, & Schweizer, 2010). However, previous research has failed to provide detailed insight into this potential disorder-specificity of emotion regulation difficulties. Therefore, the first study of this dissertation investigates potential differences between patients with MDD and patients with an eating disorder in a broad spectrum of emotion regulation difficulties.

Besides this issue of disorder-specificity, research also deals with the question whether certain affect regulation impairments are not only associated with current depressive symptoms but also with depression vulnerability (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008; Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010; Joormann, Cooney, Henry, & Gotlib, 2012). Specifically, the interplay of certain meta-mood beliefs such as a general motive of emotion avoidance and low confidence in mood regulation abilities may play an important role in the vulnerability to depression

(Campbell-Sills, Barlow, Brown, & Hofmann, 2006b; Catanzaro & Mearns, 1990). However, there is a lack of studies specifically testing whether these two constructs are associated with a higher risk for clinical depression. Therefore, the second study of this dissertation aims at testing whether vulnerable (formerly-depressed) and non-vulnerable (never-depressed) individuals differ in emotion avoidance and negative mood regulation expectancies and at testing how these two variables are associated with each other.

To gain more insight into maladaptive cognitive-affective processes associated with depression vulnerability, the third study of this dissertation examined two mechanisms of mood regulation potentially underlying another empirically supported risk factor for depression, i.e. cognitive reactivity (Scher, Ingram, & Segal, 2005). The study tests whether vulnerable and non-vulnerable individuals differ in their abilities to engage in mood-incongruent information processing and to disengage from negative material, and how these two processes of mood regulation may be linked to cognitive reactivity. Thus, the present dissertation focuses on several specific impairments of affect regulation in depression and depression vulnerability. This research may provide further insight into the aetiology of depression and depression vulnerability and may help to refine existing treatment approaches with regard to tailoring interventions that foster adaptive emotional processing.

The following section will provide a brief overview of the theoretical and empirical background of affect regulation impairments in depression. At the end of this section, the general research questions for the present studies will be outlined. Following the description of methods used in the three related studies, an overview of the specific research questions and the obtained results of each study will be given. The findings will then be summarised and discussed with respect to limitations, future research

directions, and clinical implications. Finally, each of the three studies that are part of this dissertation will be presented in detail as original articles.

2 Theoretical and empirical background

In this section, a brief introduction to MDD will be given first. Afterwards, theoretical models and previous research on the following topics will be briefly reviewed: cognitive reactivity and depression vulnerability, affect regulation, specific affect regulation deficits in depression including: meta-mood beliefs, deficits concerning the experience and differentiation of emotions, and deficits concerning the attenuation and modulation of moods and emotions. Finally, the basic research questions for this dissertation will be outlined.

2.1 Major depressive disorder

Major depressive disorder (MDD) presents as one of the core health challenges of the 21st century (Wittchen et al., 2011). It is mainly characterized by sustained negative mood as well as by anhedonia, that is diminished interest or pleasure in activities or a lack of positive affect (APA, 1994). Further symptoms are appetite and sleep disturbances, psychomotor agitation or retardation, fatigue or loss of energy, excessive feelings of worthlessness and/or guilt, diminished abilities to think and to concentrate as well as recurrent thoughts of death. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994), an MDD is to be diagnosed if five or more of these symptoms (including one of the two core symptoms: depressed mood and anhedonia) have been present during the same 2-week period and have been causing clinically significant distress or impairments in social, occupational, or other important areas of functioning. Furthermore, the symptoms must not be due to the direct physiological effects of a substance or a general medical condition.

In Europe, the 12-months prevalence for MDD is as high as 6.9 % (Wittchen, et al., 2011). This means, that approximately 30.3 million people in Europe have been suffering from an MDD in 2010. Moreover, MDD represents the most important contributor to burden of disease in Europe (Wittchen, et al., 2011). The lifetime prevalence for MDD is as high as 16.6 % (Kessler et al., 2005). The median age of onset is 25 to 32 years (Kessler, et al., 2005). A depressive episode has a mean duration of three to six months (Eaton et al., 2008; Hasin, Goodwin, Stinson, & Grant, 2005; Posternak et al., 2006). Ten to 15 % of patients experience an unremitted and chronic course of the disorder, sometimes over a period of 20 years (Boland & Keller, 2009; Eaton, et al., 2008). Subjects who experienced one episode, report four to five further episodes at average (Hasin, et al., 2005) and the risk of recurrence increases by approximately 16 % with each successive recurrence (Solomon et al., 2000). Correspondingly, the National Institute of Mental Health (NIMH) Collaborative Depression Study reports an initial recurrence rate of between 25 % and 40 % after 2 years. This rate however increases over time, up to 60 % recurrence after 5 years, 75 % after 10 years and 85 % after 15 years (Keller & Boland, 1998). In addition, many patients do only experience incomplete remission between episodes (Judd et al., 1998).

Depression is moreover associated with high lifetime comorbidity rates, 59 % of patients with an MDD also suffer from an anxiety disorder in their lives, 30 % from an impulse control disorder, 24 % from a substance use disorder (Kessler, 2003), and 31 % from a personality disorder (Hasin, et al., 2005). Furthermore, MDD is associated with higher rates of medical illness (Freedland & Carney, 2009; Katon, 1996) as well as with a 20 times increased suicide risk (Brådvik, Mattisson, Bogren, & Nettelbladt, 2008; Harris & Barraclough, 1997; Holma et al., 2010). MDD also has serious social consequences such as increased divorce rates (Kessler, Walters, & Forthofer, 1998), increased risk for

developing mental disorders in the offspring (Weissman et al., 2006), and increased distress for family members (Coyne et al., 1987). In addition, MDD is associated with profound economic consequences. In 2004, the annual cost of depression in Europe was estimated at €118 billion (Sobocki, Jönsson, Angst, & Rehnberg, 2006).

Fortunately, effective treatments are available for MDD. Psychotherapy as well as medication are viable treatments for depression (Cuijpers, van Straten, Andersson, & van Oppen, 2008; De Maat, Dekker, Schoevers, & De Jonghe, 2006; Ekers, Richards, & Gilbody, 2008; Imel, Malterer, McKay, & Wampold, 2008) and, to a lesser degree, for chronic depression (Cuijpers et al., 2010). Several independent meta-analyses suggest that psychotherapy is associated with more sustainable changes than pharmacotherapy (De Maat, et al., 2006; Imel, et al., 2008; Spielmans, Berman, & Usitalo, 2011). However, long-term effects of both psychotherapy and medication are far from satisfying (Hollon et al., 2005; Keller & Boland, 1998; Westen & Morrison, 2001). Thus, further research on aetiological and maintenance factors in depression is needed that may inform and improve existing treatments.

2.2 Cognitive reactivity and depression vulnerability

According to cognitive models of depression, cognitive schemas which appear as negatively toned representations of the self, the world, and the future play a central role in the development, maintenance, and recurrence of depression (Abramson, Metalsky, & Alloy, 1989; Beck, 1967; Kovacs & Beck, 1978). Life events have been found to elicit depressive symptoms by activating those cognitive schemas. For instance, depressive symptoms are more likely to emerge if individuals interpret negative events in terms of their own inadequacies (Ingram, Miranda, & Segal, 1998).

Beck's cognitive model of depression suggests that such negatively toned cognitive schemas, evolved by interactions in early development, lay dormant in the non-depressed state (Beck, 1967; Kovacs & Beck, 1978). Correspondingly, several studies revealed negative cognitions to be rather a correlate than a vulnerability factor of depressive symptoms (Gotlib & Cane, 1987; Hollon, Kendall, & Lumry, 1986; Silverman, Silverman, & Eardley, 1984; Wilkinson & Blackburn, 1981). According to Beck's model, these cognitive schemas can become activated by negative life events that are similar to the events that led to their development. However, Segal and Ingram (1994) refined this early model by describing two specific ways of schema activation: First, a schema can become activated directly by a perceived stimulus that corresponds to the schema content. Second, a schema can become activated indirectly by the activation of an associated schema. This latter mechanism refers to the idea that once a schema is activated, this activation spreads to associated schemas (Bower, 1981; Ingram, 1984). Moreover, Teasdale (1988) pointed out that negative mood itself can become a stressor that interacts with latent cognitive schemas to cause depressed mood. According to these models, individuals with and without latent depressogenic schemas should show comparable levels of negative thinking in the absence of schema activation. In contrast, interindividual differences in negative thinking should appear when the cognitive schemas are activated, for instance by sad mood. Such a change of depressive thinking in response to sad mood is called *cognitive reactivity* (Scher, et al., 2005; Segal, Gemar, & Williams, 1999).

Indeed, there is substantial evidence supporting Teasdale's (1988) hypothesis, that an increased cognitive reactivity is associated with a higher risk for depression. Several studies reported that formerly-depressed individuals responded with more negative thinking to a sad mood provocation as well as to naturally occurring sad mood

compared to never-depressed individuals (see Scher, et al., 2005, for a review). Moreover, two studies found that cognitive reactivity predicted depressive relapse prospectively (Segal, et al., 1999; Segal et al., 2006).

However, several studies reported an unexpected *reversed* cognitive reactivity in low-risk groups, i.e. a *reduction* of depressive thinking after a negative mood induction (Beevers, Scott, McGeary, & McGeary, 2009; Gemar, Segal, Sagrati, & Kennedy, 2001; Miranda, Persons, & Byers, 1990; Segal, et al., 2006). This finding contradicts the idea that negative mood merely activates an otherwise inactive depressive schema: According to the activation hypothesis, negative mood should either increase depressive thinking or leave it unchanged. A decrease in depressive thinking raises the question about further micro-processes underlying cognitive reactivity.

There is evidence from studies in clinical psychology that speak in favour of the hypothesis that affect regulation processes are linked to cognitive reactivity. Several studies revealed that increased emotion regulation difficulties are associated with depression as well as with an increased risk for depression (Ehring, et al., 2008; Ehring, et al., 2010; Pfeiffer, Kaemmerer, Mearns, Catanzaro, & Backenstrass, 2011). As an indicator of physiological emotion regulation capacity, heart rate variability was recently found to be associated with cognitive reactivity (Beevers, Ellis, & Reid, 2010).

2.3 Affect regulation

Sustained negative affect and reduced positive affect are the predominant features of MDD (APA, 1994). Several authors have suggested that these affective disturbances may result from impaired affect regulation abilities (Campbell-Sills & Barlow, 2007; Gross & Muñoz, 1995; Joormann & D'Avanzato, 2010; Kring & Werner, 2004). To gain a better understanding of such potential deficits it may be helpful to first clarify what *affect*

means in this context. Several authors have proposed that affect represents a superordinate category for valenced states such as emotions and mood (Rosenberg, 1998; Scherer, 2009). Whereas emotions are typically characterized by a rapid onset and a relatively short duration with specific internal or external objects of focus, moods are of longer duration and may not be focused on particular objects or situations (Ekman, 1992; Frijda, 1986). Larsen pointed out to the differences between these both affective states very vividly: "Moods nag at us, emotions scream at us." (Larsen, 2000, p. 130). However, they both share some primary features as both are felt or sensed as well as expressed to some degree by the individual, and both are associated with certain physiological responses (Larsen, 2000). Actually, most studies in this field did not set a great store by differentiating emotion regulation from mood regulation. Rather, these two constructs have been used interchangeably by many authors. This may in part be due to methodological reasons. Although mood and emotion seem to differ conceptually (at least to some degree), it may be difficult to separate them empirically. So far there is no consent regarding such questions as from which duration on an emotion turns into a mood. Similarly, it may not always be obvious whether an affective state refers to a definite object, particularly in cases when affective responses are induced by internal objects. Future research is needed to differentiate mood and emotion as well as mood regulation and emotion regulation more clearly. Hence, in the following sections the constructs of mood and emotion will not be differentiated strictly.

Affect is considered to be central to motivation and goal-directed behaviour (McClelland, 1985; Murray, 1938), and several authors have suggested that emotions may even constitute the primary motivational system of human beings (Izard, 1993; Leeper, 1948; Tomkins, 1962). In this sense, human thoughts, plans, and actions may be intended to either induce positive emotions or to avoid negative emotions, either in the

short or in the long term (Larsen, 2000; Nesse, 1990). Emotions are considered to dispose individuals to either approach or to avoid certain situations, objects, ideas, and actions (Levenson, 1999; Tooby & Cosmides, 1990). They may signalize the individual whether important goals are met or frustrated (Schwarz & Clore, 1983). From a functionalist perspective, emotions can be viewed as originally adaptive responses to various challenges and opportunities individuals are faced with in their daily lives (Levenson, 1994). Particularly negative affective responses are assumed to serve as fast feedback-signals indicating present or imminent harm, motivating the individual to take action (Averill et al., 1994; Nelissen & Zeelenberg, 2007; Nesse, 1990). Moreover, since intense negative emotions are usually experienced as aversive, they may themselves become targets of avoidance, and may in this way contribute to dreading experiences like a fear of anxiety (Chambless & Gracely, 1989), a fear of sadness (Taylor & Rachman, 1991), or even more general, a fear of emotion (Williams, Chambless, & Ahrens, 1997). Without a doubt, emotions likewise serve important interpersonal functions as they have the potential to coordinate social interactions by providing others with information about the interacting individual's emotions and intentions, for example. Furthermore, expressed emotions may evoke complementary emotions and behaviours in others that in turn may be of benefit for the interacting individual (Keltner & Kring, 1998).

Against this background, it becomes apparent how central affective responses are in daily functioning. Accordingly, the handling of these affective responses - which may include their recognition, differentiation, evaluation, and modulation - represents a fundamental day-to-day challenge for the individual. One of the most influential approaches to emotion regulation has been defining it as “[...] the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions” (Gross, 1998a, p. 275). The corresponding

process model proposed by Gross (1998a) distinguishes several emotion regulation strategies by where they typically occur on a temporal dimension. Antecedent-focused strategies occur early in time following exposure to an affect-eliciting external stimulus but before a fully developed affective response. Examples for this kind of strategies are situation selection (e.g., avoiding certain people, situations, or activities), situation modification (e.g., preparing oneself in advance of a challenging situation, convincing someone to act in a different way), attentional deployment (e.g., distraction, concentration, rumination), and cognitive change (i.e., reinterpreting the meaning of a stimulus). In contrast, response-focused strategies appear later in time when an affective response has already been generated. Gross (1998a) typically names expressive suppression as such a response modulation strategy which refers to the inhibition of ongoing emotion-expressive behaviour.

However, several authors have criticized this view in that it seems synonymous with emotion modulation, i.e. the mere (up- and) down-regulation of emotional responses (Salters-Pedneault, Steenkamp, & Litz, 2010). These authors rather suggest to broaden the construct since adaptive emotion regulation can be described as a “meta-emotional phenomenon” (Salters-Pedneault, et al., 2010) that involves a range of cognitive and behavioural processes not only concerning the attenuation and modulation of emotions but also concerning the experience, differentiation, and acceptance of emotions (Cole, Michel, & Teti, 1994; Gratz & Roemer, 2004; Gratz & Tull, 2010; Paivio & Greenberg, 1998; Salters-Pedneault, et al., 2010; Thompson, 1994; Thompson & Calkins, 1996). Adaptive emotion regulation may therefore include: (a) the awareness, understanding and acceptance of one’s own emotions as well as (b) capabilities to stay in control of impulsive behaviours, to keep acting in agreement with personal goals when experiencing aversive emotions, and abilities to flexibly apply situationally appropriate

emotion regulation strategies to modulate and attenuate emotional responses as desired in order to meet individual goals. Similarly, Thompson (1994) has described emotion regulation as consisting of different processes that are responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensity and temporal features, to accomplish one's goals (Thompson, 1994, pp. 27-28). Several models of emotion regulation or mood regulation can be found in the literature (Gross, 1998a, 1998b; Koole, 2009; Parkinson & Totterdell, 1999; Thayer, Newman, & McClain, 1994). However, of all these models the one proposed by Larsen (2000) seems the most relevant for the issues of this dissertation project. According to this control theory model of mood regulation (based on Carver and Scheier's control theory, 1982), individuals have some desired subjective states to which they regularly compare their current states. When an individual recognizes a discrepancy between the current and the desired affective state, he or she will engage in regulatory mechanisms, be it cognitive or behavioural strategies, to reduce this discrepancy. These regulatory mechanisms may seek to either induce changes in the environment (e.g., through active problem-solving) or within the individual (e.g., via reappraisal). Larsen furthermore suggested several variables in which individuals may differ referring to this model of mood regulation: (1) the extent to which they generally attend to and encode affective information in the environment, (2) the choice, frequency, and success of cognitive and behavioural affect regulation strategies, (3) their trait affective reactivity, (4) the extent to which they attend to and percept internal cues of their current affective states, (5) the extent to which they recognize discrepancies between current and desired affective states, (6) their affective set point, i.e. the level of affect they generally prefer. As will become clear in the next sections, this dissertation project is concerned with many of these variables (for point 1 see section 2.4.1, as well as Study 2; for point 2 see sections

2.4.1, 2.4.3, as well as Studies 1, 2, and 3; for point 4 and 5 see section 2.4.2, as well as Study 1; for point 6 see section 2.4.1, as well as Study 2).

Individual abilities to regulate affective responses may develop from early childhood on and through adulthood (Thompson & Goodman, 2010). Early in life, affect regulation will typically be managed by others. The child's parents may be soothing their child's distress by providing assurance, assistance, and need satisfaction. However, a transition from other-initiated affect regulation to self-initiated affect regulation will occur over time. As the infant grows up, his or her reliance on self-regulation is growing. The child develops increasing breadth, sophistication, and flexibility in the use of several affect regulation strategies. There is a whole panoply of factors influencing the development of a child's affect regulation capacities. For example, temperament may have an impact on emotion regulation due to varying thresholds for the arousal of negative emotions (Calkins & Hill, 2007), through the degree of effortful control (Kochanska, Murray, & Harlan, 2000), and through interactions with caregiver characteristics (Nachmias, Gunnar, Mangelsdorf, & Parritz, 1996). The degree to which family members encourage a child to describe his or her feelings verbally may help developing language abilities related to emotion regulation. Similarly, parental coaching including such strategies as cognitive reappraisal ("Don't worry, it's just a game!"), problem-focused coping ("What helped us last time when we faced this problem?"), and attention-shifting or distraction ("Look how beautiful this butterfly is!") contributes to the child's developing beliefs in the manageability of emotions. For example, it has repeatedly been demonstrated that children develop more constructive emotion regulation capacities when their parents respond with acceptance and support to their negative emotions (Denham, Bassett, & Wyatt, 2007; Eisenberg, 2002; Eisenberg, Spinrad, & Eggum, 2010). In addition, the way parents themselves deal with their own emotions may provide the child with important

learning models (Thompson, Laible, & Ontai, 2003). On the downside, if parents are permanently disapproving, disparaging, or denouncing their child's emotional responses this will most likely have a detrimental impact on the child's developing abilities of emotion regulation. As an extreme example, child maltreatment is associated with particularly broad and severe impairments in affect regulation (Alink, Cicchetti, Kim, & Rogosch, 2009; Burns, Fischer, Jackson, & Harding, 2012; Cicchetti & Toth, 1995; Kim & Cicchetti, 2010; Maughan & Cicchetti, 2002). Recent studies using sophisticated methodologies, revealed that, for example, physically abused children have more difficulties disengaging from perceived anger cues in a selective attention paradigm (e.g., Pollak & Tolley-Schell, 2003). However, not only child maltreatment but also more subtle forms of adverse parental behaviour including expressive inhibition and negative control are linked to children's emotion regulation deficits (Feng, Shaw, & Moilanen, 2011; Jaffe, Gullone, & Hughes, 2010; Ramsden & Hubbard, 2002). An example for how subtle various factors may work together in the development of a child's emotion regulation skills is provided by a study of Thompson and Goodman (2010). The authors demonstrated that maternal beliefs about the importance of attending to emotional experience and the quality of the mother-child relationship predicted mother-child concordance in emotional experience (Thompson & Goodman, 2010).

Likewise, neurobiological factors play an important role in the development of emotion regulation capacities. The maturation of executive functions including strategic planning, error detection and correction, inhibitory control, as well as the maturation and interaction of neurobiological systems including the prefrontal cortex, amygdala, hypothalamus, and anterior cingulate cortex (Ochsner & Gross, 2007) may enable adolescents to enlist certain emotion regulation strategies (Zelazo & Cunningham, 2007). In addition, cultural as well as social contextual factors may shape the

development of an individual's emotion regulation habits (Butler, Lee, & Gross, 2007; Keller & Otto, 2009; Matsumoto, Yoo, & Nakagawa, 2008; Miyamoto & Ma, 2011).

Deficits in emotion regulation are strongly associated with child and adult psychopathology (Aldao, et al., 2010; Cole & Deater-Deckard, 2009; Southam-Gerow & Kendall, 2002). The majority of mental disorders according to the DSM-IV is more or less characterized by difficulties in emotion regulation (Kring & Werner, 2004). Thus, deficits in emotion regulation have been discussed as transdiagnostic factors underlying various forms of psychopathology (Gross & Muñoz, 1995; Kring & Sloan, 2010; Moses & Barlow, 2006; Taylor, Bagby, & Parker, 1997). In other words, it has been proposed “[...] that emotion regulatory difficulties lie at the heart of many types of psychopathology and may be a key to their treatment.” (Werner & Gross, 2010, p. 14). In line with these theoretical models, previous empirical research observed impaired emotion regulation in several mental disorders such as depression (Backenstrass et al., 2010; Campbell-Sills, et al., 2006b; Liverant, Brown, Barlow, & Roemer, 2008), anxiety disorders (Decker, Turk, Hess, & Murray, 2008; Mennin, Heimberg, Turk, & Fresco, 2005; Roemer et al., 2009), and eating disorders (Brockmeyer et al., 2012a; Harrison, Sullivan, Tchanturia, & Treasure, 2009; Huennemeyer et al., 2012).

2.4 Specific affect regulation deficits in depression

There is increasing evidence for the assumption that MDD is associated with a broad range of difficulties in dealing with negative affect (see Joormann & D'Avanzato, 2010, for a review). However, affect regulation appears as a very broad concept that comprises a multitude of strategies, processes, and mechanisms (Bloch, Moran, & Kring, 2010; Gross & Thompson, 2007; Kring & Werner, 2004). To gain a better understanding of the development, maintenance, and recurrence of depressive episodes it appears necessary to specify which particular facets of affect regulation are involved in depression and

depression vulnerability (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010). Some effort has been made by researchers to point in this direction. For example, Aldao, Nolen-Hoeksema, and Schweizer (2010) conducted a meta-analysis on emotion regulation strategies across different forms of psychopathology. The authors found that emotion regulation strategies that are typically considered as rather maladaptive (e.g., avoidance, suppression, rumination) are generally more closely associated with depression and anxiety disorder symptoms than with eating and substance use disorder symptoms. However, this meta-analysis suffered from several limitations: First, it was mostly based on studies that merely used (clinical or non-clinical) samples with symptoms of one specific disorder each, rather than comparing samples of different mental disorders within the same study. Second, these studies did not control for potential effects of co-morbidity. Third, these studies investigated a range of different emotion regulation strategies using very heterogeneous instruments. To overcome these limitations, Aldao and Nolen-Hoeksema (2010) examined various emotion regulation strategies simultaneously in one comparatively large sample. In this second study, the authors found that typically maladaptive strategies (i.e., rumination, suppression) were associated more strongly with symptoms of depression, anxiety, and eating disorders than typically adaptive strategies (i.e., reappraisal, problem-solving). However, this study used a student sample rather than a clinical or a community sample. Furthermore, the authors investigated a set of variables which may be best regarded as antecedents of and responses to affective states (Gross, 1999; Gross & John, 2003) rather than as inherent components of the emotion regulation process itself in the sense of experiencing, differentiating, attenuating, and modulating emotions (Gratz & Roemer, 2004). In the following, research will be reviewed that sheds more light on such more inherent components of emotion regulation and relevant meta-emotional concepts along

with their associations with depression and depression vulnerability. These constructs are: (1) meta-mood beliefs in the form of generalized negative mood regulation (NMR) expectancies and general emotion avoidance, (2) impairments concerning the experience and differentiation of emotions, including lack of emotional awareness and lack of emotional acceptance, (3) impairments concerning the modulation and attenuation of moods and emotions, including mood-incongruent information processing and disengagement from negative information.

2.4.1 Meta-mood beliefs and depression: Expectancies and attitudes towards emotional experience

The ability to think consciously about oneself in a deliberate and complex fashion is a unique psychological aptitude that distinguishes human-beings from other species (Baumeister, 1998; Leary & Buttermore, 2003). One aspect of self-awareness and self-reflection comprises the monitoring and evaluation of one's own affective states (Leary, 2003; Scheier & Carver, 1983). People are not just experiencing their emotions, but are also thinking about them (Mayer & Stevens, 1994; Solomon, 2004). As a result of previous experiences, temperament, and multiple other factors people may differ in their global attitudes towards their emotions. Whereas one individual may cherish emotional experience and be open to them, another individual may generally dislike and refuse emotional experience (Cole, et al., 1994; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Individuals who tend to habitually welcome emotional experience may rather tend to consider emotions as important, beneficial, and promoting. However, individuals with a habitual belief that emotional experience is something bad or aversive may regard emotions as overwhelming, menacing, or embarrassing. Apparently, an individual's overall attitude towards emotional experience is closely intertwined with his or her general motivation to rather approach or avoid emotional experience (Maio &

Esses, 2001). An individual's general motive to avoid emotional experience may in turn be closely tied to his or her expectancies about how well he or she will cope with negative affect (Catanzaro & Mearns, 1990). This chapter aims at illuminating these two intertwined meta-emotional constructs and their relation to depression.

Negative mood regulation expectancies and depression

Already at the age of 6 months, children begin to calm down when they expect their mother to come and when they hear her footsteps (Gekoski, Rovee-Collier, & Carulli-Rabinowitz, 1983; Lamb & Malkin, 1986). This intriguing finding from developmental research reveals how early certain expectations can have an impact on affect regulation. The infant's anticipation that his or her mother will do something to attenuate his or her negative affect is a formidable example for a type of beliefs that has been labelled *negative mood regulation (NMR) expectancies* (Catanzaro & Mearns, 1990). NMR expectancies are simply defined as a cross-situational "expectancy that some behavior or cognition will alleviate a negative mood state" (Catanzaro & Mearns, 1990, p. 546). Related to Lazarus' concept of secondary appraisal (Lazarus, 1966; Lazarus & Folkman, 1984), NMR expectancies involve two subtypes of expectancies: (a) outcome expectancies (i.e., beliefs about the consequences of particular behaviour), and (b) self-efficacy beliefs (i.e., beliefs about one's ability to apply required behaviour; Bandura, 1977). Likewise, NMR expectancies share some aspects of social learning theory (Rotter, 1966) as they are examples for an individual's perception of the extent to which a particular outcome is under his or her control. Social learning theory further supposes that behaviour is predicted by two evaluations: (a) by the expectancy that this behaviour will produce a particular outcome, and (b) by the value of this outcome (Ajzen & Fishbein, 1980; Rotter, 1954). Consequently, the extent to which an individual will engage in certain mood repair strategies is assumed to depend on (a) his or her beliefs

about the efficacy of these strategies, and (b) on the desirability of the outcome (i.e., alleviated mood). Furthermore, response expectancy theory (Kirsch, 1985, 1990), an extension of social learning theory, posits that response expectancies are self-confirming in that they trigger non-volitional reactions that produce the expected outcome. Such self-confirming effects are well documented in the literature. For instance, the degree of expected pain predicted the degree of experienced pain in a cold pressor task (Baker & Kirsch, 1991). Similarly, expected levels of fear predicted levels of perceived fear and avoidance behaviour in patients with an anxiety disorder (Kirsch, 1983). Thus, strong NMR expectancies may result in successful mood repair via two pathways: (1) via the facilitation of adaptive strategies through the belief that these strategies will work, and (2) in a self-confirming manner as a direct result of these optimistic beliefs (Kirsch, Mearns, & Catanzaro, 1990).

These theoretical accounts are of certain relevance to clinical psychology: According to these theories, individuals with strong confidence in their ability to manage negative affect should be less vulnerable to symptoms of depression (Kirsch, 1985). In line with these hypotheses, previous research with non-clinical samples has repeatedly shown negative associations between NMR expectancies and depressive symptoms both cross-sectionally (Catanzaro, 1997; Jimenez, Niles, & Park, 2010) and prospectively (Davis, Andresen, Trosko, Massman, & Lovejoy, 2005; Kassel, Bornovalova, & Mehta, 2007). In addition, lower levels of NMR expectancies have been observed in patients with an MDD as compared to healthy controls (Backenstrass, et al., 2010; Pfeiffer, Kaemmerer, Mearns, Catanzaro, & Backenstrass, 2011). Furthermore, changes in NMR expectancies were associated with improvement in depressive symptom severity during CBT treatment and at 6 months follow-up (Backenstrass et al., 2006). These findings suggest that NMR expectancies may be a valuable construct for the understanding of depression

vulnerability. However, no study to date has tested this hypothesis in individuals at elevated risk for depression.

Emotion avoidance and depression

If an individual does not expect to be successful in a particular task, he or she will most likely avoid this task (Phares, 1972). In this spirit, NMR expectancies should be inversely associated with the avoidance of negative affect inducing situations (Catanzaro & Mearns, 1990; Kirsch, et al., 1990; Mearns, 1991). In line with these theoretical considerations, high avoidant coping behaviour was found to be associated with low NMR expectancies in a student sample (Catanzaro & Greenwood, 1994). However, this association may be explained as well in the way that continuous avoidance of negative affect impedes the development of adaptive affect regulation capabilities. Importantly, affect avoidance has been regarded rather as a common maladaptive function of particular affect regulation strategies than as a certain form of affect regulation (Boulanger, Hayes, & Pistorello, 2010).

Originally, the avoidance of negative affect represents a most adaptive mental process that provides obvious advantages for survival to the individual (Gilbert, 2001; Kenrick & Shiota, 2008; Lang & Bradley, 2008). In addition, several authors have proposed that negative affective responses may serve as fast feedback-signals indicating current or forthcoming harm, motivating to use appropriate coping strategies (Averill, et al., 1994; Nelissen & Zeelenberg, 2007; Nesse, 1990). Human physiology is therefore biased towards emerging and responding to aversive affect in service of returning to homeostasis (Salters-Pedneault, et al., 2010, p. 137). In turn, cognitions, motives, and behaviours may often be primarily directed toward avoiding negative affect (Nesse, 1990). However, since intense negative affect is often experienced as displeasing and

painful by the individual, it may itself become a target of avoidance, re-emerging in such phenomena like a fear of anxiety (Chambless & Gracely, 1989), a fear of sadness (Taylor & Rachman, 1991), or even more general, a fear of emotions (Williams, Chambless, & Ahrens, 1997).

In recent years, the phenomenon of affect avoidance has been seen as part of a broader construct labeled *experiential avoidance* (Boulanger, et al., 2010). Experiential avoidance is defined as attempts of an individual to reduce aversive internal experiences such as emotions, thoughts, memories or bodily sensations as well as the contexts that elicit them (Hayes, et al., 1996). Apart from physical threats, experiential avoidance is assumed to also protect the individual from the repetition of aversive psychosocial experiences such as separation or humiliation, thereby providing a defense of the individual's resources and needs (Carver & Scheier, 1998; Grawe, 2007; Hayes, et al., 1996). Increased experiential avoidance is assumed to be a consequence of strong and/or prolonged aversive experiences that overburden the individuals' regulatory capacities, and/or from a lack of suitable coping strategies (Grawe, 2007). However, this originally adaptive strategy may become harmful if applied excessively and inflexibly. Empirical evidence suggests that overly strong (habitual) avoidance motivation is associated with poor psychological and health outcomes (Coats, Janoff-Bulman, & Alpert, 1996; Elliot, Sheldon, & Church, 1997; Elliot, Thrash, & Murayama, 2011; Grosse Holtforth & Grawe, 2000). More specifically, a bulk of studies has repeatedly and consistently demonstrated significant associations between self-reported experiential avoidance and depressive symptoms in various samples such as students (Cribb, Moulds, & Carter, 2006; Fresco et al., 2007; Santanello & Gardner, 2007), bereaved adults (Boelen, van den Bout, & van den Hout, 2010), older adults (Andrew & Dulin, 2007; Marquez-Gonzalez, Losada, Fernandez-Fernandez, & Pachana, 2012), patients

with an anxiety or an affective disorder (Hayes et al., 2004; Roemer, Salters, Raffa, & Orsillo, 2005), patients with borderline personality disorder (Rüsch et al., 2007), patients with substance use disorders (Bond et al., 2011), patients with chronic pain (Costa & Pinto-Gouveia, 2011), and assault survivors (Gold, Marx, & Lexington, 2007).

Experiential avoidance is considered to be very similar to a lack of emotional acceptance since both constructs reflect an unwillingness to stay in contact with an unpleasant affective state (Hayes, et al., 1996; Kashdan, Barrios, Forsyth, & Steger, 2006). A lack of emotional acceptance has in turn been considered to be involved in the development and course of MDD (Campbell-Sills, et al., 2006b). Moreover, it has been hypothesized that depression may constitute a broad defensive response that prevents the individual from non-constructive action (Nesse, 2000) and, inter alia, impedes emotional reactions in favour of a dampened mood (Rottenberg, 2005). In line with these theoretical notions, emotional non-acceptance was found to be associated with higher levels of depressive symptoms after life stress (Shallcross, Troy, Boland, & Mauss, 2010). Furthermore, patients with an MDD or an anxiety disorder rated their own emotions to be less acceptable than healthy controls did after a negative mood induction (Campbell-Sills, et al., 2006b). Similarly, formerly-depressed students reported stronger emotional non-acceptance as well as stronger difficulties in emotion regulation than never-depressed students (Ehring, et al., 2008; Ehring, et al., 2010). Since individuals who have experienced previous depressive episodes are at elevated risk for further depressive episodes, such examinations of formerly-depressed subjects may provide insight into factors associated with depression vulnerability (Just, Abramson, & Alloy, 2001). However, previous studies in this field did not specifically address generalized emotion avoidance. Whereas emotional non-acceptance, although closely related to emotion avoidance, rather taps a "negative secondary emotional responses to one's

negative emotions" (Gratz & Roemer, 2004, p. 47), emotion avoidance presents as a general motivation to avoid the experience of emotions (Maio & Esses, 2001). One facet of such a general motive of emotion avoidance may for example appear in interpersonal situations in the form of high ambivalence over the expression of emotions (King & Emmons, 1990) driven by goals of protecting oneself from rejection, criticism, or humiliation by others (Keltner & Kring, 1998; Kennedy-Moore & Watson, 1999), as well as from becoming overwhelmed by unpleasant feelings (Kennedy-Moore & Watson, 1999). Such an ambivalence over emotional expression has been found to be greater in depressed than in non-depressed individuals (Brockmeyer et al., 2012b). Furthermore, it was found to be associated with decreased well-being, increased psychological distress, and stronger depressive symptoms in non-clinical samples (Katz & Campbell, 1994; King & Emmons, 1990, 1991; Trachsel, Gurtner, von Känel, & Grosse Holtforth, 2010).

Similarly, a general motive of emotion avoidance may also fuel maladaptive emotion regulation strategies such as expressive suppression of emotions (Gross & John, 2003). Expressive suppression of emotions was found to rather decrease the experience of positive than the experience of negative affect (Campbell-Sills, et al., 2006b), and was furthermore found to be frequently associated with various aversive outcomes such as lower levels of positive affect and reduced well-being (Gross & John, 2003), low self-esteem (Borton, Markowitz, & Dieterich, 2005), as well as heightened physiological arousal during stressful tasks (Campbell-Sills, et al., 2006b; Feldner, Zvolensky, Stickle, Bonn-Miller, & Leen-Feldner, 2006; Gross & Levenson, 1997).

Likewise, a general motivation to avoid emotional experience may lead to habitual thought suppression (Campbell-Sills & Barlow, 2007), another related emotion regulation strategy that has been found to be associated with adverse outcomes as well

(Wenzlaff & Wegner, 2000). For example, suppression of affect-laden thoughts was shown to be associated with increased physiological arousal (Wegner, Broome, & Blumberg, 1997), discomfort (Purdon & Clark, 2001), levels of anxiety and depression (Borton, et al., 2005; Roemer & Borkovec, 1994), and risk for depression and anxiety (see Beevers, Wenzlaff, Hayes, & Scott, 1999, as well as Wenzlaff & Wegner, 2000, for comprehensive reviews).

2.4.2 Depression and deficits concerning the experience and differentiation of emotions

As several authors have emphasized, adaptive emotion regulation does not only involve the modulation and attenuation of emotions but also the awareness towards, the clarity of and the acceptance of emotions (Cole, et al., 1994; Gratz & Roemer, 2004; Gratz & Tull, 2010; Paivio & Greenberg, 1998; Salters-Pedneault, et al., 2010; Thompson, 1994; Thompson & Calkins, 1996). Using a highly vivid analogy, Larsen (2000, p. 135-136) pointed out to the essential role which an individual's sensitivity to his or her current affective state may play in active affect regulation: Similar to a thermostat that must precisely sense the current room temperature to effectively regulate it, the extent to which an individual is able to effectively modulate his or her affective states may (at least in part) depend on his or her ability to accurately sense those affective states. If a thermostat only recognizes large temperature changes, then it would surely prompt temperature regulation only after the room has already become extremely hot or cold. This would probably result in a very unsteady room climate. Similarly, humans may be less able to reach and maintain affective homeostasis when they attend to or perceive cues of their current affective state only until these became very vociferous and urging. In line with this theoretical notion, recent findings point into the direction that emotional awareness may in fact represent a facilitator or even a prerequisite for

adaptive emotion modulation. For instance, self-reported dispositional mindfulness (which involves emotional awareness) was positively correlated with negative mood regulation expectancies which in turn were inversely related to depressive symptoms in a student sample (Jimenez, et al., 2010). Correspondingly, subjects high in emotional awareness made lesser errors in an affective information processing task when they were instructed to use expressive suppression during a negative mood induction procedure (Szczygieł, Buczny, & Bazińska, 2012). Another experimental study revealed that subjects high in self-reported emotional clarity are more successful in correcting for the influence of aggressive primes on subsequent evaluations (Wilkowski & Robinson, 2008). Applying physiological measures, Larsen (2000) found that individuals who were less accurate in perceiving their own heart rate showed stronger negative emotional reactivity after an experimental affect induction. Moreover, acceptance of negative affect may itself present as an effective emotion modulating strategy. In a recent experimental study using a non-clinical sample, instructed acceptance during a negative mood induction procedure led to similar reductions in subjective distress compared to instructed reappraisal (Wolgast, Lundh, & Viborg, 2011). These findings are further underscored by recent neuroimaging research. The mere instruction to self-reflect and to introspect for current emotions was associated with distinguishable activations in medial and ventrolateral prefrontal areas, in parietal regions and in the amygdala. Moreover, amygdala activity was found to decrease during emotion-introspection which led the authors conclude that mental processes of making an actual emotional state aware may already be capable of attenuating emotional arousal (Herwig, Kaffenberger, Jäncke, & Brühl, 2010). In another series of experimental studies (Lieberman, Inagaki, Tabibnia, & Crockett, 2011) it was found that self-reported distress was lower during affect labelling as compared to a condition where participants were

asked to just passively watch aversive affective pictures, and comparably low as compared to conditions where participants were asked to use reappraisal or distraction. Interestingly, participants still predicted that affect labelling would rather be ineffective in decreasing distress in the future, although having made the experience that it reduced distress which led the authors to conclude that affect labelling may work as a rather incidental form of affect regulation. Correspondingly, it has been proposed that acceptance may even involve a certain kind of reappraisal, not of a situation or stimulus that elicits an emotion but of the emotional response itself (Liverant, et al., 2008; Webb, Miles, & Sheeran, 2012). These theoretical models and empirical findings clearly underline the relevance of processes and mechanisms of emotional awareness, clarity, and acceptance in mood disorders. Thus, the present chapter will highlight empirical findings on the associations between these constructs and depression.

Lack of emotional awareness and clarity

Several authors suggested a subdivision of the construct of emotional awareness into two major components (Coffey, Berenbaum, & Kerns, 2003; Gohm & Clore, 2000, 2002). The first component may be labelled *attention to emotions*, i.e. the extent to which an individual attends to and values his or her own emotions. The second component refers to *emotional clarity*, i.e. the extent to which an individual is able to identify and to describe his or her own emotions (Coffey, et al., 2003; Gohm & Clore, 2000, 2002).

Research on associations between depression and the first of these two components (i.e., attention to one's own emotions) yielded heterogeneous results what may, to a great extent, result from conceptual and methodological differences between the studies. On the one hand, there is ample evidence for an association between high self-focused attention (which is considered to at least in part include attention to one's own affective states) and depression as well as depression vulnerability from research on

rumination (see Thomsen, 2006, for a review), language use (Rude, Gortner, & Pennebaker, 2004; Sloan, 2005), and self-reported self-awareness (see Fejfar & Hoyle, 2000, as well as Mor & Winquist, 2002, for reviews). On the other hand, there is also growing evidence for an association between low attention to one's own emotions and current depressive symptoms (Fisher et al., 2010; Rude & McCarthy, 2003; Vázquez et al., 2011). As already mentioned, these discrepancies may at least in part be explained by the different concepts and measures of self-attention used in the different studies. For example, studies on language use, self-awareness, and self-consciousness do not necessarily focus on attention to affective states but other aspects of the self. Furthermore, rumination may describe a specific way how an individual attends to and processes his or her own feelings (i.e., in a repetitive, abstract manner) rather than the individual's motive or tendency to sense and to acknowledge his or her own feelings. Moreover, rumination may paradoxically even display low attention to certain aspects of one's current affective state: Drawing on Borkovec and colleagues' avoidance theory of worry (Borkovec, Ray, & Stöber, 1998; Stöber & Borkovec, 2002), Watkins and Moulds (2007a) recently proposed that rumination may serve to avoid aversive imagery and detailed information of certain events or experiences as well as associated physiological arousal because it is characterized by rather abstract and less concrete thinking about those events or experiences. Indeed, there is some empirical support for this notion, as rumination has been found to be associated with self-reported experiential avoidance and self-reported cognitive-behavioural avoidance in student samples (Giorgio et al., 2010; Moulds, et al., 2007).

The second component of emotional awareness (i.e., emotional clarity) is closely related to another construct labelled *alexithymia*. Originally, alexithymia has been defined as a phenomenon involving (a) difficulties indentifying and describing one's

own feelings, (b) difficulties in distinguishing between feelings and bodily sensations of affective arousal, (c) a lack of fantasy, and (d) an externally oriented cognitive style (Sifneos, Apfel-Savitz, & Frankel, 1977). More recently, the construct of alexithymia has been mainly conceptualized as an impairment in cognitive-experiential components of emotional processing (Taylor, Bagby, & Parker, 1997). Alexithymia can be measured by the Toronto Alexithymia Scale (TAS), a short self-report measure (Bagby, Parker, & Taylor, 1994; Bagby, Taylor, & Parker, 1994). Using this measure, several studies found higher levels of alexithymia for depressed individuals than for healthy controls (Duddu, Isaac, & Chaturvedi, 2003; Loas et al., 1998). Furthermore, alexithymia was associated with levels of depression in depressed patients (Conrad, Wegener, Imbierowicz, Liedtke, & Geiser, 2009). In addition, several studies found an association between alexithymia and depressive symptoms in community samples (Honkalampi, Hintikka, Tanskanen, Lehtonen, & Viinamäki, 2000). Despite these findings, there is much debate about the nature and role of alexithymia in depression. Factor-analytical investigations yielded alexithymia as a construct separate and distinct from depression (Hintikka, Honkalampi, Lehtonen, & Viinamäki, 2001; Marchesi, Brusamonti, & Maggini, 2000; Parker, Bagby, & Taylor, 1991). Furthermore, alexithymia has been shown to be a stable, trait-like characteristic of depressed patients (Saarijärvi, Salminen, & Toikka, 2006; Salminen, Saarijärvi, Äärelä, & Tamminen, 1994). However, several other studies showed alexithymia to rather wax and wane with the occurrence of depressive symptoms (Honkalampi, Hintikka, Laukkanen, Lehtonen, & Viinamäki, 2001; Marchesi, Bertoni, Cantoni, & Maggini, 2008). Hence, the results so far suggest alexithymia to be trait- as well as state-dependent in depressed patients (Saarijärvi, et al., 2006). However, alexithymia is by no means a disorder-specific phenomenon since it has been shown to be associated with other mental disorders as well (Cochrane, Brewerton, Wilson, &

Hodges, 1993; Marchesi, Fontò, Balista, Cimmino, & Maggini, 2005; Schmidt, Jiwany, & Treasure, 1993). The finding that depressed patients and those with an anxiety disorder do not differ concerning their alexithymic characteristics (Marchesi, et al., 2000) lends further support to the conceptualization of alexithymia as a transdiagnostic deficit in emotional awareness (Taylor, et al., 1997).

Besides alexithymia, self-reported mood clarity as measured by the Trait Meta-Mood Scale (Salovey, Mayer, Goldman, Turvey, & Palfai, 1995) was found to be inversely associated with depressive symptoms (Salovey et al., 1995), as well as to predict depressive symptoms two years later in older adults, and to attenuate the effect of pain on depressive symptoms (Kennedy et al., 2010).

Lack of emotional acceptance

Another, related facet of experiencing and differentiating emotions refers to difficulties accepting negative emotions (Gratz & Roemer, 2004; Liverant, et al., 2008; Salters-Pedneault, et al., 2010; see also section 2.4.1). Several authors have considered low emotional acceptance to be very similar or even equivalent to experiential avoidance since both constructs reflect at least in part an unwillingness to stay in contact with aversive affective states (Boelen & Reijntjes, 2008; Hayes, et al., 1996; Kashdan, et al., 2006). However, other authors have suggested that these two constructs, though closely related, may not be equivalent (Amstadter, 2008; Campbell-Sills & Barlow, 2007). Rather they assumed that individuals who tend to judge their emotional responses to be aversive and unacceptable may likely engage in suppression or other forms of avoidance to circumvent these unpleasant experiences or to modulate them when they already occurred. Although diverging in concept, these two constructs may be difficult to separate empirically. As already outlined in section 2.4.1, recent empirical findings suggest that a lack of emotional acceptance may be a highly relevant factor in

the aetiology of depression. For instance, the extent of negative attitudes towards sadness was inversely correlated with the ability to accept negative mood during a mood induction task in formerly-depressed individuals (Singer & Dobson, 2009). In addition, depressed subjects high in self-reported anxiety about the experience of depressed mood were less able to down-regulate experimentally induced negative affect than their less anxious counterparts (Liverant, Brown, Barlow, & Roemer, 2008).

2.4.3 Depression and deficits concerning the modulation and attenuation of mood and emotions

There is ample evidence that emotion regulation deficits are more pronounced in patients with MDD than in controls (Joormann & Gotlib, 2010; Joormann, Siemer, & Gotlib, 2007; Liverant, et al., 2008; Pfeiffer, et al., 2011) and that these deficits are associated with depressive symptoms in the general population (Garnefski & Kraaij, 2006; Kassel, et al., 2007), with an increased risk for depression (Ehring, et al., 2008; Ehring, et al., 2010; Joormann, et al., 2012), and with slowed recovery from MDD (Arditte & Joormann, 2011; Kumar, Feldman, & Hayes, 2008). As mentioned before, emotion regulation is a broad construct that involves a range of different cognitive (e.g., reappraisal, reflection, brooding) and behavioural strategies (e.g., expressive suppression, distraction, withdrawal). Yet, cognition has been considered as “[...] a primary route through which emotions are regulated.” (Joormann & D’Avanzato, 2010, p. 914). Particularly, cognitive processes of attention and memory are considered to play a major role in affect regulation (Joormann & D’Avanzato, 2010). In terms of attention processes, the affect regulation literature mainly distinguishes between two basic strategies: those that focus attention on the negative mood and its elicitors and those that focus attention away from the negative mood and its elicitors (Rusting, 1998). This notion is in line with theoretical models of attention processing that distinguish between

shifting and maintaining attention (Allport, 1989; LaBerge, 1995). These both basic strategies are thought to involve a variety of processes and mechanisms. This chapter will focus on two specific mechanisms of memory and attention shifting in the scope of affect regulation: (1) disengagement from negative information, and (2) mood-incongruent information processing.

Impaired disengagement from negative information and depression

In the first year after birth, the maturation of an infant's neurobiological attention systems leads to increasing voluntary control over the ability of looking, involving the ability to disengage visually from affectively arousing stimuli (Posner & Rothbart, 2000; Rothbart, Sheese, Rueda, & Posner, 2011). These findings from developmental research demonstrate that disengagement from aversive stimuli is a fundamental strategy of affect regulation. Importantly, it has been proposed that depression is not associated with biases in early information processing but rather with difficulties in attention redirection (Williams, Watts, MacLeod, & Mathews, 1988). Consistent with this notion, previous research demonstrated that depression is not associated with a bias in initial orienting towards negative information (see Mathews & MacLeod, 2005, for a review) but rather with difficulties disengaging from negative information that once came into the focus of attention (see Joormann & D'Avanzato, 2010, for a review). Studies using a modified Stroop task with either subliminally or supraliminally presented depression-related, neutral, and positive words found no difference between depressed participants and healthy controls regarding reaction times to negative words (Lim & Kim, 2005; Mogg, Bradley, Williams, & Mathews, 1993). Similarly, no association evolved between biases for subliminally presented negative words and depressive symptoms in a student sample (Yovel & Mineka, 2005). Studies using a dot-probe task reported, however, that experimentally induced and naturally occurring negative mood both were associated

with increased attention towards negative words at long exposure durations but not at shorter ones (Bradley, Mogg, & Lee, 1997; Donaldson, Lam, & Mathews, 2007; Mogg, Bradley, & Williams, 1995). Correspondingly, studies using eye-tracking technology demonstrated that depressed individuals did not differ from controls in orienting towards negative stimuli but that they spent more time looking at these negative stimuli than controls (Caseras, Garner, Bradley, & Mogg, 2007; Eizenman et al., 2003). Moreover, recent work has demonstrated that this bias is not only associated with current depression but also with an elevated risk for depression to ensue. Impressively, Joormann, Talbot, and Gotlib (2007) were able to show that 9 to 14 years old girls whose mothers previously had experienced recurrent depressive episodes selectively attended to pictures of negative facial expressions whereas controls selectively attended to pictures of positive facial expressions in a dot-probe task. Similarly, this bias was found in a sample of formerly-depressed individuals (Joormann & Gotlib, 2007). In addition, as compared to never-depressed women, previously depressed women were found to be slower in directing their attention away from negative word stimuli after a negative mood induction (McCabe, Gotlib, & Martin, 2000). Taken together, these findings suggest that impaired disengagement from negative emotional material not just covaries with depressive symptoms but rather constitutes an important factor in depression vulnerability (Joormann & D'Avanzato, 2010).

Furthermore, several experimental studies demonstrated how an impaired ability to disengage from negative information may be linked to affect dysregulation and depressive symptoms. For example, a reduced ability to disengage attention was found to be associated with increased emotional reactivity following a negative mood induction procedure (Compton, Heller, Banich, Palmieri, & Miller, 2000). In addition, an attentional bias for negative words in a dot-probe task following a negative mood

induction interacted with life stress to predict future depressive symptoms in a student sample (Beevers & Carver, 2003). In another study with a student sample, depressive symptoms were inversely correlated with the ability to shift attention during a stressor condition (Ellenbogen, Schwartzman, Stewart, & Walker, 2002). The authors further reported that participants in this stressor condition were found to faster shift their attention away from negative as compared to positive or neutral words and that these shifts were associated with mood changes in response to the stressor.

An impaired ability to disengage from negative information may lay the foundation for further deficits in affect regulation (Joormann & Siemer, 2011). Potentially, this basic deficit of attentional deployment precludes depressed individuals from applying adaptive affect regulation techniques such as distraction (i.e., shifting attention away from negative material towards neutral material) or reappraisal (i.e., shifting attention away from negative material towards positive material). At first sight, these findings of an impaired disengagement from negative material in depression may be at odds with findings of marked emotional avoidance in depression (as highlighted in section 2.4.1). However, these paradoxical findings may be well understandable against the background of Gross' model of antecedent-focused and response-focused processes in emotion regulation (Gross, 1998a, 1998b), as well as models of deliberate/explicit and incidental/implicit emotion regulation (Berkman & Lieberman, 2009; Gyurak, Gross, & Etkin, 2011). Referring to Gross' process model of emotion regulation (Gross, 1998a, 1998b), depressed as well as depression-vulnerable individuals may use avoidance as an emotion regulation strategy prior to the occurrence of an affective response (e.g., in the form of situation selection and modification; cf. Brockmeyer et al., 2012c). However, after an affective response has been generated the depression-prone individual may be trapped and unable to disengage from the evoked negative material. This experience of

being helplessly exposed to painful feelings may in turn promote the individual's motive to employ antecedent-focused avoidance behaviour in future situations. Unfortunately, such an avoidance of affective experiences may then further diminish the individual's confidence in his or her affect regulation abilities through a lack of practicing and habituation to negative affect. Similarly, using models of deliberate and incidental emotion regulation as a framework, depression-prone individuals may deliberately seek to avoid any internal or external trigger of negative affect whereas their incidental affect regulation may be characterized by a lack of ability to disengage from activated negative material. In line with this theoretical notion, numerous studies using self-report measures in various samples have demonstrated a consistent link between deliberate forms of affect avoidance (e.g., harm avoidance, behavioural inhibition, avoidance coping, avoidant problem-solving, thought suppression) and current depression as well as an increased risk for depression (for a review see Brockmeyer et al., 2012d). In contrast, as outlined above there is growing evidence for a lack of spontaneously occurring (i.e., incidental) disengagement from negative material in depression.

Mood-incongruent information processing and depression

One facet of emotion regulation that has been studied in detail with MDD is reappraisal (Gross, 1998a). Reappraisal presents as a meta-cognitive strategy when an individual re-interprets or re-evaluates a certain situation in an alternative way, particularly in a benign or positive fashion (Gross, 1998a). Initially, reappraisal was conceptualized as an antecedent-focused emotion regulation strategy occurring before an affective response is fully generated (see also section 2.3). In contrast, recent studies have demonstrated that it can also present as a response-focused strategy changing an already generated affective response (Sheppes & Meiran, 2007). However, this kind of late reappraisal was found to be less effective in changing negative affect and to require

more cognitive control as compared to early reappraisal (Sheppes & Meiran, 2008). Experimental research has shown that cognitive reappraisal effectively reduces negative emotion without increasing sympathetic arousal as it is the case of other emotion regulation strategies such as expressive suppression (Gross, 1998; Gross & Levenson, 1997). Thus, cognitive reappraisal has typically been considered to be a rather adaptive strategy for managing negative affect (Gross, 1998a). According to cognitive theories, dysfunctional and biased appraisals represent one of the core pathomechanisms in depression and other mental disorders (Beck & Alford, 2009; Clark, 1986; Cooper, 2005; Doron & Kyrios, 2005; Williams, 2004). These theoretical models have gained increasing empirical support. For instance, it has been demonstrated that depressed subjects exhibit more negative reappraisal as compared to healthy controls (Newby & Moulds, 2010). In addition, self-reported negative appraisal predicted depressive symptoms at 6-months follow-up in a community sample (Newby & Moulds, 2011). Moreover, subjects' ability to use cognitive reappraisal in a behavioural challenge task moderated the association between stressful life events and depressive symptoms in a community sample (Troy, Wilhelm, Shallcross, & Mauss, 2010). An impaired ability to spontaneously apply cognitive reappraisal for regulating negative affect has furthermore been demonstrated in formerly-depressed subjects (Ehring, et al., 2010) suggesting that this deficit is not only associated with current depression but also with depression vulnerability. In a broader context, it has been shown that the habitual use of cognitive reappraisal to manage negative affect is associated with higher levels of positive affect, lower levels of negative affect, superior interpersonal functioning, and well-being (Gross & John, 2003).

Cognitive reappraisal of a given situation may represent a specific form of mood-incongruent information processing since cognitions that are incompatible with the

individual's mood in a current situation are used to modulate this mood (Rusting & DeHart, 2000). Mainly, Bower's *Associative Network Model* (Bower, 1981) has been the theoretical background of research on mood-congruency effects in information processing. According to this influential theory, emotions serve as memory units and are represented by particular nodes within a broad cognitive network consisting of related thoughts and memories. When such an emotional node becomes activated, this activation spreads to the network, and related cognitions/memories become evoked. In essence, the model proposes that a certain affective state activates information in memory that is congruent with this affective state. Thus, in a negative mood, retrieval of negative material from memory should be facilitated whereas in a positive mood, retrieval of positive material from memory should be facilitated. In line with this theoretical model, previous research has frequently found that individuals show a cognitive bias for mood-congruent information. People in a happy mood tend to retrieve positively valenced cognitions whereas people in a sad mood tend to retrieve negatively valenced cognitions (Bower, 1981; Fiedler, Nickel, Asbeck, & Pagel, 2003; Singer & Salovey, 1988).

However, there are also many studies that reported opposing results. For example, it has repeatedly been demonstrated that affective states can paradoxically enhance the retrieval of mood-incongruent material (Erber & Erber, 1994; Parrott & Sabini, 1990; Rinck, Glowalla, & Schneider, 1992). Such findings led several authors to hypothesize that in certain situations the mechanism of mood-congruent memory retrieval may be reversed in the service of mood regulation. In line with this idea, the recall of positive autobiographical memories was found to repair a negative mood state (Erber & Markunas, 2006; Erber, Wegner, & Therriault, 1996; Forgas, 1995; Forgas & Ciarrochi, 2002). Several authors have suggested that motivational and personality variables may

account for these variations (Parrott & Sabini, 1990; Singer & Salovey, 1988). Indeed, subjects with high NMR expectancies responded with more mood-incongruent (positive) cognitions to a negative mood induction than subjects with low NMR expectancies (Rusting & DeHart, 2000).

Forgas and Ciarrochi (2002) made a major contribution to the field by outlining their models of homeostatic cognitive strategies in affect regulation. Based on empirical findings, the authors argue that mood-congruent cognitions often maintain or even enhance preexisting affective states (Forgas, 2008). This process has been labeled *affect infusion* (Forgas, 1995, p. 39). However, in a most intriguing experiment, the authors found that subjects initially produced mood-congruent responses to a negative mood induction but - with a certain delay - reversed this pattern and spontaneously produced mood-incongruent responses (Forgas & Ciarrochi, 2002). Providing an explanation for this finding, Forgas, Johnson, and Ciarrochi (1998) proposed that - following this self-perpetuating process of affect infusion - when a certain threshold level of affect intensity is reached, a different process may occur that provides access to mood-incongruent cognitions in order to reverse mood effects (see also Erber & Erber, 2001; Forgas & Fiedler, 1996). Thus, the two countervailing cognitive processes of mood-congruent vs. mood-incongruent information processing may serve as a dynamic, homeostatic affect regulation system (Forgas, Ciarrochi, & Moylan, 2000). This assumption is in line with the findings of an earlier study (Sedikides, 1994): With reference to cognitive (Forgas, 1991; Isen, 1984) and motivational (Carver & Scheier, 1990; Morris & Reilly, 1987) models of affect regulation, the author of this study predicted that subjects in a negative mood will attempt to exit this aversive state through engaging in regulatory strategies such as the retrieval of more positive (self-referred) cognitions. Indeed, he found that subjects initially responded with negative

open-ended self-descriptions to a negative mood induction and then shifted to produce more positive open-ended self-descriptions. This finding led the author to suggest that with the passage of time, people become aware of mood-triggered negative cognitive biases and may engage in mood-regulating strategies.

Theoretical models propose that affect regulation presents as a continuous and (often) subconscious process in that information processing strategies are spontaneously switched in service to calibrate prevalent affective states (Forgas & Ciarrochi, 2000). There is indeed considerable empirical evidence that the first process in Forgas' and Ciarrochi's model (affect infusion via mood-congruency effects) typically occurs automatically without conscious awareness (for a review see Forgas & Ciarrochi, 2000). However, previous studies have furthermore revealed that a switch from mood-congruent to mood-incongruent information processing is more likely when people become aware of their current mood state (Berkowitz, Jaffee, Jo, & Troccoli, 2000) and when they experience particularly intense affective states (Ciarrochi & Forgas, 1999). Another important study by McFarland and Buehler (1997) provided evidence for the notion that individuals must first become aware of a negative affective state before they can engage in motivated recall of mood-incongruent memories to alleviate the aversive state. In a series of studies, subjects high in self-reported sensitization or emotional awareness exhibited more negative affect following a negative mood induction and recalled more positive memories as compared to subjects low in self-reported sensitization (i.e., repressors) or emotional acceptance. More generally, several studies have demonstrated a close relationship between emotional awareness and emotion regulation capabilities (e.g., Gohm, 2003; Swinkels & Giuliano, 1995).

There is increasing evidence now for the assumption that mood-incongruent information biases in affect regulation may be part and parcel of depression and

depression vulnerability (see Joormann & D'Avanzato, 2010, for a recent review). Previous studies showed that in contrast to non-depressed individuals, currently-depressed and formerly-depressed individuals had reduced access to positive autobiographic memories following a negative mood induction and did not experience an alleviation of the experimentally induced negative mood by retrieving positive autobiographical memories (Joormann & Siemer, 2004; Joormann, Siemer, et al., 2007). Similarly, Josephson, Singer, and Salovey (1996) observed that, following a negative mood induction, dysphoric and non-dysphoric individuals both initially reported mood-congruent (negative) autobiographical memories. However, subsequently, non-dysphoric individuals recalled more mood-incongruent (positive) memories whereas dysphoric individuals recalled further mood-congruent (negative) memories and coevally reported lower levels of mood repair (Josephson, et al., 1996). This deficit may in part be due to a generally higher accessibility of negative memories in depressed individuals. The association between depression and a preference for recalling negative instead of positive memories is very well documented in the literature (see Mathews & MacLeod, 2005).

However, the findings of a recent study suggest that processing mode (abstract/verbal vs. concrete/imagery) may be a decisive aspect regarding the question whether positive memories can alleviate a negative mood state or not (Werner-Seidler & Moulds, 2011). In this study, currently-depressed and formerly-depressed individuals were instructed to recall a positive memory in an either abstract or concrete fashion after a negative mood induction. Whereas subjects in the abstract condition experienced no subsequent alleviation of their negative mood, subjects in the concrete condition did. Accordingly, there is consistent evidence that depressed individuals tend to retrieve rather general than specific autobiographic memories (see Williams et al., 2007, for a

review). Several authors have suggested that by circumventing specific memories, individuals may avoid sensory and perceptual experiences of aversive autobiographic events and thereby protect themselves from mood disturbances (e.g., Hermans, Raes, Iberico, & Williams, 2006). In line with this theoretical notion, self-reported cognitive-behavioural avoidance, experiential avoidance, and thought suppression were found to be associated with autobiographic memory specificity as assessed by the autobiographic memory task (Williams & Broadbent, 1986) in student samples (Hauer, Wessel, & Merckelbach, 2006; Hermans, Defranc, Raes, Williams, & Eelen, 2005). Further empirical support for the notion that reduced autobiographic memory serves an emotion avoidance function comes from experimental research. Less specific memories were associated with less negative affective responses and intrusions when remembering a negative event (Raes, Hermans, de Decker, Eelen, & Williams, 2003). However, the downside of this particular avoidant coping style may become apparent in the long run. Overgeneral autobiographic memory interacted with life stress to predict depressive symptoms in students four to six weeks after initial assessment (Gibbs & Rude, 2004). Similarly, lower emotional intensity of sad autobiographic memories predicted levels of depression symptoms at one year follow-up (Rottenberg, Joormann, Brozovich, & Gotlib, 2005). In addition, reduced specificity of autobiographic memory predicted recovery from affective disorders (Dalgleish, Spinks, Yiend, & Kuyken, 2001).

2.5 General research questions of the present studies

Based on the outlined theoretical models and empirical findings, the three conjoint studies of the present dissertation aimed at testing whether (1) emotion regulation difficulties appear as transdiagnostic phenomena in that patients with MDD exceed healthy controls but not patients with another mental disorder (anorexia nervosa) regarding several difficulties in emotion regulation, (2) formerly-depressed individuals

show lower NMR expectancies and greater emotion avoidance than never-depressed controls, and whether these two constructs are inversely related to each other, (3) formerly-depressed individuals show reduced abilities to engage in mood-incongruent information processing and to disengage from negative information, and whether these two processes are inversely related with cognitive reactivity. These hypotheses are in need for further empirical investigation in order to provide a better understanding of how certain processes and mechanisms of affect dysregulation may work depression vulnerability. In section 3, the methods used in the three studies of this dissertation will be outlined with regard to participants, measures, procedures, and data analysis. Following this, in section 4 the specific research questions of each study will be presented and the corresponding results will be summarised and discussed with reference to previous literature. The studies are presented in detail as original articles (see Appendix C).

3 Methods of the current studies

In this section, the methods used in the three studies of this dissertation will be described. All three studies used a cross-sectional design. One study was conducted with clinical samples of patients with a current diagnosis of either MDD or anorexia nervosa. The other two studies were conducted with samples of individuals at high versus low risk for depression due to previous depressive episodes or no previous depressive episodes. Two of the studies were based on self-report measures. Beyond that, the third study used an experimental design with a negative mood induction procedure and an unobtrusive methodology for assessing certain processes of affect regulation, i.e. computerized quantitative text analysis.

3.1 Study 1: Specific emotion regulation impairments in major depression

3.1.1 Participants

Participants of this study were 41 women with an MDD, 39 women with anorexia nervosa (AN), and 60 female healthy controls (HC). All participants were Caucasian, and between 18 and 65 years of age. Patients were recruited consecutively from a university-based outpatient centre serving the community (MDD, 44%; AN, 41%) as well as from an inpatient unit of a university hospital. HC were recruited via advertisements in the local media and from the university campus. HC must not fulfil the criteria for any diagnosis according to the DSM-IV (APA, 1994). Subjects were excluded from the MDD and AN group if they had a current diagnosis of any substance use disorder or a lifetime diagnosis of mania or psychosis. By reason of the specific role emotion regulation deficits play in borderline personality disorder (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004), this diagnosis was determined as another exclusion criterion for the clinical groups. Participants with MDD must not have any comorbid eating disorder. Conversely, only participants without comorbid mood disorders were included in the AN group. According to DSM-IV, 32 patients had restricting subtype and seven had binge/purging subtype anorexia nervosa. Because of the small number of AN from the binge/purging subtype, the two groups were not differentiated in the present study. This is common practice (cf. Gilboa-Schechtman, Avnon, Zubery, & Jeczmiem, 2006; Harrison, Sullivan, S., Tchanturia, K., & Treasure, J., 2010; Svaldi, Griepenstroh, Tuschen-Caffier, & Ehring, 2012) and appears all the more defensible since previous studies using the same measure did not find any differences between patients with purely restrictive and patients with binge/purging symptoms regarding emotion regulation difficulties (Harrison, Sullivan, S., Tchanturia, K., & Treasure, J., 2010; Svaldi, et al., 2012). Twenty-three percent of the patients were currently under psychotropic medication (MDD, 29%, AN, 15%).

3.1.2 Material

The *Structured Clinical Interview for DSM-IV Axis I and II* (SCID; First & Gibbon, 2004; German version: Wittchen, Zaudig, & Fydrich, 1997) was used to assess symptoms of mental disorders. It was conducted by the first author and eight clinicians who were all specifically trained for SCID administration and had more than 3 years of experience using the SCID. Reliability of the SCID has been demonstrated in previous studies (Williams, Gibbon, First, & Spitzer, 1992).

To capture a comprehensive set of clinically relevant difficulties in emotion regulation based on an integrative conceptualization of emotion regulation, the authors used the 36-item *Difficulties in Emotion Regulation Scale* (DERS; Gratz & Roemer, 2004). Participants rate their difficulties in emotion regulation on a five-point Likert scale. The DERS consists of six discrete but interconnected subscales: (1) Non-acceptance of emotional responses: assesses the tendency to have negative secondary emotional responses to one's own negative emotions. (2) Difficulties engaging in goal-directed behaviour: reflects difficulties in concentrating and accomplishing tasks while experiencing negative emotions. (3) Impulse control difficulties: taps difficulties with remaining in control of behaviour when experiencing negative emotions. (4) Lack of emotional awareness: reflects difficulties associated with attending to and acknowledging one's own emotions. (5) Limited access to effective emotion regulation strategies: focuses on beliefs that, once in a negative mood, little can be done to attenuate one's emotions. (6) Lack of emotion clarity: taps to what extent individuals know and understand their emotions. Reliability and validity of the DERS have been demonstrated in previous studies (Ehring, et al., 2010; Gratz & Roemer, 2004; Gratz & Tull, 2010). Cronbach's α values for the present sample were in the range of 0.80 and 0.96 for the DERS subscales.

3.1.3 Procedure

After participants had given informed consent, the SCID was conducted. Next, participants completed the self-report measures.

3.1.4 Data analysis

A multivariate analysis of covariance (MANCOVA) was conducted with group as independent variable and the DERS subscales as dependent variables. Since groups differed significantly regarding age, this variable was used as a covariate. Subsequent ANCOVAs and post-hoc comparisons with Bonferroni correction for multiple testing were used to further explore group differences regarding the single subscales of the DERS. Furthermore, Cohen's *d* effect sizes for group differences were computed.

3.2 Study 2: Mood regulation expectancies and emotion avoidance in depression vulnerability

3.2.1 Participants

Since individuals who have experienced previous depressive episodes are at increased risk for future depressive episodes, an examination of recovered depressed subjects may provide insight into factors involved in depression vulnerability (Just, et al., 2001). Therefore, 20 never-depressed individuals (ND) and 20 formerly-depressed individuals (FD) participated in this study. They were recruited via advertisements in the local media. Exclusion criteria for both groups were any current axis-I disorder according to *DSM-IV* (APA, 1994). In addition, subjects in the ND group must not have had any *DSM-IV* axis-I disorder in the past. Subjects were furthermore excluded from the FD group if they had a life-time diagnosis of a manic episode, psychosis, posttraumatic stress disorder, or borderline personality disorder according to *DSM-IV*. Participants in

the FD group must have had a history of at least one currently remitted major depressive episode according to *DSM-IV*. According to the consensus recommendations (Frank, Prien, Jarrett, & Keller, 1991), current remission from depression was defined as the subject reporting minimal symptoms for a minimum of 12 weeks which means that the subject did no longer meet diagnostic criteria for a major depressive episode, and scored less than or equal to 10 on the Hamilton Depression Rating Scale (Hamilton, 1960). In line with corresponding recommendations (Dozois, Dobson, & Ahnberg, 1998) FD were also excluded if they scored higher than or equal to 12 on the Beck Depression Inventory-II (Kühner, Bürger, Keller, & Hautzinger, 2007).

3.2.2 Material

To assess current and past symptoms of mental disorders, the German version of the SCID (Wittchen, et al., 1997) was conducted by the first author who was specifically trained for SCID administration and had more than 3 years of experience using the SCID. Previous studies have demonstrated good reliability for the SCID (e.g., Williams et al., 1992).

In order to assess current levels of depressive symptoms, the *Beck Depression Inventory-II* (BDI-II; Kühner, et al., 2007) and the *Hamilton Rating Scale for Depression* (HRSD; Hamilton, 1960) were used. As a self-report measure the BDI-II consists of 21 items. Scores range from 0 to 63 and higher scores indicate higher levels of depressive symptoms. Previous studies have demonstrated good psychometric properties for the BDI-II (Kühner, et al., 2007). As a clinician-administered measure of depression severity the HRSD consists of 17 items. Scores range from 0 to 52 and higher scores indicate more severe depression. The HRSD is widely used by both researchers and clinicians (Bagby, Ryder, Schuller, & Marshall, 2004). The HRSD was conducted by the first author.

To measure current negative mood, the *Positive and Negative Affect Schedule* (PANAS; Krohne, Egloff, Kohlmann, & Tausch, 1996) was employed. The negative affect subscale of the PANAS consists of 10 items. The reliability and validity of the PANAS has been demonstrated (Krohne, et al., 1996).

In order to assess emotion avoidance, the *Emotion Avoidance Subscale* of the *Need for Affect Scale* (NAS; Appel, 2008; Maio & Esses, 2001) was used. This self-report scale consists of 13 items. For the purpose of this study and for methodological reasons, the poles of the seven-point Likert scale were changed from -3 and +3 to 0 and 7. Higher scores indicate stronger emotion avoidance. Previous research supported the reliability and validity of the scale (Appel, 2008; Maio & Esses, 2001).

To measure NMR expectancies, the 15-item short form of the *Generalized Expectancies for Negative Mood Regulation Scale* (NMR-SF; Pfeiffer, et al., 2011) was used. Higher scores indicate higher levels of expected negative mood regulation ability. The validity and the reliability of the NMR-SF were shown to be adequate (Pfeiffer, et al., 2011).

3.2.3 Procedure

First, written informed consent was obtained from all subjects before admission to the study. Thereafter, the SCID was conducted and then participants completed the self-report measures.

3.2.4 Data analysis

Groups were compared using univariate analyses of covariance (ANCOVA). Bonferroni corrections for multiple testing were applied since two dependent variables

were tested at the same time. Pearson correlation coefficients were computed to test the associations between NMR-SF and NAS scores.

3.3 Study 3: Mood Regulation and Cognitive Reactivity in Depression Vulnerability

3.3.1 Participants

The same twenty never-depressed individuals (ND) and 20 formerly-depressed individuals (FD) as in Study 2 took part in this study.

3.3.2 Material

DSM-IV diagnoses were obtained using the German version of the SCID (Wittchen, et al., 1997). The BDI-II (Beck, et al., 1996; Kühner, et al., 2007) and the HRSD (Hamilton, 1960) were used to assess depressive symptoms. Current negative mood was assessed by the PANAS (instruments described in section 3.2.2).

The *Scrambled Sentences Test* (SST; Wenzlaff & Bates, 1998) was used to assess participants' bias to interpret ambiguous information in a negative way. Subjects received a list of scrambled sentences (e.g., "bright the very dismal looks future") and were instructed to write a number above five of the six words of each scrambled sentence to produce a grammatically correct sentence in either a negative ("the future looks very dismal") or a positive ("the future looks very bright") way. Participants completed two blocks of 20 sentences, one prior to a mood induction and one after this mood induction. The order of administration of the two SST forms was counterbalanced across subjects. Participants were given 4 minutes to complete each block of sentences. They were instructed to complete as many of the sentences as possible during this time. The SST was scored by calculating the ratio of negative sentences over the total number of completed sentences. The change from the first SST form (prior to the mood

induction) to the second SST form (after the mood induction) was used as an index of cognitive reactivity. In correspondence to Segal et al. (2006), residualized change scores were computed using a simple linear regression model. Post-SST scores were predicted by pre-SST scores, and the standardized residuals for each case were saved from this model, resulting in an unconfounded measure of SST change (Cohen, Cohen, West, & Aiken, 2003). The SST has been shown to be an appropriate and methodologically sound measure of negative interpretive biases (Rude, Wenzlaff, Gibbs, Vane, & Whitney, 2002; Watkins & Moulds, 2007b).

In order to assess mood regulation processes, participants were instructed to write down their memories about a sad autobiographical event during the negative mood induction procedure. The report of each participant was split into two equal halves according to word number. The texts were then analyzed with the computerized text analysis program TAS/C. This software was originally designed for applications in the field of psychotherapy research (Mergenthaler, 1993; Mergenthaler, 1996). Using an emotion dictionary, the TAS/C calculates the proportion of words with a positive and negative emotional tone for the first and the second half of the memory task (Mergenthaler, 1993).

3.3.3 Procedure

Prior to the experimental procedure, subjects were interviewed with the SCID and the HRSD. Thereafter, participants completed the BDI-II, then the PANAS and then the first form of the SST. Subsequently, subjects participated in an 8-minute negative mood induction procedure. During this procedure, participants listened to a sad piece of music (orchestral introduction by Prokofiev entitled *Russia under the Mongolian Yoke* from the movie *Alexander Nevsky*, re-mastered at half speed, presented via earphones) and wrote

down details about an autobiographical event when they felt sad. This type of mood induction has proven to effectively induce a negative mood state in previous studies (Gemar, et al., 2001; Ingram, Bernet, & McLaughlin, 1994; Martin, 1990; Segal, et al., 2006). After the negative mood induction, participants completed the PANAS again and the second version of the SST.

3.3.4 Data analysis

Groups were compared regarding age, gender, levels of depressive symptoms (BDI-II), baseline negative mood (PANAS), and baseline interpretive bias (SST) using t-tests. Using repeated measures ANOVA it was tested whether the mood induction procedure worked equally effective in both groups. To compare the groups regarding cognitive reactivity and mood regulation processes, several ANCOVA were conducted. To control for possible confounds, BDI-II scores as well as the administration order of the two SST forms were used as covariates. To investigate specific associations between mood regulation processes and cognitive reactivity, partial correlations were computed independently for each group. To control for possibly confounding variables, BDI-II scores, baseline mood (PANAS), and SST administration order were partialled out. Finally, three corresponding multiple hierarchical regression analyses were conducted to further test whether these differential associations between cognitive reactivity and mood regulation processes in the first and second phase of the autobiographical memory task were group-specific.

4 Summary of the studies

In this section, the main issues of the three studies presented in detail as original articles will be presented. For each of the studies, a brief overview will be given on the

specific research questions the study deals with, followed by a short summary and discussion of the obtained results.

4.1 Study 1: Specific emotion regulation impairments in major depression

This study investigated specific emotion regulation difficulties simultaneously in patients with MDD, in patients with AN, and in healthy controls to investigate whether emotion regulation difficulties represent transdiagnostic phenomena underlying different forms of psychopathology.

4.1.1 Hypotheses

Hypothesis 1

Based on theoretical models that describe emotion regulation deficits as transdiagnostic phenomena (Campbell-Sills & Barlow, 2007; Kring & Sloan, 2010; Taylor, et al., 1997) and in line with previous studies that separately showed emotion regulation deficits in both disorders (Brockmeyer et al., 2012a; Ehring, et al., 2010; Harrison, et al., 2009; Liverant, et al., 2008), it was expected that MDD and AN will report greater emotion regulation difficulties than controls, but that they will not differ from each other concerning the extent of emotion regulation difficulties.

4.1.2 Results and discussion

As compared to healthy controls, both MDD and AN showed greater difficulties concerning both the experience and the differentiation as well as the attenuation and the modulation of emotions. This is in line with findings from previous studies (Gilboa-Schechtman, et al., 2006; Harrison, et al., 2009; Liverant, et al., 2008; Svaldi, et al., 2012) and supports theoretical models that attach great importance to emotion regulation

impairments in the development and maintenance of mood and eating disorders (Campbell-Sills & Barlow, 2007; Haynos & Fruzzetti, 2011).

MDD as well as AN showed comparably elevated deficits regarding the experience and differentiation of emotions (i.e., awareness, clarity, and acceptance towards one's own emotions). This finding is in line with the notion of emotion regulation difficulties as transdiagnostic phenomena (Campbell-Sills & Barlow, 2007; Kring & Sloan, 2010; Taylor, et al., 1997) and with the results of a previous study (Svaldi, et al., 2012).

However, as compared to AN, MDD exhibited greater deficits regarding the attenuation and modulation of emotions (i.e., engaging in goal-directed behaviour when distressed, impulse control, access to effective regulatory strategies). This finding is in contrast with a previous study (Svaldi, et al., 2012) but may be explained by the lower sample sizes and thus lower power in this previous study. In sum, these findings support the notion of emotion regulation difficulties as transdiagnostic phenomena, and suggest that depression may be characterized by broader and greater emotion regulation difficulties than anorexia nervosa.

4.2 Study 2: Mood regulation expectancies and emotion avoidance in depression vulnerability

The present study is the first to investigate two specific kinds of meta-mood beliefs (NMR expectancies and generalized emotion avoidance) in community-based samples of individuals at high versus low risk for depression.

4.2.1 Hypotheses

Theoretical considerations and preliminary empirical evidence for the potential role of maladaptive meta-mood beliefs in depression and depression vulnerability

(Campbell-Sills & Barlow, 2007; Campbell-Sills, et al., 2006b; Catanzaro & Mearns, 1990) gave rise to the following hypotheses:

Hypothesis 1:

Formerly-depressed individuals (high risk group) will report lower NMR expectancies than never-depressed individuals (low risk group).

Hypothesis 2:

Formerly-depressed individuals will report stronger emotion avoidance than never-depressed individuals.

Hypothesis 3:

NMR expectancies will be inversely related to emotion avoidance.

4.2.2 Results and discussion

Consistent with the first hypothesis, individuals at high risk for depression reported lower NMR expectancies than individuals at low risk for depression, irrespective of current levels of depressive symptoms. This finding is in line with previous studies which observed that low NMR expectancies are associated with higher levels of depressive symptoms (Catanzaro & Mearns, 1990; Davis, et al., 2005).

The results are also in line with the second hypothesis as vulnerable individuals showed stronger emotion avoidance than non-vulnerable individuals. By specifically assessing a generalized motive of emotion avoidance and by using a community-based sample, this finding extends results from previous studies which reported low emotional acceptance in currently-depressed individuals (Campbell-Sills, et al., 2006b) and results from studies with student samples that demonstrated greater emotion regulation

difficulties in formerly-depressed as compared to never-depressed individuals (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008; Ehring, et al., 2010).

In line with the third hypothesis, NMR expectancies were inversely associated with emotion avoidance, even after controlling for current levels of depressive symptoms. This finding supports the idea that individuals with stronger beliefs in their mood regulation abilities may strive to avoid negative emotions to a lesser degree (Catanzaro & Mearns, 1990). Correspondingly, avoidant coping was found to be inversely associated with NMR expectancies in students (Catanzaro & Greenwood, 1994). Alternatively, these findings may be interpreted in the way that permanent avoidance of negative emotions may impede the development of adaptive emotion regulation abilities. In sum, these findings strengthen the idea of specific emotion processing deficits in depression vulnerability.

4.3 Study 3: Mood Regulation and Cognitive Reactivity in Depression Vulnerability

The extent to which individuals are able to accurately assess their own emotion regulation deficits can be questioned (Robinson & Clore, 2002). Such self-evaluations may require a certain degree of insight and meta-cognitive abilities, and they may be biased by negative mood and demand characteristics. Particularly, self-reports of emotion regulation may be confounded by current experiences of aversive affective states (Cole, Martin, & Dennis, 2004). Therefore, the present study made use of a negative mood induction procedure and an unobtrusive method of assessing underlying mood regulation processes, i.e. computerized quantitative text analysis of written autobiographic memories.

Two possible mechanisms of mood regulation underlying cognitive reactivity in vulnerable and non-vulnerable individuals were examined using a cross-sectional,

experimental design. One possible mechanism may work in a way that high-risk individuals are less able to disengage from negative information in a negative mood (Caseras, et al., 2007; Eizenman, et al., 2003) thereby showing ongoing negative thinking. Another possible mechanism may work in a way that, in contrast to high-risk individuals, low-risk individuals may be more able to retrieve mood-incongruent memories in a negative mood thereby coming up with more positive cognitions. This latter process would also provide a possible explanation for the paradoxical findings of reversed cognitive reactivity in individuals at low risk for depression (Beevers, et al., 2009; Segal, et al., 2006).

4.3.1 Hypotheses

Hypothesis 1:

Based on the findings of numerous studies (see Scher, et al., 2005, for a review), formerly-depressed individuals were expected to show an increased cognitive reactivity.

Hypothesis 2:

Also based on previous studies with healthy and dysphoric individuals using different methodologies (Forgas & Ciarrochi, 2002; Josephson, et al., 1996), it was expected that formerly-depressed and never-depressed individuals will initially not differ regarding the retrieval of positively and negatively toned emotion words in an autobiographical memory task during a negative mood induction.

Hypothesis 3:

Based on theoretical models (Forgas & Ciarrochi, 2000; Forgas, et al., 1998) and previous studies with healthy and dysphoric subjects (Forgas & Ciarrochi, 2002; Josephson, et al., 1996), it was expected that differences in information processing will

occur between vulnerable and non-vulnerable individuals later in the temporal sequence. Specifically, low-risk individuals were expected to retrieve more positively toned emotion words (indicating mood-incongruent information processing) whereas high-risk individuals were expected to retrieve more negatively toned emotion words (indicating insufficient disengagement from negative information) in the second half of the autobiographic memory task.

Hypothesis 4:

Based on theoretical models of cognitive processes in affect regulation (Forgas & Ciarrochi, 2000; Forgas & Ciarrochi, 2002) and on previous studies that demonstrated associations between depression/depression vulnerability and a lack of disengagement from negative information (Eizenman, et al., 2003; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005) as well as with a lack of mood-incongruent information processing (Jutta Joormann & Siemer, 2004; Jutta Joormann, Siemer, et al., 2007), it was expected that the proportion of positively toned emotion words in the second phase of the task will be inversely associated with cognitive reactivity. Conversely, the proportion of negatively toned emotion words in the second phase of the task was expected to be positively associated with cognitive reactivity.

4.3.2 Results and discussion

Consistent with the first hypothesis, formerly-depressed individuals showed a higher cognitive reactivity as compared to never-depressed individuals. This result replicates and extends findings from previous studies (for a review see Scher, et al., 2005) by using a measure of information processing bias instead of a self-report scale. In line with the second hypothesis, vulnerable and non-vulnerable subjects were indistinguishable regarding the proportion of positive and negative emotion words retrieved in the first

phase of the autobiographical memory task. This matches findings from a previous study demonstrating that dysphoric and non-dysphoric students did not differ in initially choosing a negative autobiographical memory following a negative mood induction (Josephson, et al., 1996). As predicted in the third hypothesis, high-risk individuals retrieved fewer positive emotion words in the second phase of the autobiographic memory task. This finding may indicate that individuals who are vulnerable to depression lack the ability to sufficiently retrieve (mood-incongruent) positive autobiographic memories in a sad mood state. This extends the Josephson et al. study (1996) by analyzing the responses of formerly-depressed individuals instead of students with an elevated BDI score. The present study further extends these findings by using quantitative text analysis instead of a forced-choice paradigm for assessing memory processes in mood regulation.

However, the present findings are also in contrast to some previous studies (Joormann & Siemer, 2004; Joormann, Siemer, et al., 2007). In these previous studies, dysphoric and non-dysphoric participants as well as currently-depressed, formerly-depressed, and healthy controls did not differ in the number, specificity, and valence of their autobiographical memories. These discrepancies may be due to the different methods applied in these previous studies. The computerized quantitative text analysis used in the present study may allow for a more fine-grained examination of memory processes in mood regulation than the forced-choice paradigms or rating procedures used by the prior studies.

As opposed to the third hypothesis, vulnerable and non-vulnerable individuals did not differ regarding the number of negative emotion words retrieved in the second phase of the autobiographic memory task. Thus, vulnerable individuals may not exhibit more difficulties disengaging from negative emotion recall than non-vulnerable

individuals. This is in contrast with findings from studies using eye-tracking measures which observed that depressed subjects focused their attention longer on negative stimuli than controls did (Caseras, et al., 2007; Eizenman, et al., 2003). These discrepancies may be due to differences in the methods applied.

However, results of the present study still support the idea that disengagement from negative emotion recall is a relevant component of information processing in depression vulnerability: In line with the fourth hypothesis, the number of negative emotion words retrieved in the second phase of the task was positively correlated with cognitive reactivity in formerly-depressed individuals. A different pattern emerged for non-vulnerable individuals: Subjects who retrieved less positive emotion words in the second phase of the task exhibited a higher cognitive reactivity. This finding suggests that differential processes underlie cognitive reactivity in vulnerable and non-vulnerable individuals. For non-vulnerable individuals a lack of mood-incongruent memory retrieval may be responsible for subsequent negative thinking. For vulnerable individuals a deficit in the ability to disengage from negative stimuli may result in negative thinking.

5 General discussion

The findings of the first study suggest that a broad spectrum of difficulties in emotion regulation appears in depression as well as in anorexia nervosa. Furthermore, both disorders do not seem to differ regarding deficits in the experience and differentiation of emotions, suggesting these emotion regulation deficits to be transdiagnostic phenomena. However, depression seems to be associated more strongly with self-reported difficulties in attenuating and modulating emotions. The strengths of the study lie in (a) the relatively large sample sizes, (b) the examination of subjects with non-

comorbid disorders, and (c) the assessment of central and defining facets of emotion regulation. Moreover, this is one of the first studies comparing samples of patients with different so-called emotional disorders (Alloy & Riskind, 2006; Barlow, Allen, & Choate, 2004) concerning a number of clinically relevant difficulties in emotion regulation. The findings speak out in favour of continuing along this path. Future studies on differences in emotion regulation may broaden the present findings by covering not only samples of MDD and AN but also of anxiety and substance use disorders as these latter two were likewise found to be associated with emotion regulation difficulties (Aldao, et al., 2010). In addition, future studies may also examine whether there are differences between subclasses of these disorders. For example, it may be interesting to test whether patients with chronic and episodic depression differ from each other regarding certain aspects of emotion regulation. Similarly, it should be of interest to find out whether there are differences between various eating disorders such as anorexia nervosa, bulimia nervosa, and binge eating disorder. Gaining more insight into such differences may help to individually tailor and refine therapeutic interventions for these different disorders and subclasses of disorders.

The findings of the second study suggest that meta-mood beliefs such as generalized negative mood regulation expectancies and a generalized motivation to avoid emotional experience are involved in depression vulnerability. Furthermore the findings suggest that either low confidence in one's mood regulation skills may fuel emotional avoidance or prolonged emotional avoidance may impede the development of adaptive mood regulation skills. The strengths of the study lie in (a) the inclusion of a high-risk group, (b) the use of measures for meta-mood beliefs rather than for emotion regulation strategies, (c) the use of a community-based sample instead of a student sample. Future studies using prospective longitudinal designs may examine whether marked emotional

avoidance and low confidence in one's emotion regulation competencies represent precursors of depressive symptoms and if so, how these meta-mood beliefs interact with each other and with other variables in setting up depressive symptoms. Along these lines, one avenue for future research would be to investigate how beliefs about the feasibility of particular emotion regulation strategies influence the spontaneous selection of these particular strategies. Finding answers to these questions may help to improve prevention programmes and to refine existing treatment approaches in order to facilitate sustained recovery from depressive episodes. This may comprise informing depressed patients about the benefits and functions of certain affective responses in daily life as well as practising skills to recognize, accept, and modulate such affective responses.

The findings of the third study suggest that vulnerable individuals are less able to retrieve mood-incongruent memories in a negative mood state. Furthermore, the findings suggest that for non-vulnerable individuals a lack of mood-incongruent memory retrieval may be responsible for subsequent negative thinking whereas for vulnerable individuals a deficit in the ability to disengage from negative stimuli may result in negative thinking. Metaphorically, persisting negative cognitions in non-vulnerable individuals may be due to a reduced ability to "drench the fire with water" whereas persisting negative cognitions in vulnerable individuals may be due to a reduced ability to "stop adding fuel to the fire". The strengths of the study lie in (a) the absence of constricting behavioural instructions, and the focus on naturally occurring, spontaneous processes of mood regulation, (b) the application of quantitative text analysis as an unobtrusive method to assess cognitive processes in mood regulation instead of forced-choice paradigms or rating procedures, (c) the inclusion of a community-based high-risk group. Particularly the first two points enrich the findings in an important way: The

methodological approach used in this study allows for the assessment of naturally occurring tendencies of affect regulation. In contrast, study designs using instructions of how and when participants shall regulate some affective state may be able to assess the participants' *capacity* to regulate a certain affective state. However, many individuals with high capacities of affect regulation may still have considerable difficulties in affect regulation in their daily lives since they lack the *tendency* to actually use such affect regulation strategies (Berkman & Lieberman, 2009). Hence, the methodological approach used in the present study may provide an unobtrusive window into spontaneous cognitive-linguistic processes of affect regulation underlying cognitive reactivity. Furthermore, the inclusion of a community-based sample increases the ecological validity and generalizability of the results. The findings of this study call for further investigating cognitive mechanisms of mood regulation underlying thinking patterns in subjects vulnerable to depression. For instance, it would be of great interest to examine whether such a lack of disengagement from negative material represents a precursor of later depressive episodes. Particularly, it would be interesting if a lack of disengagement mediates the relationship between a pronounced cognitive reactivity and future depressive episodes. In addition, future studies may also benefit from focusing more on less deliberate but more incidental, spontaneously occurring forms of affect regulation (Berkman & Lieberman, 2009). Increased knowledge of such underlying mechanisms of affect regulation and negative thinking may help to refine existing treatment approaches in order to gain more sustainable improvement.

The present studies have some limitations. First, all three studies used a cross-sectional design making it impossible to draw causal conclusions. Rather than being vulnerability factors, the observed differences between formerly-depressed and never-depressed individuals could represent a scar of past depressive episodes (Just, et al.,

2001). The experience of a depressive episode may for example promote an increased alertness for and avoidance of negative mood. Similarly, low confidence in one's negative mood regulation abilities may result from actually failed attempts to improve one's mood. Thus, future studies may use prospective longitudinal designs to investigate whether such deficits in affect regulation observed in formerly-depressed individuals occur rather as precursors or as consequences of depressive episodes. Second, the first two studies relied solely on self-report measures that are always prone to memory biases and demand characteristics. Future studies may benefit from the use of additional, more objective measures (e.g., behavioural and cognitive performance tasks, physiological data) in order to tap various dimensions of affect regulation. Third, the sample sizes of study 2 and 3 were relatively small. Thus, the present findings may be regarded as rather preliminary. However, the effect sizes underlying the present findings were large and thus increase confidence that these findings can be replicated in larger samples. Finally, future research may benefit from a clearer distinction between mood regulation and emotion regulation. For instance, as suggested by Ekman (1994) it may be of interest to examine whether and how depressed mood lowers the threshold value for arousing emotions of sadness or whether and how depressed mood hampers the modulation of emotions.

The findings of the present studies unanimously suggest that treatments of depression should involve specific elements that foster adaptive affect regulation including abilities to experience and differentiate as well as abilities to modulate and attenuate one's emotions. Existing treatment approaches that already target emotional avoidance and/or emotion regulation in depression and that have gained at least preliminary evidence for their efficacy are for example Exposure-based Cognitive Therapy for depression (Kumar, et al., 2008), Expressive writing (Pennebaker, 1997),

Concreteness Training (Watkins, Baeyens, & Read, 2009), Imagery Rescripting and Reprocessing Therapy (Brewin et al., 2009), Mindfulness-based Cognitive Therapy (Ma & Teasdale, 2004; Teasdale et al., 2000), and Dialectical Behavior Therapy (Feldman, Harley, Kerrigan, Jacobo, & Fava, 2009). However, identifying disorder-specific affect regulation deficits may advance the refinement and further development of effective treatment approaches. Furthermore, implementing a more direct training of basic cognitive processes of mood regulation into such comprehensive treatment packages seems to be promising. For example, it has been suggested that training depressed individuals in intentional forgetting could prove to be an effective strategy to counteract mood-congruent biases in information processing (Joormann, Hertel, Brozovich, & Gotlib, 2005) and it has already been demonstrated that a training of attention shifting, for example using a dot probe task or mindfulness-based interventions, can reduce emotion dysregulation (Wadlinger & Isaacowitz, 2011). Mindfulness training was furthermore shown to increase working memory capacity which in turn decreased self-reported negative affect (Jha et al., 2010). Similarly, another study showed that computerized working memory training using emotional material improved the ability to disengage from goal-irrelevant, negative emotional pictures in an emotional Stroop task (Schweizer, Hampshire, & Dalgleish, 2011). These findings point to a promising avenue for future research by translating basic research findings of specific affect regulation impairments in depression into new treatment approaches.

6. Abstract

Impairments in affect regulation as well as cognitive reactivity have been considered to play important roles in the development, maintenance, and recurrence of major depressive disorder (MDD). However, there is a lack of studies investigating, (a) whether certain difficulties in emotion regulation are specific for MDD, (b) whether certain meta-mood beliefs are associated with an increased risk for MDD, and (c) whether reduced abilities to engage in mood-incongruent information processing and to disengage from negative information are associated with depression vulnerability, and whether those specific mood regulation processes underlie the mechanism of cognitive reactivity. Therefore, Study 1 of this dissertation compared patients with MDD with a clinical and a healthy control group regarding a comprehensive set of central and clinically relevant emotion regulation difficulties. Study 2 investigated two specific kinds of meta-mood beliefs [i.e., generalized negative mood regulation (NMR) expectancies and a generalized motive to avoid emotional experience (EA)] in formerly-depressed and never-depressed subjects. Finally, Study 3 tested whether two specific mood regulation processes (i.e., mood-incongruent information processing and disengagement from negative information) differentially appear in vulnerable and non-vulnerable individuals, and whether they can account for differences in cognitive reactivity. Patients with MDD reported greater emotion regulation difficulties than healthy controls and did not differ from the clinical control group regarding difficulties with the experience and differentiation of emotions, however, patients with MDD reported greater difficulties than clinical controls regarding the attenuation and modulation of emotions (Study 1). As expected, vulnerable individuals reported lower NMR expectancies and stronger EA as compared to non-vulnerable individuals, and NMR expectancies were furthermore found to be inversely associated with EA (Study 2). In the second but not in the first

phase of an autobiographical memory task, vulnerable subjects retrieved fewer mood-incongruent (positively toned) emotion words than non-vulnerable subjects (Study 3). Furthermore, vulnerable subjects with a high cognitive reactivity retrieved more negatively toned emotion words. For non-vulnerable subjects a different pattern occurred: Subjects with a high cognitive reactivity retrieved less positively toned emotion words. The findings of the three studies suggest that emotion regulation difficulties constitute transdiagnostic phenomena, and that MDD may be characterized by particularly broad and pronounced deficits in this domain. The results also suggest that maladaptive meta-mood beliefs are associated with depression vulnerability and that individuals with low confidence in their negative mood regulation abilities are concurrently characterized by a stronger avoidance of emotional experience. Furthermore, the results suggest that a reduced ability of mood-incongruent information processing is associated with depression vulnerability, and that two different memory processes of mood regulation may account for cognitive reactivity in individuals who are at high versus low risk for depression. The cross-sectional designs and the use of self-report measures as well as the relatively small sample sizes in two of the studies limit the interpretation of the obtained results. Notwithstanding, the findings of these three studies call for an implementation of specific elements that foster adaptive affect regulation (e.g., computer-assisted training of cognitive processes) into existing treatment packages for MDD.

7. References

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Appendix A: Curriculum vitae

Education / Degrees

since 2011

- Licensed psychotherapist (Postgraduate psychotherapy training, Centre for Psychological Psychotherapy, University of Heidelberg)

since 2008

- Ph.D. student, University of Heidelberg (doctoral grant from the Centre for Psychological Psychotherapy, University of Heidelberg)

2008

- Practical training as psychotherapist at the Department of General Internal Medicine and Psychosomatics, University Hospital Heidelberg

2007 – 2008

- Practical training as psychotherapist at the Department of Psychiatry, University Hospital Heidelberg

2007

- Diploma in psychology, University of Heidelberg (Diploma thesis “Subsyndromal Depression in Primary Care”)

2005

- Internship at the Department of Psychiatry, University Hospital Heidelberg

2004 – 2007

- Master’s level training in psychology, University of Heidelberg

2001 – 2004

- Basic training in psychology, University of Heidelberg

Academic positions

since 2011

- Research assistant, Department of General Internal Medicine and Psychosomatics, University Hospital Heidelberg (PD Dr. H.-C. Friederich)

2008 – 2010

- Research assistant, Department of Clinical Psychology, University of Heidelberg (Prof. Dr. M. Grosse Holtforth, Prof. Dr. A. Kämmerer)

2005 - 2006

- Undergraduate research assistant, Department of Clinical Psychology, University of Heidelberg (Prof. Dr. P. Fiedler)

2003

- Undergraduate research assistant, Institute of Psychology, University of Heidelberg (Prof. Dr. M. Amelang)

Academic and professional affiliations

- Society for Psychotherapy Research (SPR)

Journal articles

Brockmeyer, T., Bents, H., Grosse Holtforth, M., Pfeiffer, N., Herzog, W., & Friederich, H.-C. (2012). Specific emotion regulation impairments in major depression and anorexia nervosa. *Psychiatry Research*, <http://dx.doi.org/10.1016/j.psychres.2012.07.009>

Brockmeyer, T., Grosse Holtforth, M., Bents, H., Kämmerer, A., Herzog, W., & Friederich, H.-C. (2012). Starvation and emotion regulation in anorexia nervosa. *Comprehensive Psychiatry*, 53(5), 496-501. doi: 10.1016/j.comppsy.2011.09.003

Brockmeyer, T., Grosse Holtforth, M., Bents, H., Kämmerer, A., Herzog, W., & Friederich, H.-C. (in press). The thinner the better: self-esteem and low body weight in anorexia nervosa. *Clinical Psychology & Psychotherapy*, n/a-n/a. doi: 10.1002/cpp.1771

Brockmeyer, T., Grosse Holtforth, M., Pfeiffer, N., Backenstrass, M., Friederich, H.-C., & Bents, H. (2012). Mood regulation expectancies and emotion avoidance in depression vulnerability. *Personality and Individual Differences*, 53 (3), 351-354.

Brockmeyer, T., Pfeiffer, N., Grosse Holtforth, M., Zimmermann, J., Kämmerer, A., Friederich, H.-C., & Bents, H. (in press). Mood regulation and cognitive reactivity in depression vulnerability. *Cognitive Therapy and Research*, 1-9. doi: 10.1007/s10608-011-9406-7

Conference presentations

Brockmeyer, T., Grosse Holtforth, M., Krieger, T., Zimmermann, J., Pfeiffer, N., Backenstrass, M., Friederich, H.-C., & Bents, H (2012). *The different facets of avoidance in depression*. Presentation at the 3rd Joint Meeting of the SPR European and UK Chapters, Porto, Portugal.

Brockmeyer, T., Grosse Holtforth, M., Bents, H., Herzog, W., & Friederich, H.-C. (2012). *Nahrungsrestriktion und Gewichtsverlust als ein Pathomechanismus der Emotionsregulation bei Anorexia nervosa* [Dietary restraint and weight loss as pathomechanisms of emotion regulation in anorexia nervosa]. Presentation at the 30th Symposium of the Division of Clinical Psychology and Psychotherapy of the German Psychological Association (DGPs), Luxembourg.

Brockmeyer, T., Ingenerf, K., Herzog, W., & Friederich, H.-C. (2012). *Training kognitiv-behavioraler Flexibilität für Patientinnen mit Anorexia nervosa - vorläufige fMRT-Befunde aus einer randomisiert-kontrollierten Pilotstudie* [Training cognitive-behavioral flexibility in patients with anorexia nervosa - preliminary results from an fMRI-based, randomized controlled pilot trial]. Presentation at the 63rd Working Conference of the German College for Psychosomatic Medicine (DKPM), Munich, Germany.

Friederich, H.-C., Brockmeyer, T., Walther, S., Zastrow, A., Bendszus, M., Weisbrod, M., Herzog, W., Kaiser, S., Stippich, C. (2012). *Neuronale Signatur der eingeschränkten kognitiven Flexibilität bei gewichtsremittierten Anorexie-Patientinnen*. [Neuronal signature of impaired cognitive flexibility in weight-restored patients with anorexia nervosa]. Presentation at the 27th Congress of the German Society for Behaviour Therapy (DGVT), Berlin, Germany.

Brockmeyer, T., Grosse Holtforth, M., Pfeiffer, N., Kämmerer, A., & Bents, H. (2011). *Mood Regulation and the Vulnerability to Depression*. Presentation at the 42nd annual meeting of the Society for Psychotherapy Research (SPR), Berne, Switzerland.

Posters

Pfeiffer, N., Brockmeyer, T., Zimmermann, J., & Backenstrass, M. (2012). *A Two-Component Model of Cognitive Reactivity*. Poster at the 54th Conference of Experimental Psychology (TeaP), Mannheim, Germany.

Brockmeyer, T., Grosse Holtforth, M., Bents, H., Kämmerer, A., Ingenerf, K., Herzog, W., & Friederich, H.-C. (2011). *Interpersonal Motives in Anorexia nervosa*. Poster at the 14th annual meeting of the Society for Interpersonal Theory and Research (SITAR), Zurich, Switzerland.

Brockmeyer, T., Grosse Holtforth, M., Zimmermann, J., Pfeiffer, N., Bents, H., Kämmerer, A., & Friederich, H.-C. (2011). *Aspekte von Vermeidung bei Depression* [Facets of Avoidance in Depression]. Poster at the 29th Symposium of the Division of Clinical Psychology and Psychotherapy of the German Psychological Association (DGPs), Berlin, Germany.

Brockmeyer, T., Bents, H., Kämmerer, A., Ingenerf, K., Herzog, W., & Friederich, H.-C. (2010). *"Ich fühle nur noch Hunger": Emotionsregulation bei Anorexia nervosa* ["All I feel is hunger": Emotion Regulation in Anorexia nervosa]. Poster at the Congress of the German Association for Psychiatry and Psychotherapy (DGPPN), Berlin, Germany.

Brockmeyer, T., Grosse Holtforth, M., Bents, H., Kämmerer, A., Ingenerf, K., Herzog, W., & Friederich, H.-C. (2010). *"Hunger nach Freiheit": Anorexia nervosa und das Streben nach Autonomie* ["Hunger for Freedom": Anorexia nervosa and the Striving for Autonomy]. Poster at the Congress of the German Association for Psychiatry and Psychotherapy (DGPPN), Berlin, Germany.

Brockmeyer, T., Bents, H., Kämmerer, A., & Grosse Holtforth, M. (2010). *Depression und Vermeidung* [Depression and Avoidance]. Poster at the 28th Symposium of the Division of Clinical Psychology and Psychotherapy of the German Psychological Association (DGPs), Mainz, Germany.

Appendix B: Declaration

Erklärung gemäß § 8 Abs. 1 Buchst. b) der Promotionsordnung der Universität Heidelberg für die Fakultät für Verhaltens- und Empirische Kulturwissenschaften

Ich erkläre, dass ich die vorgelegte Dissertation selbstständig angefertigt, nur die angegebenen Hilfsmittel benutzt und die Zitate gekennzeichnet habe.

Erklärung gemäß § 8 Abs. 1 Buchst. c) der Promotionsordnung der Universität Heidelberg für die Fakultät für Verhaltens- und Empirische Kulturwissenschaften

Ich erkläre, dass ich die vorgelegte Dissertation in dieser oder einer anderen Form nicht anderweitig als Prüfungsarbeit verwendet oder einer anderen Fakultät als Dissertation vorgelegt habe.

Name, Vorname _____

Datum, Unterschrift _____

Appendix C: Original articles

1. **Brockmeyer, T.**, Bents, H., Grosse Holtforth, M., Pfeiffer, N., Herzog, W., & Friederich, H.-C. (2012). Specific emotion regulation impairments in major depression and anorexia nervosa. *Psychiatry Research*, <http://dx.doi.org/10.1016/j.psychres.2012.07.009>
2. **Brockmeyer, T.**, Grosse Holtforth, M., Pfeiffer, N., Backenstrass, M., Friederich, H.-C., & Bents, H. Mood regulation expectancies and emotion avoidance in depression vulnerability (2012). *Personality and Individual Differences*, *53* (3), 351-354, <http://dx.doi.org/10.1016/j.paid.2012.03.018>
3. **Brockmeyer, T.**, Pfeiffer, N., Grosse Holtforth, M., Zimmermann, J., Kämmerer, A., Friederich, H.-C., & Bents, H. Mood regulation and cognitive reactivity in depression vulnerability (2011). *Cognitive Therapy and Research*, 1-9. doi: 10.1007/s10608-011-9406-7

Specific emotion regulation impairments in major depression and anorexia nervosa

Timo Brockmeyer^{*a, b}, Hinrich Bents^b, Martin Grosse Holtforth^c, Nils Pfeiffer^d, Wolfgang Herzog^a, & Hans-Christoph Friederich^a

^aUniversity Hospital Heidelberg,
Department of General Internal Medicine and Psychosomatics,
Im Neuenheimer Feld 410, 69120 Heidelberg, Germany

^bUniversity of Heidelberg, Centre for Psychological Psychotherapy,
Hauptstrasse 47-51, 69117 Heidelberg, Germany

^cUniversity of Zurich, Department of Psychology,
Binzmühlestrasse 14 / Box 19, 8050 Zürich, Switzerland

^dSchön Klinik Roseneck,
Am Roseneck 6, 83209 Prien am Chiemsee, Germany

Corresponding author:

Timo Brockmeyer

phone: +49 6221 5637153, email: timo.brockmeyer@med.uni-heidelberg.de

Abstract

Emotion regulation (ER) difficulties have been considered to play an important role in the development and maintenance of various mental disorders such as depression and anorexia nervosa. However, previous research has failed to provide detailed insight into the disorder-specificity of ER difficulties. Therefore, the present study investigated specific ER difficulties in female samples of patients with major depression, patients with anorexia nervosa, and healthy controls (total sample: $N = 140$). As compared to healthy controls, both clinical groups reported greater ER difficulties concerning both the experience and the differentiation as well as the attenuation and the modulation of emotions. Patients in both clinical groups reported comparably elevated ER difficulties regarding the experience and differentiation of emotions. However, depressed patients reported stronger ER difficulties regarding the attenuation and modulation of emotions as compared to patients with anorexia nervosa. These findings support the notion of ER difficulties as transdiagnostic phenomena, and suggest that depression may be characterized by broader and greater ER difficulties than anorexia nervosa.

Keywords: Depression, Anorexia nervosa, Eating disorders, Emotion regulation

1. Introduction

Adaptive emotion regulation (ER) has been proposed to involve a range of cognitive and behavioral processes concerning the experience and differentiation as well as the attenuation and modulation of emotions (Gross and Muñoz, 1995; Gratz and Roemer, 2004; Gratz and Tull, 2010). Recent etiological models of mood and of eating disorders have attached great importance to deficits in ER (Gross and Muñoz, 1995; Taylor et al., 1997; Campbell-Sills and Barlow, 2007; Kring and Sloan, 2010; Haynos and Fruzzetti, 2011), supporting the notion that ER deficits may be regarded as transdiagnostic factors (Gross and Muñoz, 1995; Taylor et al., 1997; Campbell-Sills and Barlow, 2007; Kring and Sloan, 2010). In line with these theoretical models, previous empirical research observed impaired ER in depression (Liverant et al., 2008; Pfeiffer et al., 2011; Brockmeyer et al., 2011b) as well as in eating disorders such as anorexia nervosa (Gilboa-Schechtman et al., 2006; Harrison et al., 2009; Brockmeyer et al., 2011a). Common symptoms of both disorders have been considered to serve an ER function. For example, rumination can be considered a rather abstract and verbal style of information processing which may thereby protect the depressed individual from aversive imagery and associated physiological arousal (Watkins & Moulds, 2007). In anorexia nervosa, restrictive eating, weight loss, and excessive exercising have been considered to provide an escape from aversive emotional arousal (Haynos and Fruzzetti, 2011).

However, the question whether certain ER difficulties are more strongly associated with certain mental disorders than with others has barely been addressed directly in empirical research. In their recent meta-analysis, Aldao, Nolen-Hoeksema, and Schweitzer (2010) have delineated the relationships between various ER strategies and specific mental disorders. Their results indicated that dysfunctional ER strategies (e.g., avoidance, suppression, rumination) were generally more closely associated with depression and anxiety disorders than with eating and substance use disorders. However, this meta-analysis was primarily

based on studies that (i) used only samples with symptoms of one specific disorder each, (ii) have not controlled for the potential influence of comorbid disorders, and that (iii) have investigated different ER strategies with heterogeneous instruments. In consequence, the meta-analysis did not allow concluding stringently whether specific mental disorders were differentially associated with ER deficits. To overcome these shortcomings, Aldao and Nolen-Hoeksema (2010) tested various ER strategies simultaneously in subject samples suffering from symptoms of different mental disorders. In their study, they found that maladaptive strategies (i.e., rumination, suppression) were associated more strongly with symptoms of depression, anxiety, and eating disorders than adaptive strategies (i.e., reappraisal, problem-solving). However, this study used a student sample rather than a clinical sample. Furthermore, the authors investigated some variables (e.g., rumination, problem-solving) that can best be considered antecedents of and responses to emotions (Gross, 1999; Gross and John, 2003) rather than being inherent components of the ER process itself in the sense of experiencing, differentiating, attenuating, and modulating emotions (Gratz and Roemer, 2004).

In studies that compared women suffering from clinical eating disorders and female healthy controls, initially observed differences regarding ER deficits disappeared when levels of depression and anxiety were statistically controlled for (Eizaguirre et al., 2004; Gilboa-Schechtman et al., 2006). Only a few studies have compared clinical samples of patients with eating disorders and patients with mood and/or anxiety disorders so far. These previous studies failed to find any difference between these groups regarding ER deficits (Brockmeyer et al., 2011a; Svaldi et al., 2012). However, these studies were based on rather small sample sizes and thus may have been underpowered. For example, Svaldi et al. (2012) compared, amongst others, 20 patients with anorexia nervosa, 16 patients with major depressive disorder, and 42 healthy controls. The authors concluded that their findings are in line with the notion

of ER deficits as transdiagnostic risk and/or maintaining factors that do not appear to be disorder-specific. Yet, they called for replications with larger samples of patients.

To overcome some of the limitations of previous studies, the present study compared larger samples of patients suffering from either a major depressive disorder or an eating disorder (anorexia nervosa) regarding central ER difficulties. Based on theoretical assumptions of ER deficits as transdiagnostic phenomena (Taylor et al., 1997; Campbell-Sills and Barlow, 2007; Kring and Sloan, 2010) and in line with previous studies that separately demonstrated impaired ER in both disorders (Brockmeyer et al., 2011a; Brockmeyer et al., 2011b; Gilboa-Schechtman et al., 2006; Liverant et al., 2008; Harrison et al., 2009; Pfeiffer et al., 2011) it was expected that the two patient groups will show greater ER difficulties than healthy controls, but that they will not differ from each other concerning the extent of ER difficulties.

2. Methods

2.1 Participants

The sample consisted of 41 women with a major depressive disorder (MDD), 39 women with anorexia nervosa (AN), and 60 female healthy controls (HC). Patients were recruited consecutively from a university-based outpatient centre serving the community (MDD, 44%; AN, 41%) as well as from an inpatient unit of a university hospital. HC were recruited via advertisements in the media and from the university campus. All participants were Caucasian and between 18 and 65 years of age. Exclusion criteria for the HC group were any diagnosis according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994). Exclusion criteria for both clinical groups were a current diagnosis of any substance use disorder, and a lifetime diagnosis of a manic episode or psychosis. Borderline personality disorder was chosen as another exclusion criterion due to the special role ER difficulties play in this disorder (Lieb et al., 2004). Further exclusion criteria for the MDD group were any comorbid eating disorder. Conversely, patients with anorexia nervosa were

carefully assessed for a current mood disorder, as only patients without comorbid mood disorders were included in the study. In the AN group, 32 patients had restricting subtype and 7 patients had binge/purge subtype anorexia nervosa. Due to the small number of AN from the binge/ purging subtype, the two groups were not differentiated in the present study. This seems all the more justifiable since in a previous study no differences were found between AN and women with bulimia nervosa regarding ER difficulties (Harrison et al., 2010). Twenty-three percent of the patients were taking psychotropic medication (MDD, 29%, AN, 15%). Written informed consent was obtained from all participants. The study had been approved by the ethics committee of the university.

2.2 Material

In order to assess current and past episodes of mental disorders, the *Structured Clinical Interview for DSM-IV Axis I and II* (SCID; German version: Wittchen et al., 1997) was conducted by the first author and 8 clinicians who were all specifically trained for SCID administration and had more than 3 years of experience using the SCID. The SCID has demonstrated good reliability in previous studies (Williams et al., 1992). The *Difficulties in Emotion Regulation Scale* (Gratz and Roemer, 2004) was used to assess specific ER difficulties. The DERS is a 36-item self-report scale that allows capturing a broad array of essential deficits in emotional processing. Participants are asked to rate their ER difficulties on a five-point Likert scale. The DERS has six discrete but interconnected subscales: (1) Non-acceptance of emotional responses: This subscale assesses a tendency to have negative secondary emotional responses to one's own negative emotions. (2) Difficulties in engaging in goal-directed behavior: Items of this subscale reflect difficulties concentrating and accomplishing tasks when experiencing negative emotions. (3) Impulse control difficulties: This subscale taps difficulties with remaining in control of behavior when experiencing negative emotions. (4) Lack of emotional awareness: This subscale is composed of items reflecting difficulties associated with attending to and acknowledging one's emotions. (5)

Limited access to effective ER strategies: This subscale focuses on beliefs that, once upset, little can be done to regulate one's emotions. (6) Lack of emotion clarity: This subscale taps how much individuals know and understand the emotions they are experiencing. Reliability and validity of the original English as well as the German version of the DERS have been demonstrated in previous studies (Gratz and Roemer, 2004; Ehring et al., 2010; Gratz and Tull, 2010). Cronbach's α values in the present study were in the range of 0.80 and 0.96 for the DERS subscales.

3. Results

3.1 Sample characteristics

The mean body mass index was 14.91 ($SD = 1.74$) for AN, 21.25 ($SD = 1.61$) for MDD, and 21.59 ($SD = 1.75$) for HC. In the MDD group 17.9% had a severe depressive episode, 76.9% had a moderate depressive episode, and 5.1% had a mild depressive episode. Participants in the MDD group had a mean age of 35 years ($SD = 13$ years). The mean age of participants in the AN group was 25 years ($SD = 7$ years). In the HC group, participants had a mean age of 34 years ($SD = 14$ years). The groups differed significantly from each other regarding age, $F(2, 137) = 9.25, p < 0.001$. To account for this potential confound, age was used as a covariate in further analyses.

3.2 Group differences regarding ER difficulties

A multivariate analysis of covariance (MANCOVA) was conducted with subject group as independent variable and the subscales of the DERS as dependent variables. MANCOVA results indicated significant differences between the groups, *Pillai's trace* = 0.61, $F(12, 264) = 9.74, p < 0.001$. Subsequent ANCOVAs with subject group as the independent variable and the single DERS subscales as the dependent variable revealed significant group differences in terms of all DERS subscales. Results are summarized in Table 1. Post-hoc comparisons with Bonferroni corrections revealed that MDD reported greater ER difficulties than HC across all subscales of the DERS with large effect sizes (Non-acceptance,

$d = 1.34$; Awareness, $d = 1.01$; Clarity, $d = 1.42$; Goal-directed behavior, $d = 1.74$; Impulse control, $d = 1.42$; Access to ER strategies, $d = 2.28$). Similarly, AN reported greater ER difficulties than HC in all but one subscale of the DERS (Goal-directed behavior, $d = 0.55$) likewise with large effect sizes (Non-acceptance, $d = 0.83$; Awareness, $d = 0.97$; Clarity, $d = 1.15$; Impulse control, $d = 0.95$; Access to ER strategies, $d = 1.55$). MDD and AN did not differ regarding those subscales of the DERS that primarily capture the experience and differentiation of emotions (Non-acceptance, $d = 0.32$; Awareness, $d = 0.12$; Clarity, $d = 0.12$). However, MDD reported greater ER difficulties than AN in the domain of attenuating and modulating emotions, with small to large effect sizes (Goal-directed behavior, $d = 0.94$; Impulse control, $d = 0.35$; Access to effective ER strategies, $d = 0.53$). The restricting AN subgroup did not differ from the binge/purge AN subgroup in any of the DERS subscales, all $p > 0.17$. Running the analysis with AN from the binge/purge type excluded resulted in the same pattern of results. Since individuals with AN are usually younger than individuals with MDD, the analysis was also repeated without age as a covariate. The corresponding ANOVA revealed largely the same results. However, in this analysis, AN differed from HC regarding all DERS subscales, including Goal-directed behavior, $p = 0.02$. Furthermore, in this analysis, AN and MDD did not differ regarding the Impulse control subscale, $p = 0.16$.

4. Discussion

The present study simultaneously investigated a comprehensive set of specific ER difficulties in patients currently suffering from either a major depressive disorder or anorexia nervosa. In line with previous studies, the findings of the present study underscore the relevance of ER deficits with reference to mood and eating disorders since both clinical groups reported increased ER difficulties as compared to healthy controls (Gilboa-Schechtman et al., 2006; Liverant et al., 2008; Harrison et al., 2009; Brockmeyer et al., 2011a; Brockmeyer et al., 2011b; Pfeiffer et al., 2011).

Also in line with previous studies (Brockmeyer et al., 2011a; Svaldi et al., 2012), the notion of ER difficulties as transdiagnostic phenomena (Taylor et al., 1997; Campbell-Sills and Barlow, 2007; Kring and Sloan, 2010) was supported given that MDD and AN did not differ regarding those aspects of ER that are concerned with experiencing and differentiating emotions (awareness, clarity, and acceptance towards one's own emotions). However, the present results also suggest that depression may be characterized by broader and greater self-reported impairments in ER. Particularly those aspects of ER that refer to the attenuation and modulation of emotions (engaging in goal-directed behavior when distressed, impulse control, access to effective ER strategies) seem to be more impaired in depression than in anorexia nervosa. This finding is in contrast to previous studies which found no differences between MDD and AN regarding the DERS total score (Brockmeyer et al., 2011a), as well as regarding all but one DERS subscale (Svaldi et al., 2012). These discrepancies may in part be explained by the lower sample sizes and thus lower power to detect group differences in the previous studies. However, Svaldi et al. (2012) observed even greater difficulties regarding impulse control in AN as compared to MDD. Furthermore, in the study by Svaldi et al. (2012) mean scores on DERS subscales in the AN group seem considerably higher than in the present study. This may be explained by differences in other clinical or sociodemographic variables (e.g., education), in which the two study samples may have differed. For example, both AN and MDD were considerably younger in the present study as compared to the study by Svaldi et al. (2012).

In any case, it seems valuable to further examine specific ER difficulties as well as their behavioral and motivational features in MDD and AN. Even if subjects affected by these two disorders do not differ in terms of some ER difficulties, they may differ in the ways they try to cope with them. For example, MDD may be especially prone to engage in excessive rumination whereas AN may be especially prone to engage in restricted eating or excessive exercising. In keeping with previous studies that separately demonstrated ER deficits in

depression (Liverant et al., 2008; Brockmeyer et al., 2011b; Pfeiffer et al., 2011) and in anorexia nervosa (Gilboa-Schechtman et al., 2006; Harrison et al., 2009; Brockmeyer et al., 2011a), the findings of the present study suggest that treatments of depression and anorexia nervosa should include interventions that foster adaptive ER strategies. Indeed, several treatment approaches for these disorders already target the improvement of ER (Exposure-Based Cognitive Therapy for Depression, Kumar et al., 2008; Emotion Acceptance Behavior Therapy for Anorexia nervosa, Wildes and Marcus, 2011). However, identifying specific ER deficits in different mental disorders may advance the refinement and further development of those treatment approaches.

The present study holds some limitations. First, all participants in this study were female. Thus, results cannot be generalized to male patients. A considerable strength of the present study was the application of strict exclusion criteria: Samples of individuals with only depression and no comorbid anorexia nervosa and vice versa were recruited to examine differences in ER between disorders. As a side-effect however, the resulting samples may be less representative of typical treatment-seeking clinical groups. Another point is that only one single measure of ER was used. Thus, it cannot be ruled out that there are additional disorder-specific ER deficits that were not assessed in this study. Likewise, further clinical or sociodemographic variables that were not assessed systematically in the present study (e.g., education) may have impacted the results. Furthermore in the current study, lifetime diagnoses were not assessed, limiting conclusions of ER specificity. In addition, AN of the restricting subtype and AN of the binge/purge subtype were merged into one sample. Importantly, the same pattern of results emerged when AN of the binge/purge subtype were excluded from the analysis and, in line with previous studies (Harrison et al., 2008), these two subsamples did not differ from each other regarding any of the DERS subscales. However, these analyses may have been underpowered due to the small sample sizes of AN of the binge/purge subtype. Therefore, future research with larger and more balanced sample sizes

may examine in more detail whether there are differences in ER between the restricting and the binge/purge subtype. Finally, it should be noted that the results may be biased by measurement artefacts. The use of self-report measures is always limited by memory biases and demand characteristics. Therefore, future studies should use more objective measures (e.g., behavioral and cognitive performance tasks, physiological data) in addition to self-report measures to further investigate disorder-specific ER deficits. For example, Harrison et al. (2010) found that women with an eating disorder compared to healthy controls showed greater attentional biases towards angry faces in an emotional Stroop task. In addition, Joos et al. (2009) found that AN showed increased fear when exposed to anger-related pictures. Regarding depression, several recent studies using experimental paradigms showed that MDD exhibit ER deficits such as a lack of disengagement from negative material, an impaired ability to spontaneously apply cognitive reappraisal, as well as a reduced access to positive autobiographic memories (for a review see, Joormann and D'Avanzato, 2010).

In contrast to previous studies comparing AN and MDD regarding ER deficits, the present study used larger samples and thus had more power to detect differences between the groups. Furthermore, the present study used subjects with non-comorbid disorders and thus eliminated shared variance among the disorders as a possible confound. Moreover, the present study assessed defining and central facets of ER rather than constructs that are related to ER. Hence, the present study offers new insight to differential associations of specific ER difficulties with depression and anorexia nervosa. The findings of the present study suggest that both disorders are characterized by impairments of central aspects of ER that refer to both the experience and differentiation as well as the attenuation and modulation of emotions. Furthermore, the present findings suggest that these disorders do not differ regarding the first of these two dimensions (experiencing and differentiating), but that major depressive disorder is associated more strongly with the second dimension (attenuating and modulating) than

anorexia nervosa. Future research should continue investigating the role of ER difficulties in the aetiology and treatment of mental disorders.

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Table 1 Group differences regarding emotion regulation difficulties

Difficulties in Emotion Regulation Scale - Subscale	Women with major depressive disorder (<i>n</i> = 41) <i>M</i> (<i>SD</i>)	Women with anorexia nervosa (<i>n</i> = 39) <i>M</i> (<i>SD</i>)	Healthy female controls (<i>n</i> = 60) <i>M</i> (<i>SD</i>)	ANCOVA <i>F</i> (2, 136) <i>p</i>
<i>Experience and differentiation of emotions</i>				
Non-acceptance of emotional responses	18.89 (6.09) ^a	16.74 (7.07) ^a	11.98 (3.99) ^b	19.57 < 0.001
Lack of emotional awareness	18.44 (3.71) ^a	18.97 (5.10) ^a	14.62 (3.78) ^b	15.39 < 0.001
Lack of emotion clarity	14.51 (4.29) ^a	13.95 (4.88) ^a	9.42 (2.71) ^b	24.99 < 0.001
<i>Attenuation and modulation of emotions</i>				
Difficulties in engaging in goal-directed behaviour	18.32 (4.26) ^a	13.85 (5.18) ^b	11.40 (3.65) ^b	32.31 < 0.001
Impulse control difficulties	16.24 (6.32) ^a	14.03 (6.40) ^b	9.37 (2.58) ^c	23.81 < 0.001
Limited access to effective emotion regulation strategies	26.76 (7.86) ^a	22.54 (8.13) ^b	12.82 (3.61) ^c	61.65 < 0.001

Different superscripts denote significant group differences after post hoc comparisons with Bonferroni corrections.

Mood Regulation Expectancies and Emotion Avoidance in Depression Vulnerability

Timo Brockmeyer^{1,5}, Martin Grosse Holtforth², Nils Pfeiffer³, Matthias Backenstrass⁴, Hans-Christoph Friederich¹, & Hinrich Bents⁵

¹University Hospital Heidelberg,
Department of General Internal Medicine and Psychosomatics,
Im Neuenheimer Feld 410, 69120 Heidelberg, Germany

²University of Zurich, Department of Psychology,
Binzmühlestrasse 14 / Box 19, 8050 Zürich, Switzerland

³Schön Klinik Roseneck,
Am Roseneck 6, 83209 Prien am Chiemsee, Germany

⁴Institute of Clinical Psychology, Hospital Stuttgart,
Tunzhoferstraße 14-16, 70191 Stuttgart, Germany

⁵University of Heidelberg, Centre for Psychological Psychotherapy,
Hauptstrasse 47-51, 69117 Heidelberg, Germany

Corresponding author:

Timo Brockmeyer

Phone: +49 6221 5637153, E-Mail: timo.brockmeyer@med.uni-heidelberg.de

Abstract:

Impaired mood regulation has been considered to be a vulnerability factor for depression. However, there is a lack of studies specifically testing whether a) negative mood regulation (NMR) expectancies and b) emotion avoidance (EA) are associated with the risk for clinical depression. Therefore, the present study investigated these two specific facets of emotion processing in 20 formerly-depressed individuals (FD) and 20 never-depressed individuals (ND). As expected, FD reported lower NMR expectancies and stronger EA as compared to ND, suggesting that these two variables are associated with depression vulnerability. Furthermore, NMR expectancies were negatively associated with EA, indicating that individuals with lower confidence in their negative mood regulation abilities are concurrently characterized by a stronger avoidance of emotional experience. These findings strengthen hypotheses of specific emotion processing deficits in depression vulnerability.

1. Introduction

Persistent mood disturbances are the predominant feature of major depressive disorder (APA, 1994). Several authors have suggested that these disturbances may result from impaired mood regulation abilities (Joormann & D'Avanzato, 2010). Thus, research on individual differences regarding mood regulation has the potential to contribute to the understanding of depression and depression vulnerability.

Negative mood regulation (NMR) expectancies are defined as “the expectancy that some behavior or cognition will alleviate a negative mood state” (Catanzaro & Mearns, 1990, p. 546). According to response expectancy theory, individuals with strong confidence in their ability to regulate negative mood should be less prone to symptoms of depression (Kirsch, 1985). In line with this hypothesis, previous research with non-clinical samples has demonstrated negative associations between NMR expectancies and depressive symptoms both concurrently (Catanzaro & Mearns, 1990) and prospectively (Davis, Andresen, Trosko, Massman, & Lovejoy, 2005). Furthermore, depressed patients were found to have lower NMR expectancies than healthy controls (Backenstrass et al., 2010; Pfeiffer, Kaemmerer, Mearns, Catanzaro, & Backenstrass, 2011). This suggests that NMR expectancies may be an important variable in the etiology of clinical depression. However, at the moment there is no study that tested this hypothesis in individuals at risk for depression.

Previous research has proposed that individuals with stronger confidence in their ability to regulate negative mood may be less likely to avoid experiences of negative mood (Catanzaro & Mearns, 1990). Correspondingly, not only NMR expectancies but also emotion avoidance (EA) has been hypothesized to be involved in the etiology of clinical depression (Campbell-Sills, Barlow, Brown, & Hofmann, 2006). Indeed, emotional non-acceptance, a construct closely related to EA (Kashdan, Barrios, Forsyth, & Steger, 2006), was found associated with higher levels of depressive symptoms after life stress (Shallcross, Troy, Boland, & Mauss, 2010). In addition, patients with anxiety and depression rated their own

emotions to be less acceptable after a negative mood induction than healthy controls did (Campbell-Sills, et al., 2006). Similarly, recovered depressed individuals have been found to report stronger emotional non-acceptance as well as stronger difficulties in emotion regulation than never-depressed individuals (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008). Since individuals who have experienced previous depressive episodes are at high risk for future episodes, such examinations of recovered depressed subjects may provide insight to factors involved in depression vulnerability (Just, Abramson, & Alloy, 2001). However, previous studies on vulnerability factors for depression did not specifically address EA. Emotional non-acceptance, although closely related to EA, rather taps "negative secondary emotional responses to one's negative emotions" (Gratz & Roemer, 2004, p. 47) whereas EA presents as a general motivation to avoid the experience of emotions (Maio & Esses, 2001).

At the moment there are no studies investigating deficits concerning NMR expectancies and EA in subjects who are at increased risk for clinical depression. To overcome this lack, the present study tested a community-based sample of individuals with versus without a history of depression. It was expected that formerly-depressed individuals report lower NMR expectancies (Hypothesis 1) as well as stronger EA (Hypothesis 2) than never-depressed individuals. Furthermore, NMR expectancies were predicted to be negatively associated with EA (Hypothesis 3).

2. Method

2.1 Participants

The subjects sample consisted of 20 never-depressed individuals (ND) and 20 formerly-depressed individuals (FD). Participants were recruited via advertisements in the local media. They were only included if they did not suffer from any current axis-I disorder according to the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 1994). Additional exclusion criteria for the ND group were any *DSM-IV* axis-I disorder in the past. Exclusion

criteria for the FD group were a life-time diagnosis of a manic episode, psychosis, posttraumatic stress disorder, or borderline personality disorder according to *DSM-IV*. Participants were included to the FD group if they had a history of at least one currently remitted major depressive episode according to *DSM-IV*. Following the consensus recommendations (Frank, Prien, Jarrett, & Keller, 1991), current remission from depression was defined as the subject reporting minimal symptoms for a minimum of 12 weeks which means that the subject did no longer meet diagnostic criteria for a major depressive episode, and scored less than or equal to 10 on the Hamilton Depression Rating Scale (Hamilton, 1960). In line with corresponding recommendations (Dozois, Dobson, & Ahnberg, 1998) FD were also excluded if they scored higher than or equal to 12 on the Beck Depression Inventory-II (Kühner, Bürger, Keller, & Hautzinger, 2007). Written informed consent was obtained from all subjects before admission to the study. All subjects received €20 for their participation in the study. The study protocol had been approved by an ethics committee of the local university.

2. Measures

2.1 Assessment of depressive symptoms

In order to assess current and past episodes of mental disorders, the German version of the Structured Clinical Interview for *DSM-IV* (Wittchen, Zaudig, & Fydrich, 1997) was conducted by the first author who was specifically trained for SCID administration and had more than 3 years of experience using the SCID. The SCID has demonstrated good reliability in previous studies (e.g., Williams et al., 1992).

The Beck Depression Inventory-II (BDI-II; Kühner, et al., 2007) and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) were used to assess current levels of

depressive symptoms. The BDI-II is a self-report measure of depression severity that consists of 21 items. Total scale scores range from 0 to 63 (higher scores indicate more depressive symptoms). The reliability and validity of the BDI-II have been well established in clinical and non-clinical samples (Kühner, et al., 2007). The HRSD is a clinician-administered measure of depression severity that consists of 17 items. Total scores range from 0 to 52 (higher scores indicate more severe depression). The HRSD is widely used by both researchers and clinicians (Bagby, Ryder, Schuller, & Marshall, 2004).

2.2 Assessment of current mood

The Positive and Negative Affect Schedule (PANAS; Krohne, Egloff, Kohlmann, & Tausch, 1996) was used to measure current negative mood. The negative affect scale of the PANAS consists of 10 items. Empirical evidence supports the reliability and validity of this measure (Krohne, et al., 1996).

2.3 Measures of EA and NMR expectancies

The Emotion Avoidance Subscale of the Need for Affect Scale (NAS; Appel, 2008; Maio & Esses, 2001) was used to assess emotion avoidance. The scale consists of 13 items. For the purpose of this study, the poles of the seven-point Likert scale were changed from -3 and +3 to 0 and 7. Higher scores indicate stronger emotion avoidance. Previous research supported the reliability and validity of the NAS (Appel, 2008; Maio & Esses, 2001).

The 15-item short form of the Generalized Expectancies for Negative Mood Regulation Scale (NMR-SF; Pfeiffer, et al., 2011) was used to assess NMR expectancies. Higher scores indicate higher levels of expected negative mood regulation ability. The validity and the reliability of the NMR-SF have been shown to be adequate (Pfeiffer, et al., 2011).

2.4 Procedure

After participants had given informed consent, the SCID interview was conducted. Next, participants completed the BDI-II, PANAS, NMR-SF, and the NAS.

2.5 Data analyses

FD and ND were compared using t-tests and univariate analyses of covariance (ANCOVA). Since two dependent variables were tested at the same time, the significance level of $\alpha = .05$ was corrected using the Bonferroni procedure. Pearson correlation coefficients were computed to test the associations between NMR-SF and NAS scores.

3. Results

3.1 Sample characteristics

Table 1 summarizes demographic and clinical characteristics of the sample. The groups did not differ regarding age and gender ($ps > .10$). FD reported significantly more depressive symptoms than ND, $t(38) = 2.53, p < .05$. However, the mean BDI-II score of FD was clearly below the recommended cut-offs for the classification of individuals as dysphoric or depressed (Dozois, et al., 1998). Nevertheless, BDI-II scores were used as a covariate in any further analyses. There were no group differences regarding current negative mood as assessed by the PANAS, $ps > .06$.

--- Table 1 about here ---

3.2 Group differences regarding NMR expectancies and EA

To test the hypotheses of lower NMR expectancies (1) and stronger EA (2) in FD, two separate ANCOVAs were conducted, one with NAS scores and one with NMR-SF scores as the dependent variable. In both analyses, group was used as the independent variable. BDI-II scores were used as a covariate. Results are displayed in Table 2. ANCOVA results revealed that FD reported lower NMR expectancies as well as stronger EA than ND.

--- Table 2 about here ---

3.3 Association between NMR expectancies and EA

Pearson correlation coefficients were computed to test the third hypothesis. NMR expectancies were negatively correlated with EA, $r = -.61, p < .001$. This association was highly significant, even after controlling for BDI-II scores in both variables $r_{\text{partial}} = -.53, p < .001$.

4. Discussion

The present study is the first to investigate NMR expectancies and EA in a community based sample of FD and ND subjects. Consistent with the first hypothesis, FD individuals reported lower NMR expectancies than ND individuals, irrespective of current depressive symptoms. This finding adds further evidence to previous studies which observed that low NMR expectancies predicted higher depressive symptoms (Davis et al., 2005; Catanzaro & Mearns, 1990; Pfeiffer et al., 2011).

Furthermore, the results are in line with the second hypothesis: FD reported stronger EA than ND. This finding extends results from previous studies which reported reduced emotional acceptance in currently depressed individuals (Campbell-Sills, et al., 2006). Furthermore, this result confirms previous studies which used student samples to show that emotion regulation difficulties were increased in FD as compared to ND (Ehring, et al., 2008). The present study extends those findings by specifically assessing EA and by using a community-based sample.

In line with the third hypothesis, NMR expectancies were negatively associated with EA, even when controlling for depressive symptoms. This finding supports the notion that individuals with a stronger belief that they can regulate negative mood may strive to avoid negative mood to a lesser degree (Catanzaro & Mearns, 1990). Correspondingly, high avoidant coping has been found to be associated with low NMR expectancies in students (Catanzaro & Greenwood, 1994). However, the reverse may be true as well, since permanent avoidance of negative emotions may foreclose an adequate development of emotion regulation skills.

These present findings underline the potential relevance of training adaptive mood regulation skills to reduce the risk for depression (Feldman, Harley, Kerrigan, Jacobo, & Fava, 2009). An overly strong EA in depressed patients may further be targeted explicitly by guided exposure to previously avoided emotional material. Indeed, in a trial on Exposure-Based Cognitive Therapy for Depression, increases in mindfulness were associated with decreases in avoidance, rumination and depressive symptoms (Kumar, Feldman, & Hayes, 2008).

The present study has some limitations. First, the present study used a cross-sectional design. Rather than being vulnerability factors, the observed differences between FD and ND may represent a scar of past depressive episodes (Just, et al., 2001). The experience of a depressive episode may lead to heightened alertness for and avoidance of negative mood.

Likewise, beliefs of not being able to adequately regulate negative mood may result from actually failed attempts to improve mood. Therefore, future studies should use prospective longitudinal designs to investigate whether NMR expectancies and EA appear as precursors or as consequences of depressive episodes. Second, the presented results are based on self-report measures, which are sensitive to memory biases and demand characteristics. However, it has been demonstrated in previous studies that the NAS and the NMR scale were not affected by social desirability (Backenstrass, et al., 2010; Maio & Esses, 2001). Nevertheless, future studies may profit from using experimental designs and more objective measures (e.g., behavioral and cognitive performance tasks) to investigate the roles of EA and NMR expectancies as well as their interplay in depression vulnerability. Third, the sample size of the current study was relatively small. Thus, the present findings should be regarded as preliminary. Future studies need to replicate the present study in order to confirm its conclusions. However, the effect sizes underlying the present findings were large and thus emphasize the relevance of the present findings.

Notwithstanding these limitations, the findings of the present study, although preliminary, underline the importance of NMR expectancies and EA as specific facets of emotion processing in depression vulnerability.

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Table 1 *Sample characteristics*

Group	<i>n</i>	Age (years)		Number of previous depressive episodes		% currently on psychotropic medication	% currently in psychotherapy	BDI-II		
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>			<i>M</i>	<i>SD</i>	
Caucasian										
Formerly-depressed	16	4	56	12	4 _a	4.12	40	25	6.00 _a	4.33
Never-depressed	15	5	48	17	0	0	0	0	3.05	2.91

Note. _a significantly different from group of never-depressed individuals at $p < .05$

Table 2 Group differences regarding negative mood regulation expectancies and emotion avoidance

	Never-depressed ($n = 20$) $M (SD)$	Formerly-depressed ($n = 20$) $M (SD)$	$F(1, 37)$	p	d
NMR-SF	61.20 (7.98)	51.65 (7.73)	9.02 ^a	< .01	1.23
NAS	25.25 (9.96)	40.75 (16.43)	7.53 ^a	< .01	1.14

d = Cohen's estimate of effect size; NMR-SF, Generalized Expectancies for Negative Mood Regulation Scale Short Form; NAS, Need for Affect Scale, Emotion Avoidance Subscale; ^a significant after Bonferroni correction.

Mood Regulation and Cognitive Reactivity in Depression Vulnerability

Timo Brockmeyer^{1,6*}, Nils Pfeiffer^{2*}, Martin Grosse Holtforth³, Johannes Zimmermann⁴, Annette Kämmerer⁵, Hans-Christoph Friederich¹, & Hinrich Bents⁶

¹Centre for Psychosocial Medicine, University Hospital Heidelberg,
Department of General Internal Medicine and Psychosomatics,
Im Neuenheimer Feld 410, 69120 Heidelberg, Germany

²Schön Klinik Roseneck,
Am Roseneck 6, 83209 Prien am Chiemsee, Germany

³University of Zurich, Department of Psychology,
Binzmühlestrasse 14 / Box 19, 8050 Zürich, Switzerland

⁴University of Kassel, Department of Psychology,
Holländische Straße 36-38, 34127 Kassel, Germany

⁵University of Heidelberg, Department of Psychology,
Hauptstrasse 47-51, 69117 Heidelberg, Germany

⁶University of Heidelberg, Centre for Psychological Psychotherapy,
Hauptstrasse 47-51, 69117 Heidelberg, Germany

The first two authors contributed equally to the study.

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Corresponding author:

Timo Brockmeyer

Centre for Psychosocial Medicine, University Hospital Heidelberg,
Department of General Internal Medicine and Psychosomatics,

Im Neuenheimer Feld 410, 69120 Heidelberg, Germany

Phone: +49 6221 5637153; E-mail: timo.brockmeyer@med.uni-heidelberg.de

Abstract

There is substantial evidence supporting the hypothesis that cognitive reactivity is an important variable in the etiology of depression. However, there is a lack of studies examining possible mechanisms that underlie cognitive reactivity. The present study tested whether two specific mood regulation processes differentially appear in vulnerable and non-vulnerable individuals, and whether they can account for differences in cognitive reactivity. In a cross-sectional experimental design, 20 formerly-depressed individuals (FD) were compared with 20 never-depressed individuals (ND). In an autobiographical memory task both groups differed concerning the use of positively and negatively toned emotion words: FD retrieved fewer positive emotion words than ND in the second phase of this task. Furthermore, FD with a high cognitive reactivity retrieved more negatively toned emotion words. In the ND group there was a different pattern: Subjects with a high cognitive reactivity retrieved less positively toned emotion words. Two different cognitive processes seem to account for cognitive reactivity in individuals who are at high versus low risk for depression.

Keywords: Depression, vulnerability, emotion regulation, mood regulation, cognitive reactivity

Mood Regulation and Cognitive Reactivity in Depression Vulnerability

According to cognitive models of depression, cognitive structures in the form of negatively toned representations of the self, the world, and the future play a crucial role in the development, maintenance, and recurrence of depression (Abramson, Metalsky, & Alloy, 1989; Beck, 1967; Kovacs & Beck, 1978). Negative life events have been found to trigger depression by activating these cognitive structures or schemas. For example, individuals are more likely to develop depressive symptoms if they interpret negative events in terms of their own inadequacies and inferiority (Ingram, Miranda, & Segal, 1998).

Beck's cognitive model of depression suggests that the depressogenic schemas, evolved by interactions in early development, lay dormant in the non-depressed state (Beck, 1967; Kovacs & Beck, 1978). This is in line with several studies revealing apparent negative cognitions to be rather a correlate than a risk factor for depressive symptoms (Gotlib & Cane, 1987; Hollon, Kendall, & Lumry, 1986; Silverman, Silverman, & Eardley, 1984; Wilkinson & Blackburn, 1981). According to Beck's model these depressogenic schemas are activated by negative life events that are similar to the events that led to their development. However, Segal and Ingram (1994) refined this early model by describing two specific ways of schema activation: First, a schema can become activated directly if a perceived stimulus corresponds to the schema content. Second, a schema can become activated indirectly by the activation of an associated schema. This latter mechanism refers to the idea that once a schema is activated, this activation spreads to associated schemas (Bower, 1981; Ingram, 1984). Moreover, Teasdale (1988) pointed out that negative mood itself can act as a stressor that interacts with latent depressogenic schemas to cause depressed mood. According to these models, individuals with and without latent depressogenic schemas should be comparable with regard to their level of negative thinking in the absence of schema

activation. In contrast, interindividual differences in negative thinking should appear when the cognitive schemas are activated, for instance by sad mood. A change of depressive thinking in response to sad mood is called cognitive reactivity (Scher, Ingram, & Segal, 2005; Segal, Gemar, & Williams, 1999).

Indeed, there is substantial evidence supporting Teasdale's (1988) hypothesis, that an increased cognitive reactivity is a risk factor for depression. A large number of studies reported that formerly depressed individuals responded with more negative thinking to a sad mood provocation as well as to naturally occurring sad mood compared to never-depressed individuals (Scher et al., 2005). Moreover, two studies observed that cognitive reactivity predicted depressive relapse prospectively (Segal et al. 1999, 2006).

However, several studies reported an unexpected *reversed* cognitive reactivity in low-risk groups, i.e. a *reduction* of depressive thinking after a negative mood induction (Beevers, Scott, McGeary, & McGeary, 2009; Gemar, Segal, Sagrati, & Kennedy, 2001; Miranda, Persons, & Byers, 1990; Segal et al., 2006). This contradicts the notion that negative mood merely activates a depressive schema: According to the activation hypothesis negative mood should either increase depressive thinking or leave it unchanged. A decrease in depressive thinking raises the question about which processes underlie cognitive reactivity.

There is evidence from studies in clinical psychology that speak in favor of the hypothesis that emotion regulation processes are linked to cognitive reactivity. Several studies revealed that increased emotion regulation difficulties are associated with depression as well as the risk for depression (Ehring, Fischer, Schnülle, Bösterling, & Tuschen-Caffier, 2008; Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010; Pfeiffer, Kaemmerer, Mearns, Catanzaro, & Backenstrass, 2011). Recently, heart rate variability (as an indicator of

physiological emotion regulation capacity) has been shown to be associated with cognitive reactivity (Beevers, Ellis, & Reid, 2010).

Studies from social psychology may help to shed more light on the processes underlying cognitive reactivity. It has been very well documented in the literature that under normal circumstances individuals exhibit a cognitive bias for mood-congruent information: People in a happy mood tend to retrieve thoughts and memories with a positive content whereas people in a sad mood tend to retrieve information with a negative content (Blaney, 1986; Bower, 1981; Fiedler, Nickel, Asbeck, & Pagel, 2003). However, this mechanism may be reversed in service of mood regulation. For instance, recalling positive autobiographical memories can serve to repair a sad mood state (Erber & Markunas, 2006; Erber, Wegner, & Therriault, 1996; Forgas, 1995; Forgas & Ciarrochi, 2002). More specifically, the use of mood incongruent cognitions for mood regulation seems to be initiated with a certain delay after a mood induction (Sedikides, 1994). Forgas and Ciarrochi (2002) used sentence and word completion tasks to record subjects' cognitions after a mood induction. They observed that subjects first completed the tasks using mood congruent words. But while working on the task subjects switched from mood congruence to mood incongruence.

Such use of mood incongruent cognitions for mood regulation seems to be disturbed in depression. Josephson, Singer, and Salovey (1996) induced negative mood in their subjects and observed that subjects consistently reported mood congruent autobiographical memories directly after the mood induction. But subsequent mood congruence depended on the level of depressive symptoms: Whereas subjects with low depression scores recalled mood incongruent memories, dysphoric subjects showed mood congruence and reported lower levels of mood repair (Josephson et al., 1996). Correspondingly, Joormann and colleagues found that dysphoric as well as currently and formerly depressed individuals had

reduced access to positive autobiographical memories after a sad mood induction and reported less mood repair (Joormann & Siemer, 2004; Joormann, Siemer, & Gotlib, 2007).

These findings offer a possible explanation for the puzzling findings of a reversed cognitive reactivity in individuals at low risk for depression: It may be that non-vulnerable individuals, contrary to vulnerable individuals, are able to retrieve positive (i.e. mood-incongruent) thoughts and memories to regulate their negative mood state and thereby end up with more positive thinking.

The second possible answer to the question of how cognitive reactivity and emotion regulation may be associated refers to the ability to disengage from processing negative information. It has been demonstrated that a reduced ability to disengage one's attention from negative stimuli is associated with increased negative emotional reactivity to those stimuli (Compton, 2000; Ellenbogen, Schwartzman, Stewart, & Walker, 2002). Applying this approach to clinical depression, eye-tracking studies revealed that depressed individuals spent more time looking at negative stimuli than controls (Caseras, Garner, Bradley, & Mogg, 2007; Eizenman et al., 2003; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005). These findings offer another explanation for the question as to which processes may underlie cognitive reactivity. Individuals at increased risk for depression may be unable to disengage from negative thoughts and memories that are triggered by negative mood. In consequence, these subjects may end up with ongoing negative thinking.

These empirical findings give rise to the following hypotheses. (1) Replicating previous findings, subjects who are at high risk for depression should exhibit an increased cognitive reactivity (Scher, et al. 2005). (2) In correspondence to earlier studies (Forgas & Ciarrochi, 2002; Josephson et al., 1996) subjects who are at high vs. low risk for depression should not differ in terms of retrieving positive and negative emotion words in an autobiographical memory task immediately after a negative mood induction. (3) However,

differences should appear later in information processing (Forgas & Ciarrochi, 2002; Josephson et al., 1996): With a certain delay after the mood induction the two groups should differ concerning the retrieval of positive and negative emotion words as an indicator of mood regulation, i.e., low-risk subjects should retrieve more positive emotion words (i.e. mood-incongruent information) whereas high-risk subjects should show a bias for negative emotion words (i.e. mood-congruent information). (4) As suggested by previous results, the proportion of positive emotion words in the second phase should be *negatively* associated with cognitive reactivity (Eizenman et al., 2003; Koster et al., 2005). Conversely, the proportion of negative emotion words in the second phase should be *positively* associated with cognitive reactivity (Joormann & Siemer, 2004; Joormann et al., 2007). The following study tests these hypotheses in a cross-sectional design with samples of formerly and never-depressed subjects (Just, Abramson, & Alloy, 2001).

Method

Participants

Participants were recruited via advertisements in the media. The sample consisted of 20 never-depressed individuals (ND) and 20 formerly-depressed individuals (FD). All participants were Caucasian and between 19 and 69 years of age. Subjects were neither admitted to the ND group nor to the FD group if they suffered from any current DSM-IV axis-I diagnosis (APA, 1994). Furthermore, subjects were only included to the ND group if they had not suffered from any DSM-IV axis-I disorder in the past. To be admitted to the FD group, subjects had to have suffered from at least one currently remitted major depressive episode according to DSM-IV. Following the consensus recommendations (Frank, Prien, Jarrett, & Keller, 1991), remission from depression was defined as the patient reporting minimal symptoms for a minimum of 12 weeks, no longer meeting diagnostic criteria for a major depressive episode, and a score of less than or equal to 10 on the Hamilton Depression

Rating Scale-17 (Hamilton, 1960). Additionally, FD were excluded if they scored higher than or equal to 12 on the Beck Depression Inventory-II (Beck, Steer, & Brown, 1996; Kühner, Bürger, Keller, & Hautzinger, 2007), which is in line with corresponding recommendations (Dozois, Dobson, & Ahnberg, 1998). Further exclusion criteria for FD subjects were a life-time diagnosis of a manic episode, psychosis, posttraumatic stress disorder, or borderline personality disorder according to DSM-IV. DSM-IV diagnoses were obtained using the German version of the Structural Clinical Interview for DSM-IV (SCID; Wittchen, Zaudig, & Fydrich, 1997). All subjects received €20 for their participation in the study. Written informed consent was obtained from all subjects before admission to the study. The study protocol had been approved by an ethics committee of the local university.

Measures

Depressive symptoms. The Beck Depression Inventory-II (BDI-II; Beck et al., 1996; Kühner et al., 2007) and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) were used to assess depressive symptoms. The BDI-II is a self-report measure consisting of 21 items describing symptoms of depression. Total scale scores range from 0 to 63, with a higher score indicating more depressive symptoms. The reliability and validity of the BDI-II have been well established (Beck et al., 1996; Kühner et al., 2007). The HRSD is a clinician-administered measure consisting of 17 items to assess the presence and severity of depressive symptoms. Total scores range from 0 to 52, with higher scores indicating more severe depression. The HRSD is widely used by both researchers and clinicians (Bagby, Ryder, Schuller, & Marshall, 2004).

Current mood. The Positive and Negative Affect Schedule (PANAS; Krohne, Egloff, Kohlmann, & Tausch, 1996; Watson, Clark, & Tellegen, 1988) was used to assess negative mood before and after the mood induction procedure. The negative affect scale of the

PANAS is composed of 10 items. Empirical evidence supports the reliability and validity of the PANAS (Krohne et al., 1996).

Cognitive reactivity. The Scrambled Sentences Test (SST; Wenzlaff & Bates, 1998) was used to assess participants' bias to interpret ambiguous information. Participants received a list of scrambled sentences (e.g., "bright the very dismal looks future") and were instructed to write a number above five of the six words of each scrambled sentence to produce a grammatically correct sentence in either negative ("the future looks very dismal") or positive ("the future looks very bright") ways. Participants were presented with two blocks of 20 sentences, one prior to the mood induction and one after the mood induction. The order of administration of the two SST forms was counterbalanced across subjects. Participants were given 4 minutes to complete each block. They were instructed to complete as many of the sentences as possible during this time. The SST was scored by calculating the ratio of negative sentences over completed sentences. The change from the first SST form (prior to the mood induction) to the second SST form (after the mood induction) was used as an index of cognitive reactivity. In correspondence to Segal et al. (2006), residualized change scores were computed using a simple linear regression model. Post-SST scores were predicted by pre-SST scores, and the standardized residuals for each case were saved from this model, resulting in an unconfounded measure of SST change (Cohen, Cohen, West, & Aiken, 2003). The SST has been shown to be an appropriate and methodologically sound measure of a negative interpretive bias (Rude, Wenzlaff, Gibbs, Vane, & Whitney, 2002; Watkins & Moulds, 2007).

Mood regulation. In order to assess participants' mood regulation, participants were instructed to write down their memories about a sad autobiographical event during the negative mood induction procedure. The report of each participant was split into two equal halves according to word number and analyzed with the text analysis program TAS/C. This

software was originally designed for applications in the field of psychotherapy research (Mergenthaler, 1993, 1996). Using an emotion dictionary, the TAS/C was used to calculate the proportion of words with a positive and negative emotional tone for the first and the second half of the memory task (Mergenthaler, 1993).

Procedure

Prior to the experimental procedure, subjects were interviewed with the SCID and the HRSD. Furthermore, they completed the BDI-II. Thereafter, participants completed the PANAS and then the first form of the SST. Subsequently, subjects participated in the negative mood induction procedure: For 8 minutes participants listened to a sad piece of music and wrote down details about an autobiographical event when they felt sad. The music was the orchestral introduction by Prokofiev entitled *Russia under the Mongolian Yoke* from the movie *Alexander Nevsky*. The segment played to participants was re-mastered at half speed and presented through earphones. This type of mood induction has been found to effectively induce a negative mood state in previous studies (Gemar et al., 2001; Ingram, Bernet, & McLaughlin, 1994; Martin, 1990; Segal et al., 2006). After the negative mood induction, participants completed the PANAS again and the second version of the SST.

Results

Demographic and Clinical Characteristics of the Sample

Participants in the ND group had a mean age of 48 years ($SD = 17$ years), and most were female ($n = 15$; 75%). The mean age of participants in the FD group was 56 years ($SD = 12$), and the majority of FD subjects was female ($n = 16$; 80%). The groups did not differ significantly concerning age and sex ($ps > .10$). All participants were Caucasian. Mean scores on the BDI-II were 3.05 ($SD = 2.91$) for ND and 6.00 ($SD = 4.33$) for FD. This difference reached statistical significance, $t(38) = 2.53, p < .05$. However, the mean score of FD was far under recommended cut-offs for the classification of individuals as dysphoric or

depressed (Dozois et al., 1998). In any further analyses interindividual differences in depressive symptoms were controlled for by using BDI-II scores as a covariate.

On average, subjects in the FD group had suffered four previous depressive episodes ($SD = 4.12$). In the FD group eight subjects (40%) currently used psychotropic medication, and five participants (25%) were currently in psychotherapeutic treatment. There were no significant differences between both groups regarding baseline negative mood as measured by the PANAS, and baseline negative thinking as measured by the SST prior to the mood induction, $ps > .06$.

Manipulation Check

Mood could be effectively manipulated by the negative mood induction procedure. There was a significant main effect of time (pre-post mood induction) across groups as measured by the negative affect scale of the PANAS, $F(1, 40) = 42.08, p < .001$. Moreover, neither the effect of the group variable nor the effect of the interaction between group and time was statistically significant, $ps > .14$. Thus, the mood manipulation was equally effective for both groups, which is essential for an adequate mood induction (Scher et al., 2005).

Group Differences Regarding Mood Regulation and Cognitive Reactivity

To test the hypotheses regarding group differences in negative mood regulation and cognitive reactivity, several analyses of covariance (ANCOVAs) were conducted. Results are displayed in Table 1. To test the first hypothesis (FD exhibit an increased cognitive reactivity compared to ND), a first ANCOVA was conducted with SST change score as the dependent variable and group as the independent variable. To control for possible confounds, BDI-II scores as well as the administration order of the two SST forms were used as covariates. ANCOVA results revealed that FD showed a greater cognitive reactivity as compared to ND.

To test the second hypothesis (FD and ND do not differ regarding retrieval of emotion words in the first phase of the autobiographical memory task), two further ANCOVAs were conducted, one with the proportion of positively toned emotion words, and one with the proportion of negatively toned emotion words in the first half of the autobiographical memory task as the dependent variable. In both analyses group was the independent variable and BDI-II scores were entered as a covariate. ANCOVA results revealed that FD and ND did not differ regarding the retrieval of positively and negatively toned emotion words in the first half of the autobiographical memory task.

To test the third hypothesis (FD retrieve more negatively toned and less positively toned emotion words in the second half of the autobiographical memory task than ND), another two ANCOVAs were conducted, one with proportion of positively toned emotion words, and one with proportion of negatively toned emotion words in the second half of the autobiographical memory task as the dependent variable. Again, group was the independent variable, and BDI-II scores were entered as covariate. ANCOVA results revealed that FD retrieved fewer positively toned emotion words in the second half as than ND did. However, FD did not differ from ND regarding negatively toned emotion words retrieved in the second half of the task.

Associations Between Mood Regulation and Cognitive Reactivity

To test the fourth hypothesis regarding associations between retrieval of emotion words and cognitive reactivity, partial correlations were computed independently for each group. To control for possibly confounding variables, BDI-II scores, baseline mood, and SST administration order were partialled out. Correlations are displayed in Table 2. In both groups cognitive reactivity correlated neither with the proportion of positively toned emotion words nor with the proportion of negatively toned emotion words retrieved in the first phase of the autobiographical memory task. In contrast, the number of positively toned emotion

words retrieved in the second half of the task was negatively associated with cognitive reactivity in ND. In FD, the proportion of negatively toned emotion words retrieved in the second half was positively associated with cognitive reactivity.

To test whether these associations between retrieval of emotion words and cognitive reactivity were significantly different in both groups, several hierarchical multiple regression analyses were conducted with the SST residualized change score as the dependent variable. The first regression analysis tested whether groups differed regarding the association between the proportion of positively toned emotion words retrieved in the second half of the task and cognitive reactivity. In a first step, BDI-II scores and SST administration order were entered as predictors into the equation. In the second step, proportion of positively toned emotion words in the second half of the task and group were added to the model. Finally, the interaction between group and the proportion of positively toned emotion words was entered into the equation. Results are displayed in Table 3. Neither group nor the proportion of positively toned emotion words were significant predictors for cognitive reactivity. However, the effect of the interaction between both variables was significant.

To further test whether there was also a differential association between retrieval of negatively toned emotion words in the second half of the task and cognitive reactivity in the two groups, a corresponding multiple hierarchical regression analysis was conducted, again with cognitive reactivity (SST change score) as the dependent variable. Again, BDI-II score and SST administration order were entered in the first step. This time, group and proportion of negatively toned emotion words retrieved in the second half of the task were entered in the second step, and the interaction between group and the proportion of negatively toned emotion words in the third step. Results are displayed in Table 4. As in the first regression, neither group nor the proportion of negatively toned emotion words was a significant predictor for cognitive reactivity. However, their interaction was significant. It needs to be

mentioned that in both regression analyses the order of administration of the two SST forms as well as baseline mood were also significant predictors of cognitive reactivity.

Three corresponding multiple regression analyses were conducted to test the associations between cognitive reactivity and the proportion of negatively and positively toned emotion words in the first phase of the autobiographical memory task. The results indicated that the single associations did not differ across the two groups. To conclude, the associations between cognitive reactivity and the proportion of negatively and positively toned emotion words were statistically significant between both groups only in the second half of the autobiographical memory task.

Discussion

Consistent with the first hypothesis, FD showed a higher cognitive reactivity as compared to ND. Thus, the present study replicated findings from earlier studies (Scher et al., 2005). Consistent with the second hypothesis, ND and FD did not differ regarding the proportion of positive and negative emotion words retrieved in the first half of the autobiographical memory task. This is in line with findings from previous studies showing that dysphoric and non-dysphoric individuals did not differ in choosing a negative autobiographical memory immediately after a sad mood induction (Josephson et al., 1996).

However, as predicted in the third hypothesis, FD retrieved fewer positive emotion words in the second half of the task. Thereby, the results of the present study suggest that depression-vulnerable individuals suffer from a deficit in retrieving (mood-incongruent) positive information in a sad mood state. This finding is in line with the results of Josephson et al. (1996). However, the current results extend the previous findings by analyzing the responses of subjects who had formerly suffered from clinical depression instead of students who had experienced an elevated BDI score. Furthermore, the present study extends these

findings by using text analysis as a tool instead of a forced-choice paradigm for assessing memory processes.

The results of the present study diverge from the findings of Joormann and colleagues (Joormann & Siemer, 2004; Joormann et al., 2007). In their studies, dysphoric and non-dysphoric participants as well as depressed, formerly depressed, and controls did not differ in the number, specificity, and valence of autobiographical memories. These discrepancies could be due to the different measures applied. The computerized text analysis used in the present study may allow for a more fine-grained examination of cognitive processes than the forced-choice or rating procedures used by the mentioned studies.

Contrary to the third hypothesis, FD and ND did not differ regarding the number of negative emotion words retrieved in the second half of the task. Thus, it cannot be concluded that FD exhibit more difficulties in disengaging from negative emotion recall than ND. This is in contrast with findings from previous studies using eye-tracking measures (Caseras et al., 2007; Eizenman et al., 2003). In these studies, depressed participants focused their attention longer on negative stimuli than healthy controls did. These discrepant findings could be due to differences in the measures applied, however, further studies are needed to clarify these discrepancies.

Nevertheless, results of the present study lend support to the idea that disengagement from negative emotion recall is a relevant component of information processing in individuals who are at risk for depression: In correspondence to the fourth hypothesis, the number of negative emotion words retrieved in the second half of the task was positively correlated with cognitive reactivity in FD, indicating that FD who retrieved more negative emotion words in the second half of the task exhibited a higher cognitive reactivity. In the ND group a different pattern emerged: ND who retrieved less positive emotion words in the second half of the task exhibited a higher cognitive reactivity. These differential associations

between positive and negative emotion recall and cognitive reactivity constitute the most intriguing finding of the present study. This finding suggests that differential processes underlie cognitive reactivity in vulnerable and non-vulnerable individuals. For non-vulnerable individuals a lack of mood-incongruent memory retrieval may be responsible for subsequent negative thinking. For vulnerable individuals a deficit in the ability to disengage from negative stimuli may result in negative thinking.

The present finding regarding the association between the disengagement deficit and cognitive reactivity in FD is in line with findings from previous studies using eye-tracking procedures that demonstrated disengagement deficits in depressed patients as well as an association between this deficit and an increased emotional reactivity in healthy subjects (Caseras et al., 2007; Compton, 2000; Eizenman et al., 2003; Ellenbogen et al., 2002).

The present finding on the association between lack of mood-incongruent information retrieval and cognitive reactivity in ND is in line with previous findings from studies that demonstrated associations between lack of access to mood-incongruent memories and lack of mood repair in dysphoric and FD (Joormann & Siemer, 2004; Joormann et al., 2007; Josephson et al., 1996).

The present study has some limitations. First, the sample size was relatively small. However, the fact that several effects reached statistical significance in the predicted direction increases confidence that these findings can be replicated in larger samples. Second, the cross-sectional design of the study limits interpretations of the results in the way that differences between FD and ND may be due to scar effects of past depressive episodes rather than being attributable to depression vulnerability (Just et al., 2001). Future research on the assumed links between cognitive reactivity and mood regulation in the vulnerability for depression should use prospective longitudinal study designs and bigger sample sizes. A further limitation refers to the inequality of the two SST forms.

Notwithstanding, the present study also has some considerable strengths such as the absence of constricting, behavioral instructions (e.g., the instruction to reappraise or suppress emotions during an experimental procedure), and consequently the focus on naturally occurring, spontaneous processes of mood regulation. A further strength of the present study lies in the quantitative text analysis. Previous studies that examined autobiographical memories were mostly based on rating procedures and forced-choice paradigms only (Joormann & Siemer, 2004; Joormann et al., 2007; Josephson et al., 1996). Future studies may choose to extend the range of dependent variables further and use physiological measures to gain insight into the mechanisms underlying cognitive reactivity.

The present study encourages a more detailed investigation of cognitive processes involved in the regulation of emotions in the treatment of depression. Recently, treatment approaches with an increased focus on emotion regulation deficits have been successfully applied to depression (Brewin et al., 2009; Feldman, Harley, Kerrigan, Jacobo, & Fava, 2009; Grosse Holtforth et al., submitted; Hayes et al., 2007). In addition to these comprehensive treatment packages, it may be beneficial to implement a more direct training of basic cognitive processes of mood regulation. It has repeatedly been demonstrated that a training of attention shifting, for example using a dot probe task or mindfulness-based interventions, has the potential to reduce emotion dysregulation (Wadlinger & Isaacowitz, 2011). The present study stimulates further investigations into the role of such distinct cognitive mood regulation processes in the etiology and treatment of depression.

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Table 1 Group differences regarding cognitive reactivity and retrieval of emotion words in the autobiographical memory task

	Never-depressed (<i>n</i> = 20)	Formerly-depressed (<i>n</i> = 20)	Statistic
	<i>M</i> (<i>SD</i>)		
SST change	-0.35 (0.45)	0.32 (1.26)	$F(1, 36) = 4.29, p < .05$
Positive emotions 1 st half	0.03 (0.02)	0.04 (0.03)	$F(1, 37) = 1.26, ns$
Negative emotions 1 st half	0.05 (0.03)	0.04 (0.03)	$F(1, 37) = 0.75, ns$
Positive emotions 2 nd half	0.04 (0.02)	0.02 (0.02)	$F(1, 37) = 4.76, p < .05$
Negative emotions 2 nd half	0.06 (0.03)	0.05 (0.03)	$F(1, 37) = 0.22, ns$

Note. SST change standardized residual change score in the Scrambled Sentences Test (index of cognitive reactivity); Positive emotions 1st half proportion of positively toned emotion words in the 1st half of the autobiographical memory task; Negative emotions 1st half proportion of negatively toned emotion words in the 1st half of the autobiographical memory task; Positive emotions 2nd half proportion of positively toned emotion words in the 2nd half of the autobiographical memory task; Negative emotions 2nd half proportion of negatively toned emotion words in the 2nd half of the autobiographical memory task

Table 2 Partial correlations between mood regulation indicators and cognitive reactivity with BDI-

II scores, baseline mood, and SST administration order as covariates

	SST change	
	Never-depressed ($n = 20$)	Formerly-depressed ($n = 20$)
Positive emotions 1 st half	-.24	.22
Negative emotions 1 st half	.16	.39
Positive emotions 2 nd half	-.56*	.38
Negative emotions 2 nd half	-.01	.58*

Note. SST change standardized residual change score in the Scrambled Sentences Test (index of cognitive reactivity); Positive emotions 1st half proportion of positively toned emotion words in the 1st half of the autobiographical memory task; Negative emotions 1st half proportion of negatively toned emotion words in the 1st half of the autobiographical memory task; Positive emotions 2nd half proportion of positively toned emotion words in the 2nd half of the autobiographical memory task; Negative emotions 2nd half proportion of negatively toned emotion words in the 2nd half of the autobiographical memory task

* $p < .05$

Table 3 Differential associations between retrieval of positive emotion words and cognitive reactivity in formerly depressed and never depressed individuals

	<i>B</i>	<i>SE B</i>	<i>β</i>
Step 1			
BDI-II	0.01	0.04	.02
SST order	-0.66	0.28	-.34*
Baseline mood	0.14	0.06	.37*
Step 2			
Group	0.55	0.31	.28
Positive emotions	4.85	6.33	.11
Step 3			
Interaction Group * Positive emotions	26.61	12.16	.49*

$R^2 = .32$ for step 1; $\Delta R^2 = .06$ for step 2 (*ns*); $\Delta R^2 = .08$ for step 3 ($p < .05$). * $p < .05$

Note. *BDI-II* Beck Depression Inventory – II; *SST order* administration order of the two forms of the Scrambled Sentences Test; *Positive emotions* ratio of positive emotion words in the second half of the autobiographical memory task

Table 4 Differential associations between retrieval of negative emotion words and cognitive reactivity in formerly-depressed and never-depressed individuals

	<i>B</i>	<i>SE B</i>	β
Step 1			
BDI-II	0.01	0.04	.02
SST order	-0.66	0.28	-.34*
Baseline mood	0.14	0.06	.37*
Step 2			
Group	0.51	0.29	.26
Negative emotions	5.81	4.18	.19
Step 3			
Interaction Group * Negative emotions	18.66	7.69	.69*

$R^2 = .32$ for Step 1; $\Delta R^2 = .08$ for step 2 (*ns*); $\Delta R^2 = .09$ for step 3 ($p < .05$). * $p < .05$

Note. *BDI-II* Beck Depression Inventory – II; *SST order* administration order of the two forms of the Scrambled Sentences Test; *Negative emotions* ratio of negative emotion words in the second half of the autobiographical memory task