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Regular article

**The association between depressive symptoms and executive control impairments
in response to emotional and non-emotional information**

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Abstract

Depression has been linked with impaired executive control and specific impairments in inhibition of negative material. To date, only a few studies have examined the relationship between depressive symptoms and executive functions in response to emotional information. Using a new paradigm, the Affective Shift Task (AST), the present study examined if depressive symptoms in general, and rumination specifically, are related to impairments in inhibition and set shifting in response to emotional and non-emotional material. The main finding was that depressive symptoms in general were not related to inhibition. Set shifting impairments were only observed in moderate to severely depressed individuals. Interestingly, rumination was related to inhibition impairments, specifically when processing negative information, as well as impaired set shifting as reflected in a larger shift cost. These results are discussed in relation to cognitive views on vulnerability for depression.

Keywords: depression, rumination, executive control, inhibition, shifting

Introduction

Major depressive disorder (MDD) represents a mental health problem with a life time prevalence of around 16% (Kessler et al., 2003). Given the evidence that relapse rates remain very high despite the existing pharmacological and psychological treatments for depression (Gotlib, Kurtzman, & Blehar, 1997; Kessler, Chiu, Demler, & Walters, 2005), recent efforts have been directed to predict the onset as well as relapse of depression. Identification of the underlying vulnerability factors for the development, maintenance, and recurrence of depression is an important challenge with clinical relevance, informing treatment and prevention programs.

Cognitive theories have emphasized the role of information processing biases in the etiology and maintenance of depression. The presence and automatic reactivation of negative self-referent schemas, defined as organizational structures in memory developed based on one's own experiences (Beck, 1967, 1995), have an important influence on the way information is processed in depression. Schema-congruent negative information processing biases are proposed to play an important role regarding vulnerability for depression (Alloy et al., 2000; Clark, Beck, & Alford, 1999). These theoretical claims have instigated a wealth of research into the relationship between information processing impairments and depression.

Depression-related cognitive processing

Since the proposal of Beck's cognitive theory, a wealth of studies have examined the presence of emotionally-specific information-processing bias (Clark et al., 1999). Robust evidence has been observed for emotional-specific processing bias at the level of memory (Gotlib, Roberts, & Gilboa, 1996; Matt, Vazquez, & Campbell, 1992; Rusting, 1998) and attention (for a review see De Raedt & Koster, 2009). Despite these interesting findings, biases in emotion processing in depression have not been studied in relation to the basic cognitive mechanisms of executive control. Arguably, a better understanding of the relation

between specific aspects of executive control and biased emotion processing will improve our theoretical understanding of information processing impairments in depression. Toward this goal, the present investigation examined if (1) depressive symptoms in general and (2) rumination, a typical cognitive processing style observed in depression that remains active after remission (Roberts, Gilboa, & Gotlib, 1998), are related to executive control impairments in response to emotional and non-emotional material. We first present an overview of studies reported in the literature on depression and executive control.

Executive control impairments in depression

Growing neuropsychological evidence suggests that depression is associated with impairments in executive control. Recent neuroimaging studies have documented that depression is associated with reduced brain activity in areas including the anterior cingulate cortex (ACC) and dorsolateral prefrontal cortex (DLPFC) (Davidson, Pizzagalli, Nitschke, & Putnam, 2002). The executive control impairments may stem from the hypoactivation of these prefrontal areas which are thought to subserve cognitive control (Smith & Jonides, 1999). Depression is also characterized by sustained and prolonged amygdala activity in response to negative information (Surguladze et al., 2005; Taylor & Fragopanagos, 2005). It is assumed that reduced top-down control over negative information could be an underlying mechanism causing enhanced emotional reactivity and vulnerability for depression (Irwin et al., 2004; L  ppanen, 2006).

With regard to the specific cognitive operations related to executive control, factor analysis of tasks measuring facets of executive control revealed three executive functions that are moderately correlated with one another but are clearly separable: (1) monitoring and updating of working memory representations, (2) inhibition and (3) mental set shifting (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). The executive functions most frequently related to depression in the literature are inhibition and set shifting. *Cognitive*

inhibition refers to the ability to effectively inhibit the processing of previously relevant or irrelevant distracting information. The *set shifting* function concerns the ability to shift back and forth between multiple tasks, operations or mental sets (Monsell, 1996). It is still unclear which specific executive control functions are impaired in depression, inhibition and/or set shifting.

Depressive symptoms and inhibition impairments

A large amount of literature has investigated depression-related impairments in inhibition. The evidence for inhibition impairments when processing non-emotional information is limited and appears to be limited to severely depressed patients (e.g., Kaiser et al., 2003). However, there is increased evidence that depression is related to a valence-specific inhibition impairment (Joormann, Yoon, & Zetsche, 2007). Results from studies using the negative affective priming (NAP) paradigm indicated that depressed patients (Goeleven, De Raedt, Baert, & Koster, 2006) and dysphoric undergraduates (Joormann, 2004) are characterized by reduced inhibition of negative information. Research with formerly depressed individuals suggests that the valence specific inhibition impairment even persists after recovery from depression (Joormann, 2004; Joormann & Gotlib, 2007). Moreover, a study by Joormann, Talbot and Gotlib (2007) showed that never-depressed daughters of mothers who have experienced recurrent depressive episodes were also characterized by enhanced attention for negative information. These findings support the idea that reduced inhibition of negative material is a stable cognitive vulnerability factor for depression rather than a variable impairment associated with a depressed mood state.

Depressive symptoms and set shifting impairments

One of the most commonly used tests to assess set shifting ability is the Wisconsin Card Sorting Task (WCST) (WCST; Heaton, 1981; Heaton, Chelune, Talley, Kay, & Curtiss, 1993). The aim of the WCST is to determine what rule should be used to sort target cards to

match key cards that vary in stimulus dimensions. Feedback is given to participants about correct and incorrect matches and they must be able to adjust their performance when the rule unexpectedly changes. Participants exhibit impaired performance when making more perseverative errors, they persist in performing the task according to the old rule despite receiving feedback that their matches are incorrect.

On computerized non-emotional versions of this task, dysphoric undergraduates and depressed patients make more perseverative errors, suggesting a general shifting impairment (Harvey et al., 2004; Merriam, Thase, Haas, Keshavan, & Sweeney, 1999; Rogers et al., 2004). Given that a robust empirical literature indicates that the emotional nature of the stimuli affects the performance of individuals with depression, shifting has also been investigated in the presence of emotional material. For instance, Murphy et al. (1999) developed an affective shifting task that required subjects to respond to either positive or negative target words while inhibiting responses to words of the competing affective category and also to shift attention from one affective category to the other. Deveney and Deldin (2006) developed an emotional modification of the WCST in which emotional words were written on the target cards, but the emotional valence was irrelevant to successful performance on the task. The results of both studies (Murphy et al., 1999; Deveney & Deldin, 2006) indicate that depressed individuals show the largest impairment on set shifting when stimuli are negatively valenced. There is evidence to indicate that set shifting impairments are a stable vulnerability factor as a study found set shifting impairments in a remitted group of MDD-patients (Paelecke-Habermann, Pohl, & Lelow, 2005).

Rumination, vulnerability for depression, and inhibition impairments

Impairments in executive control could be related to typical information processing styles in depression. Depression is related to a range of cognitive distortions and alterations in thinking style. Of particular relevance is the tendency to ruminate, defined as “behaviors and

thoughts that focus one's attention on one's depressive symptoms and on the implications of these symptoms" (Nolen-Hoeksema, 1991, p 569). Rumination has been implicated in the etiology, maintenance and exacerbation of depressed mood states (Nolen-Hoeksema, Morrow, & Fredrickson, 1993). This inflexible thinking style appears to be stable beyond depressive episodes and increases vulnerability to depression (Roberts et al., 1998). In the literature, two different types of rumination are distinguished (Treyner, Gonzalez, & Nolen-Hoeksema, 2003). The first, reflective pondering, is a more adaptive form of rumination and reflects the degree to which individuals engage in cognitive problem solving to improve their mood. The second, depressive brooding, is a more maladaptive form of rumination and reflects the degree to which individuals passively focus on negative mood and problems. Depression is specifically characterized by high levels of brooding (Joormann, Dkane, & Gotlib, 2006).

To date, only few studies have investigated which specific executive mechanisms are associated with rumination. Attentional inflexibility may occur in ruminators because of inhibition and/or set shifting problems. Based on previous research, it has been proposed that rumination is caused by impaired cognitive inhibition (Hester & Garavan, 2005; Linville, 1996; Shapiro, 2002; Ursin, 2005; Watkins & Brown, 2002). However, concerning rumination and inhibition, the data are rather mixed. While some studies fail to find an association between rumination and inhibition (Goeleven et al., 2006), other studies find that depressed ruminators show difficulties in inhibiting negative information (Joormann, 2006). These findings suggest that reduced inhibition might be at the basis of enhanced elaboration of negative material or rumination.

Rumination and set shifting impairments

In comparison with the inhibition function, the set shifting function is even less investigated in relation to rumination. Research findings show that ruminators make more perseverative errors on the Wisconsin Card Sorting Task (WCST) relative to controls,

indicating a general set shifting impairment (Davis & Nolen-Hoeksema, 2000). To date, research using neuropsychological tests of set shifting in ruminators has only used non-emotional stimuli and consequently cannot inform about whether set shifting is impaired in the context of emotional material.

A recent study by Whitmer and Banich (2007) investigated inhibition as well as set shifting related to different types of rumination. In this study a task switching paradigm based on a design developed by Mayr and Keele (2000) was used to determine if executive control impairments in ruminators were associated with inhibition or set shifting impairments in the context of non-emotional information. It was found that the two types of rumination, reflective pondering and brooding, were associated with different executive dysfunctions. Brooding was specifically associated with a impairment in inhibition.

Taken together, to date only a few studies have tried to assess the different executive functions, inhibition, and set shifting in the context of depression. In addition, studies in the past mainly examined executive functions in relation to depressive symptoms in general, with only limited research investigating the effect of rumination (Christopher & MacDonald, 2005). Finally, only a few studies have examined the influence of depressive symptoms on executive functions in response to emotional as well as non-emotional material in one single design. This is an important limitation as previous research indicates that emotion processing is more severely impaired than information processing in general (Joormann et al., 2007b).

Aims of the present study

The aims of the present study were two-fold. First, we investigated whether individuals with depressive symptoms show impairments in inhibition as well as set shifting in response to emotional and non-emotional material. A second important aim was to investigate whether rumination, a specific core cognitive processing style observed in depression, is related to impairments in inhibition as well as set shifting in response to

emotional and non-emotional material. To our knowledge, our study is the first to examine these aims in a single paradigm. For these purposes, we developed an emotional version of the task switching paradigm of Whitmer and Banich (2007), the Affective Shift Task (AST), that we applied to a relatively large sample of healthy and dysphoric or sub-clinically depressed undergraduates. An advantage of conducting this study with a dysphoric sample is the exclusion of medication use, which can influence cognitive functioning (for a review see Amado-Boccaro, Gougoulis, Poirier, Galinowski, & Loo, 1995).

Method

Participants

In this study, 120 undergraduates of Ghent University participated in return for credits or financial compensation (8 €). They were recruited by means of an on-line participant panel system. Participants completed the BDI-II-NL (Beck, Steer, & Brown, 1996; Van Der Does, 2002) as a screening measure. On the day of the experiment, they completed the BDI-II-NL again and 14 participants were removed because their BDI-II-NL score changed. Moreover, 10 participants were excluded because of drug or medication (including antidepressant) use. This resulted in a final sample of 96 participants (83 females, 13 males) ranging from 17 to 25 years in age ($M=19$, $SD=1$). Participants who scored below the cut-off of 14 were classified as healthy controls ($N=54$). Based on the cut-offs (Beck et al., 1996), participants who scored 14 or higher were classified as dysphoric or sub-clinically depressed ($N=42$).

Material

Self-report questionnaires

Depressive symptoms. The Beck Depression Inventory - Second Edition (BDI-II-NL) was used to measure depressive symptoms (Beck et al., 1996; Van Der Does, 2002). The BDI-II-NL is a 21-item self-report measure which assesses the severity of a range of affective,

somatic and cognitive symptoms of depression. Individuals rate each symptom on a scale ranging from 0 to 3 (scores on the BDI-II-NL could range from 0 to 63). The acceptable reliability and validity of the BDI-II have been well documented (Beck, Steer, & Garbin, 1988).

Rumination. The Ruminative Response Scale (RRS-NL) was used to measure rumination (Nolen-Hoeksema & Morrow, 1991; Raes & Hermans, 2007). The RRS-NL is a 26-item self-report measure and consists of items that describe responses to a depressed mood that are focused on the self, symptoms, or consequences of depressed mood. Participants are requested to indicate how often they engage in these responses using a four-point Likert scale ranging from 1 (almost never) to 4 (almost always). Total rumination scores range from 26 to 104. A factor analysis of the RRS has identified two separate subscales that are differentially related to depressive symptoms. The first, reflective pondering, consists of five questions that assesses the degree to which individuals engage in cognitive problem solving to improve their mood and the second, brooding, consists of five items that assesses the degree to which individuals passively focus on the reasons for their distress. The RRS is a reliable and valid measure of rumination with good psychometric properties (Treyner et al., 2003).

Affective Shift Task

Material. The task was programmed using E-prime 2.0 software package and ran on a Windows XP computer with a 100 Hz, 19-inch colour monitor.

The stimuli were faces and were taken from the Karolinska Directed Emotional Faces (KDEF) (Lundqvist, Flykt, & Öhman, 1998). All faces were adjusted to exclude interference of background stimuli (hair) and were adjusted to the same size (326 x 326 pixels). Based on intensity (1 = not at all – 9 = completely) and arousal (1 = calm - 9 = aroused) ratings a total of 12 happy (Intensity: $M = 6.69$, $SD = 3.89$; Arousal: $M = 1.59$, $SD = 2.01$) and 12 angry (Intensity: $M = 6.23$, $SD = 1.66$; Arousal: $M = 3.82$, $SD = 1.95$) faces were selected from a

validation study of the KDEF picture set (Goeleven, De Raedt, Leyman, & Verschuere, 2008). Faces were chosen as stimuli material instead of words as depression seems to be characterized by disruptions in the interpersonal domain (Gotlib & Hammen, 2002). Recently, researchers have begun to use other people's facial expressions in order to investigate depression and the role of rumination as faces are ecologically valid interpersonal stimuli (Joormann et al., 2006; Raes, Hermans, & Williams, 2006). In particular, angry faces were selected as they have a direct relevance to depression, which is characterized by fear of social rejection (Barnett & Gotlib, 1988).

A modified version of the task switching paradigm of Mayr and Keele (2000) and Whitmer and Banich (2007) was used to obtain an index of inhibition (the capacity to inhibit previously relevant information) and an index of set shifting (the capacity to shift mental set from old to new information) in response to emotional as well as non-emotional material. In this cue shifting task one white cue word 'emotion', 'gender' or 'colour' is centrally presented for 500 ms on every trial, signalling the task-relevant feature of the subsequently presented faces. Immediately after this cue word, four faces are centrally displayed on the screen, presented in a squared grid (2 x 2 matrix) that is shown on a black background. Each face could differ on three distinct stimulus dimensions: emotion (angry or happy), gender (male or female) and colour (dark-grey or light-grey). Participants were instructed to detect and locate the face that differs from the others as fast and accurately as possible. They were asked to perform this odd-one-out search based on the relevant dimension indicated by the preceding cue word. They had to react by pressing the corresponding button on a response box: the four buttons on the response box were arranged so that they were spatially compatible with the four faces displayed on the screen. Participants were instructed to rest both thumbs on the lower left and right keys and both index fingers on the upper left and right keys and to press the correct button corresponding to the location of the deviant face in the squared grid. After a

response a blank screen was presented for 100 ms before the cue for the next trial appeared. The faces appeared on the screen until participants made a response. The deviant face was randomly assigned to one of the four locations in the squared grid. Within each stimulus display, each face differed from the others on only one dimension (i.e. emotion, gender or colour). Deviants on all three dimensions were present within each stimulus display, with always one different face regarding emotion, gender, and colour being presented. An example of a trial sequence and stimulus display is presented in Figure 1.

(Figure 1 about here)

Design. In line with Whitmer and Banich (2007), we calculated effects for inhibition and set shifting based on comparisons between specific trial types, see Table 1 for more details. One *trial type* consists of two or three cued dimensions/trials programmed in sequence. The order of the cued dimensions was pseudo-random depending on the trial type. A trial type is determined by the relation between the final cued dimension and the preceding one or two trials. Four trial types were programmed with the additional constraint that inhibitory, control, and unclassified trial types occurred equally often, with 48 trials for each of these trial types, and the remaining 72 trials were repeat trials. All calculations were based on response to the last trial in the sequence. The cued dimensions (emotion, gender and colour) of this last trial were presented equally often (i.e. on the 48 inhibitory trials the three cued dimensions emotion, gender or colour were each presented 16 times). On the inhibitory trials, all combinations of the emotion dimensions (happy and angry) on trial 1 and 3 were included in the analyses.

Reaction times were used to obtain separate measures of inhibition and set shifting (Whitmer & Banich, 2007). To obtain a measure of *inhibition*, performance on inhibitory trials were compared to performance on control trials. On inhibitory trials, the cue on the last trial is different from the cue on the immediately preceding trial ($n-1$) but the same as the cue

two trials back ($n-2$) (e.g., emotion-gender-emotion). Control trials are defined as those in which the cue is different from the cue on the preceding two trials ($n-1$ and $n-2$), which also have different cues from each other (e.g., colour-gender-emotion). To obtain a measure of inhibition not confounded by shifting abilities, performance on inhibitory trials was compared with performance on control trials. Both are preceded by at least two set shifts, but in the inhibitory trials a recently abandoned task set has to be activated again (e.g., emotion at the end of an emotion-gender-emotion sequence) (Mayr & Keele, 2000). *Set shifting* cost is measured by the additional time it takes to respond to non-inhibitory trials that require the use of a different task set than used in the previous trial (e.g. shift from gender to emotion) as compared to repeat trials, in which the same task set is used on two trials (e.g., emotion-emotion). Set shifting costs are thought to reflect time needed to reconfigure the cognitive processes involved in the representation of the to-be-used task set (Monsell, 2003).

(Table 1 about here)

Procedure

After completing the informed consent form, participants performed the Affective Shift Task. They practiced the Affective Shift Task until they responded correctly to 80% out of a maximum of 12 trials. Only in these practice trials a feedback sign ('correct' or 'incorrect') appeared for 500 ms before the 100 ms blank screen. They then completed two blocks of test trials, each consisting of 108 trial sequences, with a short break in between. Participants completed the self-report questionnaires BDI-II-NL (Van der Does, 2002) and RRS-NL (Raes et al., 2007) at the end of the session to avoid mood induction effects. At the end of the experiment all participants were fully debriefed.

Results

Overall results

For the analyses of reaction times, median scores were used, which allows maximum inclusion of observations. Only full trials in which all three trials were correct were included. When taking this criterion into account, average accuracy was 94%. All further analyses were based on response to the last or third trial.

A 3 (cue: emotion, gender, colour) x 4 (trial type: inhibitory, control, unclassified, repeat) repeated measures ANOVA was used to assess overall performance across the whole task. Analyses revealed a main effect of trial type, $F(3,93) = 24.29$, $p < .001$. Response on inhibitory trials ($M = 1631$ ms, $SD = 289$ ms) was significantly longer than RT on control ($M = 1552$ ms, $SD = 260$ ms), $t(95) = 4.77$, $p < .001$, and unclassified trials ($M = 1546$ ms, $SD = 271$ ms), $t(95) = 5.03$, $p < .001$. Response on repeat trials ($M = 1494$ ms, $SD = 260$ ms) was significantly faster than RT on control trials, $t(95) = 3.82$, $p < .001$; unclassified trials, $t(95) = 3.98$, $p < .001$ and inhibitory trials, $t(95) = 8.52$, $p < .001$. These patterns of response latencies are similar to those found by Mayr and Keele (2000) and Whitmer and Banich (2007).

Analyses revealed a main effect of cue, $F(2,94) = 640.94$, $p < .001$. Response on the cued dimension of emotion ($M = 1829$ ms, $SD = 339$ ms) was significantly longer than the response on the cued dimension of gender ($M = 1761$ ms, $SD = 282$ ms), $t(95) = 2.95$, $p < .01$. Response on the cued dimension of colour ($M = 906$ ms, $SD = 228$ ms) was significantly faster than the response on emotion, $t(95) = 31.83$, $p < .001$; and gender, $t(95) = 33.99$, $p < .001$. Due to the very fast response on colour and the large difference with responses on emotion and gender, we excluded from further analyses trial types where colour was programmed as the last trial in the sequence.

Dysphoric versus non-dysphoric individuals

Participants

Based on the cut-offs of the BDI-II (Beck et al., 1996), participants who scored below the cut-off of 14 were classified as healthy controls ($N = 54$), and participants who scored 14 or higher were classified as dysphoric or sub-clinically depressed ($N = 42$). Age and gender of the participants in both groups were not different (see Table 2).

(Table 2 about here)

Inhibition

To obtain an index of general inhibition capacity, performance on inhibitory trials was compared with performance on control trials (see Table 1). A Mixed ANOVA with Trial Type (inhibitory, control) and Cue Type (emotion, gender) as within subject factors and Group (dysphorics, non-dysphorics) as between subject factor revealed a significant main effect of Cue Type, $F(1,94) = 4.18, p < .05$, with response on the cued dimension emotion ($M = 1903$ ms, $SD = 396$ ms) being slower than response on gender ($M = 1838$ ms, $SD = 337$ ms), $t(95) = 2.15, p < .05$. Analyses revealed a main effect of Trial Type, $F(1,94) = 6.60, p < .05$, with response on inhibitory trials ($M = 1902$ ms, $SD = 353$ ms) being slower than response on control trials ($M = 1839$ ms, $SD = 362$ ms), $t(95) = 2.53, p < .05$. Importantly, analyses revealed a significant Cue Type x Trial Type interaction, $F(1,94) = 10.07, p < .01$. This showed that response on the inhibitory gender trials ($M = 1839$ ms, $SD = 330$ ms) and the control gender trials ($M = 1836$ ms, $SD = 413$ ms) was not significantly different, $t(95) < 1$; whereas response on the inhibitory emotion trials ($M = 1965$ ms, $SD = 440$ ms) was slower than response on the control emotion trials ($M = 1842$ ms, $SD = 406$ ms), $t(95) = 4.04, p < .001$. There were no main or interaction effects involving Group, all $F_s < 1$, indicating that there was no general inhibition impairment in the dysphoric group.

To investigate valence specific inhibition impairments, performance on the inhibitory emotion trials only, with all possible combinations of the emotion dimensions (happy, angry) on trial 1 (T1) and 3 (T3), was investigated (see Table 1). A Mixed ANOVA with Emotion T1 (happy, angry) and Emotion T3 (happy, angry) as within subject factors and Group (dysphorics, non-dysphorics) as between subject factor revealed a significant main effect of Emotion on trial 3, $F(1,94) = 30.86, p < .001$, with faster responding on the cued emotion dimension angry ($M = 1903$ ms, $SD = 445$ ms) compared to responding on the cued emotion dimension happy on trial 3 ($M = 2213$ ms, $SD = 604$ ms), $t(95) = 5.52, p < .001$. The Emotion T1 x Emotion T3 interaction was significant, $F(1,94) = 8.49, p < .01$. There were no main or interaction effects involving Group, all $F_s < 1$, indicating that there was no valence-specific inhibition impairment in the dysphoric group.¹

Set shifting

To obtain an index of general set shifting, performance on shift trials (the control and unclassified trials) were compared with performance on repeat trials (see Table 1). To investigate set shifts from emotional to non-emotional material and vice versa, we only included the control and unclassified trials with a shift from emotion (trial 2) to gender (trial 3) and a shift from gender (trial 2) to emotion (trial 3) and we included repeat trials only with emotion and gender as cued dimensions on trial 3. A Mixed ANOVA with Trial Type (control, unclassified, repeat) and Cue Type (trial 3: emotion, gender) as within subject factors and Group (dysphorics, non-dysphorics) as between subject factor revealed a main effect of trialtype, $F(2,93) = 9.48, p < .001$, with faster responding on repeat trials ($M = 1757$ ms, $SD = 297$ ms) compared to control trials ($M = 1856$ ms, $SD = 361$ ms), $t(95) = 3.43, p < .01$; and unclassified trials ($M = 1826$ ms, $SD = 335$ ms), $t(95) = 3.08, p < .01$. This finding reveals the expected shift cost. There were no main or interaction effects involving Group, all $F_s < 1$, indicating that there was no general set shifting impairment in the dysphoric group.²

To investigate valence-specific set shifting impairments, performance on the control and unclassified trials only with shifts from emotion (trial 2: angry, happy) to gender (trial 3) was investigated (see Table 1). A Mixed ANOVA with Trial Type (control, unclassified) and Valence specific (angry, happy) as within subject factors and Group (dysphorics, non-dysphorics) revealed no significant effects, all $F_s < 3$.

Low ruminators versus high ruminators

Participants

To investigate the association between rumination and attentional control, 50 participants were categorized as high ruminators ($RRS \geq 56$) and 46 as low ruminators ($RRS < 56$) based on a median split of the total RRS-score. The RRS-score was significantly higher in the high rumination group ($M = 65, SD = 7$) compared to the low rumination group ($M = 42, SD = 8$), $t(94) = 15.489, p < .001$. Note that the number of dysphoric participants were not distributed evenly over both rumination groups, $\chi^2(1, N = 96) = 29.21, p < .001$, with 35 dysphoric participants in the high rumination group, 7 dysphoric participants in the low rumination group, 39 non-dysphorics in the low rumination group and 15 non-dysphorics in the high rumination group.

Inhibition

To obtain an index of general inhibition ability, performance on inhibitory trials was compared with performance on control trials (see Table 1). The Mixed ANOVA with Trial Type (inhibitory, control) and Cue Type (emotion, gender) as within subject factors and Group (low ruminators, high ruminators) as between subject factor revealed the same main and interaction effects found with the dysphorics versus non-dysphorics classification: a main effect of Cue Type, $F(1,94) = 5.02, p < .05$, a main effect of Trial Type, $F(1,94) = 6.70, p < .05$, and a significant Cue Type x Trial Type interaction, $F(1,94) = 8.88, p < .01$. There were

no main or interaction effects involving Group, all $F_s < 1$, indicating that there was no general inhibition impairment in the rumination group.

To investigate valence specific inhibition impairments, performance on the inhibitory emotion trials only, with all possible combinations of the emotion dimensions (happy, angry) on trial 1 (T1) and 3 (T3), was investigated (see Table 1). A Mixed ANOVA with Emotion T1 (happy, angry) and Emotion T3 (happy, angry) as within subject factors and Group (low ruminators, high ruminators) as between subject factor revealed the same main and interaction effects as found with the dysphorics versus non-dysphorics classification, with a main effect of Emotion on trial 3, $F(1,94) = 30.20, p < .001$ and an Emotion T1 x Emotion T3 interaction, $F(1,94) = 7.58, p < .01$. These effects could be subsumed under the predicted three-way interaction effect involving group, $F(1,94) = 4.50, p < .05$. When participants had to react on a happy face in the last or third trial and they had inhibited a happy face in the first trial, there was no significant difference in response on the third trial between the high ruminators ($M = 2063$ ms, $SD = 593$ ms) compared to the low ruminators ($M = 2212$ ms, $SD = 783$ ms), $t(94) < 1$. When participants had to react on an angry face in the last or third trial and they had inhibited an angry face in the first trial, the high ruminators showed a faster response on the third trial ($M = 1739$ ms, $SD = 469$ ms) compared to the low ruminators ($M = 1956$ ms, $SD = 490$ ms), $t(94) = 2.21, p < .05$. These results indicate that there was impaired inhibition of negative material, the first angry face, in the high rumination group (see Figure 2).

(Figure 2 about here)

We performed a stepwise regression analysis with the valence-specific inhibition impairment as dependent variable and scores for depressive symptoms (BDI-II), depressive rumination or brooding and reflective pondering (RRS) as independent variables. The variable that was predictive of the impaired inhibition was depressive brooding, $F(1,94) = 5.47, p < .05$, with $B = -28.59, S.E. B = 12.22, \beta = -.23, R^2 = .05$. Higher scores on depressive brooding were

associated with impaired inhibition of negative material, reflected in a faster response on an angry face in the last trial when participants had inhibited an angry face in the first trial. The zero-order correlations of the variables that were entered in the regression analyses are presented in Table 3.

(Table 3 about here)

Set shifting

To obtain an index of general set shifting ability, performance on shift trials (the control and unclassified trials) were compared with performance on repeat trials (see Table 1). A Mixed ANOVA with Trial Type (control, unclassified, repeat) and Cue Type (trial 3: emotion, gender) as within subject factors and Group (low ruminators, high ruminators) as between subject factor revealed the same main effect of Trial Type as found with the dysphorics versus non-dysphorics classification, $F(2,93) = 8.67, p < .001$. There was a Trial Type x Group interaction, $F(2,93) = 3.25, p < .05$. Further analysis showed that the shift cost [$((RT\ control + RT\ unclassified)/2) - RT\ repeat$] was higher for the high ruminators ($M = 132\ ms, SD = 223\ ms$) compared to the low ruminators ($M = 32\ ms, SD = 148\ ms$), $t(94)=2.56, p < .01$, indicating that there was a general set shifting impairment in the high rumination group (see Figure 3).

(Figure 3 about here)

Stepwise regression analysis with shifting impairment as dependent variable and scores for depressive symptoms (BDI-II), depressive rumination or brooding and reflective pondering (RRS) as independent variables. The variable that was predictive of the shifting impairment was depressive brooding, $F(1,94) = 10.78, p < .001$, with $B = 15.71, S.E. B = 4.78, \beta = .32, R^2 = .10$. Higher scores on depressive brooding were associated with impaired shifting, reflected in higher shift cost.

To investigate valence specific set shifting impairments, performance on the control and unclassified trials only with shifts from emotion (trial 2: angry and happy) to gender (trial 3) was investigated (see Table 1). A Mixed ANOVA with Trial Type (control, unclassified) and Valence specific (angry, happy) as within subject factors and Group (low ruminators, high ruminators) revealed no significant effects, all $F_s < 2$.

Discussion

In the present study we examined whether depressive symptoms and rumination were related to impairments in executive control. A first aim was to investigate whether sub-clinically depressed individuals show impairments in inhibition as well as set shifting in response to emotional and non-emotional material. A second aim was to investigate whether depressive rumination, a specific cognitive feature of depression, is related to impairments in inhibition as well as set shifting in response to emotional and non-emotional material. For this purpose, we administered a new emotional version of the task switching paradigm of Whitmer and Banich (2007), the Affective Shift Task (AST), in a relatively large sample of healthy and sub-clinically depressed undergraduates.

In line with Whitmer and Banich (2007), we calculated effects for inhibition and set shifting based on comparisons between specific trial types. The trial types elicited response relationships similar to those found by Whitmer and Banich (2007), with slower responding on inhibitory trials compared to responding on shift trials and with faster responding on repeat trials compared to responding on inhibitory and shift trials. These observations attest to the validity of the Affective Shift Task. Two major findings resulted from our manipulations: (1) individuals with depressive symptoms showed few impairments in executive control, whereas (2) individuals who tend to ruminate showed inhibition impairments when processing negative material and shifting impairments as reflected in a larger shift cost. These results are

of importance to the research investigating the underlying mechanisms of impaired information processing and mood regulation in depression. These findings are discussed below.

When comparing dysphorics to healthy controls, the results showed no differences in executive control performance. No effects were observed of depressive symptoms on inhibition as well as set shifting performance in response to non-emotional and emotional information. There only was an association between depression scores and set-shifting when individuals with moderate-severe depression ($BDI-II \geq 20$) were compared to non-depressed individuals. The absence of an association between depressive symptoms ($BDI-II \geq 14$) in general and executive control impairments is in line with previous studies (Daches, Mor, Winquist, & Gilboa-Schechtman, in press; Whitmer & Banich, 2007), in which it was shown that depression was not associated with impairments in executive control when depressive rumination was statistically controlled for. In line with previous studies (Merriam et al., 1999), higher levels of depression severity were associated with some executive control impairments, in our study at the level of set-shifting when attention had to be shifted from emotional to non-emotional material. As higher depression scores are characterized by higher levels of rumination and brooding in particular it may still be that these effects are driven by the association between rumination and executive control rather than the relation between depression and executive control. It is noteworthy that because of the strong correlation between depression severity and brooding, it is difficult to examine the singular effect of rumination, independent from depression in more severely depressed individuals. The number of participants with high depression scores ($BDI-II \geq 20$) does not permit examining distinct relations between depression severity, rumination, and executive control.

A second aim of this study was to investigate the association between a specific cognitive feature of depression, rumination, and executive control. Several theorists have

argued that impairments in executive control are related to individual differences in the tendency to ruminate (Davis & Nolen-Hoeksema, 2000). In this study individuals who tend to ruminate showed a specific impairment in inhibiting negative information. This inhibition impairment was most strongly predicted by depressive rumination or brooding, which is in line with previous research (Whitmer & Banich, 2007). Although, several researchers have proposed that rumination is caused by a failure of inhibitory executive control (Hester & Garavan, 2005; Linville, 1996; Shapiro, 2002; Ursin, 2005; Watkins & Brown, 2002), to date only few studies have directly investigated this association systematically (Goeleven et al., 2006; Joormann, 2006; Whitmer & Banich, 2007).

In addition to inhibition impairments, high ruminators showed impaired set shifting, which was reflected in a larger shift cost compared to low ruminators. In the context of set shifting, no valence specific effects were observed. Importantly, we observed that this shifting impairment was most strongly predicted by depressive rumination or brooding. The association between brooding and shifting impairments is in line with previous research (Whitmer and Banich, 2007). Although in Whitmer and Banich (2007), depressive rumination was rather moderately associated with a poor ability to shift task set, $r(48) = .34, p < .05$, they observed that shifting difficulties were more pronounced in anger rumination (repetitive, unintended thoughts about angry experiences) and intellectual reflection (intellectual self-reflection). The absence of valence-specific shifting impairments is in line with previous research showing shifting impairments in the context of non-emotional material (Davis & Nolen-Hoeksema, 2000; Whitmer & Banich, 2007).

The present study improves over previous studies which have mainly examined executive functions in relation to depressive symptoms in general, with only limited research on rumination. In addition only few studies have examined the influence of depressive symptoms and rumination separately on executive functions in response to emotional as well

as non-emotional material. To our knowledge, our study is the first to examine whether depressive symptoms in general and rumination specifically are related to impairments in inhibition as well as set shifting in response to emotional and non-emotional material within a single paradigm. There are several interesting implications of these findings. First, these data provide empirical evidence to support the idea that reduced inhibition and shifting capacity is associated with rumination. This study adds to a growing literature showing that executive control plays an important role in rumination (Donaldson, Lam, & Mathews, 2007). At a broader level these data show the link between information processing characteristics and thinking styles. Second, our results showed that depressive rumination or brooding was the strongest predictor of the inhibition and shifting impairments compared to reflective pondering and BDI-II scores. These findings suggest that rumination, a typical cognitive feature of depression, could be more proximally related to executive control impairments than the broad construct of depression. We suggest that future research on executive control impairments in depression should focus on processes such as depressive rumination.

The findings of the current study are of importance regarding vulnerability for depression, given that executive control impairments associated with rumination may contribute to the affective core symptoms of depression such as anhedonia, sustained negative affect and problems in mood regulation (Joorman, Yoon, & Zetsche, 2007). However, the nature of the proposed executive control impairments related to rumination requires further investigation. That is, the association between rumination and information processing in the present study does not inform about causality and as such it is not possible to determine the functional relationship between, ruminative, information processing impairments and depression. One account is that the association between rumination and executive function is due to rumination reducing executive capacity (Watkins & Brown, 2002). Alternatively, we have recently proposed that executive control impairments rather cause rumination (De Raedt

& Koster, 2009; Whitmer & Banich, 2007). Future research has to focus on the causal relationship between executive impairments and rumination. Nevertheless, at present, an improved understanding of the executive mechanisms affected in ruminators can be important clinically because it allows developing therapeutic interventions that remediate impairments in processing in high ruminators.

There are some limitations to the present study. We used an undergraduate dysphoric sample. Despite the advantages of the ability to exclude medication use, it is equally important to generalise the effects to a clinically depressed sample. Moreover, undergraduates may be characterized by relatively good executive functions. For these two reasons our study may represent an underestimation of the association between depression and executive functions. Finally, this study was the first to use the Affective Shift Task. Despite the meaningful results obtained in this study, further work is necessary to establish further the reliability and validity of this task. One crucial point is the ability of the task to examine inhibition and set shifting impairments in relation to emotional as well as non-emotional material. At present we cannot exclude the possibility that performance on the non-emotional trials is influenced by the presence of emotional information in this task. Nevertheless, our results are in line with those found by Whitmer and Banich (2007) using non-emotional material, suggesting that our effects are not influenced by the presence of emotional information in the task.

In sum, we aimed to link emotion processing in depression to the basic cognitive psychology literature on executive control because a better understanding of the relation between cognitive control and biased emotion processing can improve our theoretical understanding of information processing deficiencies in depression. In conclusion, the results of this study offer new insights into the association between executive control and depressive symptoms. The findings indicate that rumination, but not dysphoria, is related to impairments

in inhibition and shifting functions. These findings warrant further research into the trajectory of executive control and rumination in the development of depression.

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Footnotes

¹ Additional analyses with individuals scoring above the clinical cut-off score ($\text{BDI-II} \geq 20$, $N = 23$), matched to individuals with the lowest BDI-II scores ($N = 23$), revealed no general or valence specific inhibition impairment.

² Additional analyses with individuals scoring above the clinical cut-off score ($\text{BDI-II} \geq 20$, $N = 23$), matched to individuals with the lowest BDI-II scores ($N = 23$), revealed a significant Trial Type x Cue Type x Group interaction effect, $F(2,43) = 5.58$, $p < .01$, indicating a higher shift cost in the depressed group when shifting from emotion to gender, $t(44) = 2.51$, $p < .05$.

Table 1

Trial types and measures of executive function.

Trial Types	Cue Trial 1 (n-2)	Cue Trial 2 (n-1)	Cue Trial 3 (n)
Trials requiring a Set Shift:			
48 Inhibitory Trials (a-b-a)	emotion	gender or colour	emotion
	gender	emotion or colour	gender
	colour	emotion or gender	colour
48 Control Trials (c-b-a)	gender (colour)	colour (gender)	emotion
	colour (emotion)	emotion (colour)	gender
	gender (emotion)	emotion (gender)	colour
48 Unclassified Trials (b-b-a)	gender (colour)	gender (colour)	emotion
	emotion (colour)	emotion (colour)	gender
	emotion (gender)	emotion (gender)	colour
Trials without a Set Shift:			
72 Repeat Trials (a-a)		emotion	emotion
		gender	gender
		colour	colour
Measuring of Executive Function			
Inhibition			
Indexed:	RT to Inhibitory Trials - RT to Control Trials		
Interpretation:	High inhibition scores reflect good executive ability		
Set Shifting			
Indexed:	Average of RT to Non-inhibitory Trials [RT to Control + RT to Unclassified/2] - RT to Repeat Trials		
Interpretation:	Smaller difference is better executive ability		

Table 2

Age and Gender of the Participants as a Function of Group.

	N	Age <i>M</i> (<i>SD</i>)	Gender <i>M/F</i>
Non-dysphorics	54	18.94 (1.56)	8/46
Dysphorics	42	18.43 (0.63)	5/37
Low Ruminators	46	18.91 (1.38)	10/36
High Ruminators	50	18.54 (1.13)	3/47

Table 3

Zero-order correlations of the variables that were entered in the stepwise regression analyses.

	Inhibition capacity of negative material	General shift cost	BDI total	RRS total	RRS brooding	RRS pondering
Inhibition capacity of negative material						
General shift cost	.08					
BDI total	-.11	.19				
RRS total	-.14	.29**	.65**			
RRS brooding	-.24*	.32**	.61**	.83**		
RRS pondering	-.02	.06	.19	.52**	.27**	

* $p < .05$, ** $p < .01$

Figure 1. An Example of a Trial Sequence and Stimulus Display.

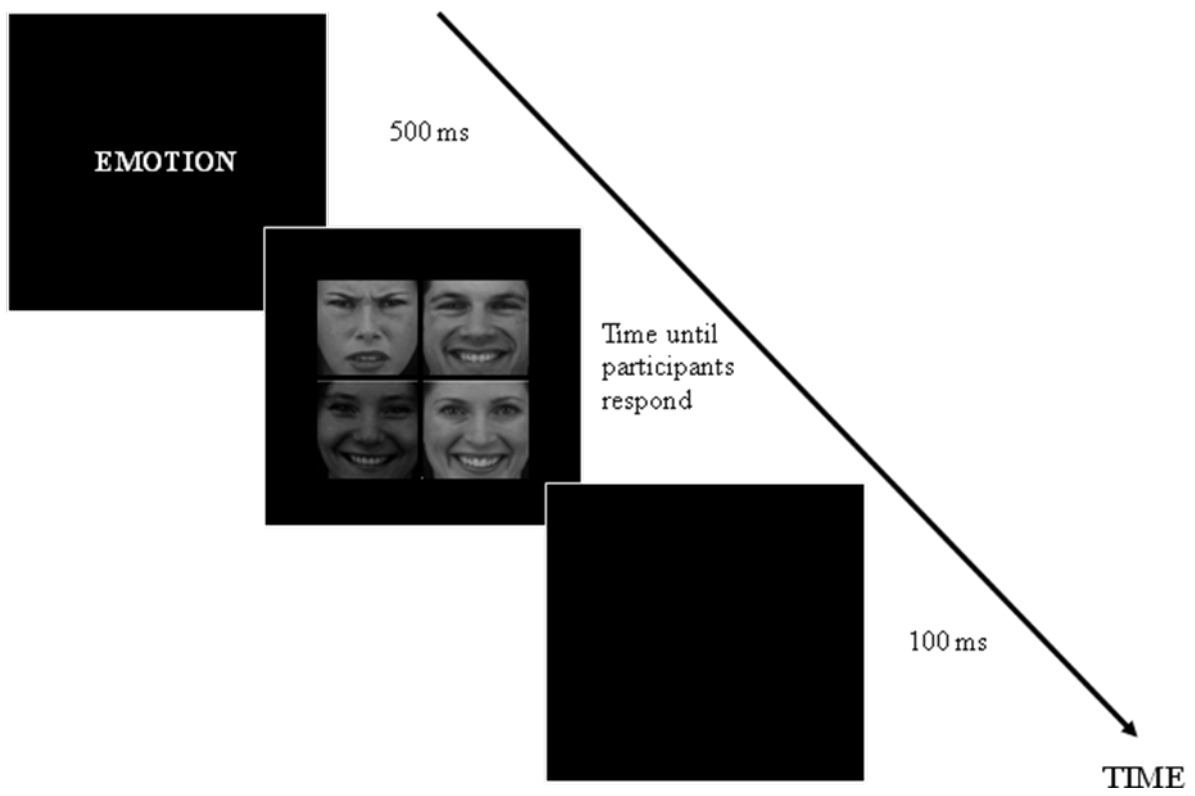


Figure 2. Valence-specific Inhibition Capacity as a Function of Rumination.

Impaired inhibition of negative material, angry face on the first trial, in the high rumination group.

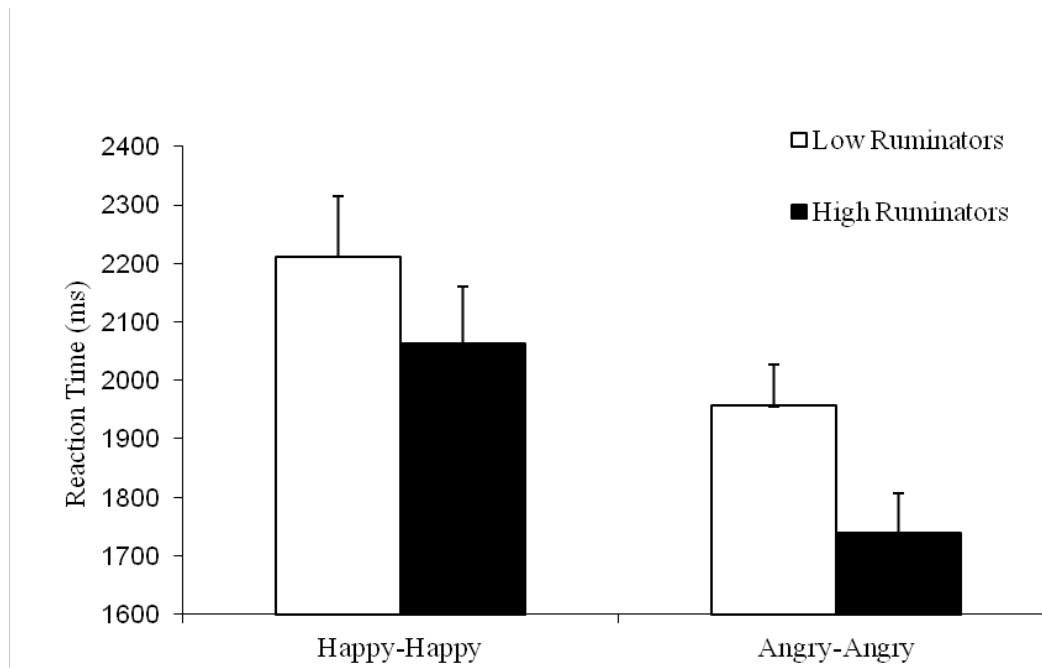


Figure 3. Shift Cost as a Function of Rumination.

General set shifting impairment in the high rumination group.

