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Brief Report

Depressive symptoms and cognitive control in a mixed antisaccade task: Specific effects of depressive rumination

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Abstract

Growing empirical evidence suggests that cognitive and affective problems in depression may be a reflection of cognitive control impairments. However, to date, the nature of such impairments is still poorly understood and further investigation of this topic is required to advance current knowledge on the underlying vulnerability factors for depression. Using a mixed antisaccade paradigm, the present study examined if depressive symptoms in general, and more specifically rumination, are related to impairments in cognitive control functions such as inhibition and switching. The results on antisaccade latency and error rates indicated that depressive symptoms in general were not related to impairments in inhibition and switching. However, rumination was associated with impaired inhibition such that high, compared to low, ruminators had slower antisaccade latencies. No group differences were observed on antisaccade error rates. Implications for understanding the underlying vulnerability factors for the development of depressive symptoms are discussed.

Key words: depression, rumination, cognitive control, inhibition, switching

Introduction

Depression is associated with a wide range of affective and somatic symptoms as well as cognitive impairments (Austin, Mitchell, & Goodwin, 2001). Growing empirical evidence suggests that cognitive problems in depression may be a reflection of fundamental impairments in the central executive system (Joormann, Yoon, & Zetsche, 2007), which is mainly responsible for the control and regulation of cognitive processes and response selection. In support of these findings recent neuroimaging evidence shows that depressed individuals have reduced brain activity in areas such as the anterior cingulate cortex (ACC) and dorsolateral prefrontal cortex (DLPFC). The executive control impairments in depression may stem from the hypoactivation of these prefrontal areas which are thought to subserve cognitive control (Davidson, Pizzagalli, Nitschke, & Putnam, 2002).

Evidence from computerized neuropsychological tasks also shows that depressed individuals have impaired performance on tasks measuring cognitive control namely inhibition and switching (Joormann et al., 2007). These cognitive control functions moderately correlate with one another, but are clearly separable (Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). The evidence from neuropsychological tasks is inconclusive as the tasks have mainly relied on reaction time measures that provide indirect indices of cognitive processing (Weierich, Treat & Hollingworth, 2008). The use of manual response latencies as a measure of cognitive control impairments in depression was recently criticized as depression is characterized by deficits in motor response and response selection (Joormann et al., 2007). Therefore, it is important that cognitive control functions are also assessed using paradigms that provide a more direct assessment of cognitive control in depression.

A promising paradigm that provides a more direct assessment of cognitive control is the antisaccade task (Hallet, 1978). In a recent latent variable analysis of several tasks measuring executive processes of working memory it was found that the antisaccade task

loaded highly and more reliably on the inhibition function compared to many other tasks (Miyake et al., 2000). In a typical antisaccade trial, participants are instructed to generate a saccade to the mirror position of an abrupt peripheral cue in the opposite hemi-field from where a cue was initially presented. Performance on antisaccade trials is usually compared to performance on prosaccade trials where participants are simply instructed to look towards the cue, eliminating any conflict between volitional and stimulus-driven processes. The main dependent variables in the antisaccade task are saccade latency (time elapsed between onset of cue and the first saccade generated in the right direction) and error rates (misdirected saccades). Successful antisaccade performance requires the simultaneous inhibition of the cue and the generation of a volitional saccade to the mirror side of the cue, two processes that are believed to be programmed in parallel and compete for execution (Massen, 2004). Thus, the antisaccade paradigm provides a useful framework for investigating top-down cognitive control influences over stimulus-driven bottom-up processes (see Hutton & Ettinger, 2006, for a review).

Antisaccade performance has been studied in various psychiatric disorders to investigate cognitive control (see Hutton & Ettinger, 2006, for a review). As cognitive control impairments are considered an important cognitive vulnerability factor in depression (De Raedt & Koster, 2010; Joormann et al., 2007), the first aim of the present study was to investigate whether individuals with depressive symptoms show impairments on inhibition and switching. The few studies investigating antisaccade performance in unipolar depression (Smyrnis, Eydokimidis, Stefanis, Avramopoulos, & Constantinidis, 2003; Sweeney, Strojwas, Mann, & Thase, 1998) and dysphoria (Derakshan, Salt, & Koster, 2009) have found increased antisaccade error rates in this population, thought to reflect reduced cognitive control. In the present study, we included individuals scoring either high or low on the BDI-II (Beck, Steer, & Brown, 1996), with all dysphoric or sub-clinically depressed participants scoring 14 or

higher. It has been demonstrated that young persons showing depressive symptoms are at risk for developing clinical depression prospectively (Fergusson, Horwood, Ridder & Beautrais, 2005).

The second aim of the present study was to examine the relationship between cognitive control and specific cognitive symptoms of depression. Recently, it has been proposed that rumination, a typical cognitive feature of depression and an important cognitive vulnerability factor implicated in the aetiology, maintenance and recurrence of depression, could be even more proximally related to cognitive control impairments than the broad construct of depression (Koster, De Lissnyder, Derakshan, & De Raedt, in press). Rumination is defined as a recurrent series of thoughts focused on the causes, symptoms, and implications of one's depression (Nolen-Hoeksema, 1991). A ruminative thinking style has considerable negative consequences and is an important cognitive vulnerability factor for depression. Numerous studies have demonstrated that rumination is related concurrently with depressive symptoms and prospectively with the onset, severity and duration of depression, and recovery from depression (see Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008, for a review). In the literature, two types of rumination are distinguished (Treynor, 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 especially characterized by high levels of brooding (Treynor et al., 2003).

There is increased evidence to show that cognitive control impairments in depression are most pronounced for negative information. However there is also some evidence for cognitive control impairments when processing non-emotional information (Joormann et al., 2007). To date, only a few studies have investigated the relation between depressive

symptoms, rumination and impaired cognitive control in the context of emotional (De Lissnyder, Koster, Derakshan, & De Raedt, 2010; Joormann, 2009) and non-emotional (Davis & Nolen-Hoeksema, 2000; Whitmer & Banich, 2007) material. Interestingly, Whitmer and Banich (2007) investigated inhibition as well as switching in rumination in response to non-emotional material using a behavioural paradigm. The results of their study indicated that depressive rumination or brooding was specifically associated with impaired inhibition, and not with switching, for non-emotional material. Using an adapted version of the paradigm (Whitmer & Banich, 2007), by including non-emotional as well as emotional information, we largely replicated their findings. We observed a general switching impairment and impaired inhibition specifically for negative material in relation to rumination. Moreover, the variable that was most predictive of the cognitive control impairments was brooding (De Lissnyder, Koster, Derakshan, & De Raedt, 2010). In these studies depressive rumination was more strongly linked to cognitive control than general depressive scores. The aim of the present study is to further investigate cognitive control in response to non-emotional information. Provided that studies using behavioural tasks rely on manual responding we sought to examine these findings using an eye-movement paradigm, *the mixed antisaccade task* (Ansari, Derakshan, & Richards, 2008).

To our knowledge, the current study is the first using the mixed antisaccade task to examine the relationship between depressive symptoms in general, rumination more specifically and the cognitive control functions *inhibition* and *switching*. We hypothesised that depressive symptoms and in particular rumination will be associated with a) inhibition impairments as indicated by impaired performance on antisaccade trials, and b) switching impairments as indicated by larger switch costs.

Method

Participants

The initial sample included 48 adults (two participants were excluded from analysis due to poor tracking). The final sample included 46 participants (30 females, 16 males) ranging from 19 to 50 years in age (*M*=28, *SD*=7). They were recruited by means of on-line participant panel systems of Birkbeck and University College, University of London. Participants completed the BDI-II (Beck et al., 1996) as a screening measure in order to be considered for the experiment. Upon invitation for the experiment, they completed this scale again. Participants who scored below 14 were classified as non-dysphoric (*N*=24). Participants who scored 14 or higher were seen as dysphoric or sub-clinically depressed (*N*=22) (Beck et al., 1996). An advantage of conducting this study with a dysphoric sample is the exclusion of medication use, which can influence cognitive functioning (see Amado-Boccara, Gougoulis, Poirier, Galinowski, & Loo, 1995, for a review). Individuals reporting that they were currently taking any psychiatric medication were excluded from the study. All had normal to corrected vision and were allowed to wear their glasses or contact lenses. Participants were paid (£8) for their contribution. The study was approved by the ethics committee at Birkbeck.

Materials

Self-report questionnaires

Beck Depression Inventory - Second edition (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report measure with robust psychometric properties (Beck, Steer, & Garbin, 1988) 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 and scores on the BDI-II could range from 0 to 63.

Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991). The RRS is a 22-item self-report measure which consists of items that describe responses to a depressed

mood that are focused on the self, symptoms, or consequences of the 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 22 to 88. A factor analysis of the RRS has identified two separate subscales that are differentially related to depressive symptoms, reflective pondering and brooding. The RRS is a reliable and valid measure of rumination with good psychometric properties (Treynor et al., 2003).

Eye-tracking system

A remote camera mounted below the computer monitor using the LC Technologies 'Eyegaze' system (LC Technologies Inc., 2003) tracked the participants' eye-movements. Infrared-based eye-tracking software was used that generates raw gaze location data at a sampling rate of 60Hz, allowing eye-movements to be recorded every 16.67 ms. The system uses the Pupil-Centre Corneal Reflection (PCCR) method to estimate gaze points (the intersection of the optic axis with the screen). The eye-tracker is calibrated using 9 fixation points, with which the software indicates whether or not valid gaze points can be calculated. The 'Eyegaze' system accommodates for several sources of error such as head range variation and pupil diameter variation. Data are collected from the eye for which the most rapid and accurate calibration is obtained.

The presentation of the stimuli was controlled by the DMDX program (Forster & Forster, 2003) which ensures millisecond timing accuracy. The stimuli were presented in 24-bit colour on a 1024x768 LCD monitor (ViewSonic 700b, cell response-time 35 ms). The eye-tracking system and DMDX were automatically synchronised at the beginning of each trial.

Mixed antisaccade paradigm

Participants were required to complete anti- and prosaccade tasks in two distinct conditions: In a *single* block, participants completed trials of the same task, either anti- or

prosaccade, in two different blocks. In a *mixed* block, anti- and prosaccade trials were presented in a pseudo-random sequence requiring participants to switch between both tasks. There is a paradoxical improvement in antisaccade performance when individuals are required to switch between anti- and prosaccade trials compared to repeat trials such that antisaccade latencies become faster and error rates increase on switch compared to repeat trials. This *antisaccade switch benefit* has been observed in a number of studies (e.g., Hodgson, Golding, Molyva, et al., 2004; Experiment1; Cherkasova, Manoach, Intriligator & Barton, 2002) using a variety of mixed antisaccade paradigms. This antisaccade switch benefit has been argued to reflect a greater allocation of attentional resources in the more attention demanding switch trials (Kristjansson, Chen, & Nakayama, 2001). Investigating this switch benefit is interesting as a recent study found evidence of a general switching impairment in relation to rumination (De Lissnyder et al., 2010).

Each trial began with the word 'Ready' (1000 ms) followed by a central fixation symbol (300 ms). In the single block this fixation symbol was a white cross (2°x2°). In the mixed condition the fixation symbol was either a white diamond (1.2°x2.4°) or a white circle (1.7° diameter), indicating the type of response (either anti- or prosaccade) to be made on that trial (see Figure 1). After symbol offset and a 200 ms blank screen, an oval cue appeared for 600 ms 11° to either the left or right side of the screen. Participants were required to direct their gaze as fast as possible either 'away' (antisaccade) from the oval cue, to its mirror position in the opposite hemi-field from the cue, or 'toward' the cue (prosaccade). The experiment comprised two sessions of 144 experimental trials each (288 trials in total). The order of the blocks in each session could be either mixed-single-mixed or single-mixed-single counterbalanced in a between-subject design. In each session there were 72 trials in the single block and 72 trials in the mixed block. The order of the pro- and antisaccade task (36 trials each) in the single blocks was randomized within subjects over both sessions. In the mixed

blocks, the instructional meaning of the circle and diamond fixation symbols for the task to be performed was counterbalanced in a within-subject design. Practice trials preceded each block.

Procedure

Participants completed the experiment in two sessions that were separated by a short break. After completing the informed consent form, participants rated their mood on five 100 mm Visual Analogue Scales (0: not at all-100: very much), including 'happy', 'irritable', 'sad', 'anxious', and 'comfortable', to indicate their mood states at that moment (VAS; Bond, Shine, & Bruce, 1995). They completed these scales also during the break and at the end of the experiment. To perform the antisaccade task, participants were seated directly in front of the computer monitor with their chin placed on a chin-rest, located 60 cm from the screen, in a dimly lit cubicle. The eye-tracker was calibrated before each session, speed and accuracy were emphasised.

After the antisaccade task participants completed a distractor task[1] before they completed the Beck Depression Inventory (BDI-II; Beck et al., 1996) and the Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991). At the end of the experiment all participants were fully debriefed.

Data preparation and methods of statistical analyses

Saccades were defined as eye-movements with velocities exceeding 30°/s (Massen, 2004; Reuter, Jager, Bottlender, & Kathmann, 2007) and amplitudes exceeding 3° which were made between cue onset and offset. Latency and percentage error of the first correct saccade were calculated. An *incorrect saccade* was defined as the first saccade after cue onset towards the cue (on antisaccade trials), or away from the cue (on prosaccade trials). The *latency* of the

first correct saccade was defined as the elapsed time between the onset of and the generation of the saccade in the correct direction. Trials were excluded from analysis if (1) eye-tracking was interrupted due to lost pupil; (2) there were no eye-movements; and (3) the onset of the first saccade was shorter than 83 ms (i.e. anticipatory trials). Overall less than 8% of the data was lost. Analyses on the VAS scales showed that the task had no effect on participants' mood, all $Fs<2$.

Two main functions can be assessed using the mixed antisaccade task. *Inhibition* performance was assessed by comparing saccade latency and error rates between pro- and antisaccades. *Switching* performance was assessed by comparing saccade latency and error rate in the single block, where anti- and pro-saccade trials were repeated, to the mixed block where anti- and prosaccade trials were pseudo randomised. Switch cost was calculated by subtracting the mean correct latencies in the single block from the mean correct latencies in the mixed block (e.g., Ansari et al., 2008). The same method was applied to error rates[2].

For analyses concerning levels of *depression*, to compare single versus mixed blocks, Mixed ANOVAs with Task (antisaccade, prosaccade) and Block Type (single, mixed) as within subject factors and Group (dysphoric, non-dysphoric) as between subject factor were carried out on correct antisaccade latencies and error rates. The analyses concerning levels of *rumination* were exactly the same as the analyses concerning levels of depression, except the Group factor included low and high ruminators (see below for details).

Results

Group characteristics

First, to investigate the effects of depressive symptoms on cognitive control, participants who scored below 14 were classified as non-dysphoric (N=24). Participants who scored 14 or higher were seen as dysphoric or sub-clinically depressed (N=22) (Beck et al.,

1996). Dysphoric and non-dysphoric individuals did not differ on age, $t(44)<2$, or gender, $X^2(1,N=46)<1$.

Second, to investigate the effects of rumination on cognitive control, participants were categorized as high ruminators or low ruminators based on a median split of the total RRS-scores. Provided that individuals were pre-selected on depression scores there was a substantial range in rumination scores. It is important to note that the cut-off used to categorize individuals as high and low ruminators was similar to the mean rumination scores reported by Joormann and Tran (2009) using the same rumination questionnaire in an undergraduate sample. Twenty-three participants were classified as low ruminators (RRS<45.5) and 23 participants as high ruminators (RRS≥45.5). Note that the number of dysphoric individuals were not distributed evenly over both rumination Groups, $X^2(1,N=46)=8.71$, $p<.01$, with dysphoric individuals being over-represented in the high rumination group. No group differences on age, $t(44)<1$, or gender, $X^2(1,46)<1$, (15 females, 8 males) were found between the ruminator and non-ruminator Groups.

There were no significant correlations between age or gender and depressive symptoms (BDI-II), total rumination score (RRS), brooding or reflection. Depressive symptoms (BDI-II) were correlated with the total RRS scores, $r(46)=.60$, $p<.001$, and with the scales reflection, $r(46)=.31$, $p<.05$ and brooding, $r(46)=.55$, $p<.001$. Brooding was significantly correlated with reflection, $r(46)=.61$, $p<.001$.

Dysphoric versus non-dysphoric individuals

Correct saccade latencies

Analyses on mean correct saccade latencies revealed a main effect of Task, $F(1,44)=190.73$, $p<.001$, indicating that participants were slower on antisaccade (M=260 ms, SD=32 ms) than prosaccade trials (M=207 ms, SD=23 ms). The main effect of Block Type,

$F(1,44)=7.73$, $p<.01$, showed that participants were faster in the mixed (M=230 ms, SD=29 ms) compared to the single block (M=238 ms, SD=23 ms). The Task x Block Type interaction, $F(1,44)=56.48$, $p<.001$, indicated that participants had faster antisaccade latencies in the mixed (M=249 ms, SD=36 ms) compared to single block (M=271 ms, SD=32 ms), $t(45)=6.07$, $p<.001$. No differences were observed on prosaccade latencies, $t<2$. Of most relevance to our hypotheses, there were no effects involving the factor Group (all $Fs<1$).

Error rates

Analyses on error rates revealed a main effect of Task, $F(1,44)=81.70$, $p<.001$, with more errors on antisaccade (M=24.97%; SD=15.07%) than prosaccade trials (M=8.82%; SD=8.05%). The main effect of Block Type, $F(1,44)=54.47$, $p<.001$, indicated more errors in the mixed (M=20.19%; SD=11.43%) than the single block (M=13.60%; SD=10.41%). The Task x Block Type interaction, $F(1,44)=26.53$, $p<.001$, indicated more errors on antisaccade trials in the mixed (M=30.29%; SD=16.93%) than the single block (M=19.64%; SD=14.23%), $t(45)=8.62$, $p<.001$. There was a Block Type x Group interaction, $F(1,44)=8.45$, $p<.01$. The results showed that while both non-dysphoric and dysphoric individuals committed more errors in the mixed compared with the single block (non-dysphorics: M=22.43%; SD=12.02%; M=13.41%; SD=9.29%; $t(23)=7.41$, $p<.001$; and dysphorics: M=17.72%; SD=10.46%; M=13.80%; SD=11.72%; $t(21)=3.10$, $p<.01$), the difference on error rates between mixed and single blocks was greater in the non-dysphoric Group (Merror mixed – Merror single: M=9.02%; SD=5.96%) compared to the dysphoric individuals (M=3.92%; SD=5.93%), $t(44)=2.91$, $p<.01$. No other effects involving Group reached significance, all $Fs<2$.

Low vs. high ruminators

Correct saccade latencies

Figure 2 shows mean correct saccade latencies for each of the anti- and prosaccade tasks in each of the single and mixed blocks as a function of Group. The main effects were similar to the effects reported for dysphoric and non-dysphoric individuals. Of most relevance to our hypotheses, the Task x Group interaction, $F(1,44)=7.94$, $p<.01$, showed that high ruminators had slower antisaccade latencies (M=271 ms, SD=33 ms) compared to the low ruminators (M=250 ms, SD=28 ms), $t(44)=2.37$, $p<.05$. There were no Group differences on prosaccade latencies, $t<1$. No other main or interaction effects involving Group reached significance, all $Fs<1$.

To investigate which variable was most predictive of the slower antisaccade latencies we performed a stepwise regression analysis with antisaccade latency as dependent variable and scores for depressive symptoms (BDI), depressive rumination or brooding and reflective pondering (RRS) as independent variables. The only variable that proved to be predictive of the antisaccade latencies was depressive brooding, $F(1,44)=4.99$, $p<.05$, with $B=3.51$, $SEB=1.57$, $R^2=10\%$, $t(44)=2.23$, $p<.05$.

Error rates

An ANOVA with the error rates as dependent variable revealed that rumination was not associated with error rates, all $F's<1$. Furthermore, regression analyses including brooding and reflection, similar to the latency analyses, did not reveal significant effects of these rumination styles.

Discussion

Models of depression have emphasized the role of cognitive control impairments as risk factor for the onset and maintenance of depression (De Raedt & Koster, 2010; Joormann et al., 2007). In addition, there is emerging evidence that cognitive control impairments are linked specifically to cognitive symptoms of depression such as rumination (see Koster et al.,

in press, for a review) compared to depressive symptoms in general. In the present study we examined whether depressive symptoms in general, and rumination more specifically, are related to impairments in cognitive control, particularly the inhibition and switching functions in response to non-emotional material. For this purpose, we administered a modified version of the mixed antisaccade task in a sample of non-dysphoric and dysphoric undergraduates. The antisaccade task is regarded as a successful tool in demonstrating cognitive control impairments in various psychiatric disorders.

As main performance measures, we examined saccade latency (latency of the first correct saccade) and error rates (misdirected saccades). The pattern of findings in the mixed antisaccade task is consistent with previous research (Ansari et al., 2008) with overall greater error rates and slower latencies on the antisaccade compared to the prosaccade trials. Moreover, the typically observed antisaccade switch benefit is consistent with recent observations that an additional attention demanding task actually speeds up antisaccade latencies (Kristjansson et al., 2001). Of major importance to the aims of the current study were the findings that (1) depressive symptoms in general were not related to impairments in cognitive control, but interestingly (2) depressive rumination was associated with impaired inhibition of non-emotional material. The results of this study are of importance to the research investigating the underlying mechanisms of the cognitive control impairments and mood regulation problems in rumination and depression. These findings are discussed in turn below.

When comparing dysphoric to non-dysphoric individuals, the results showed no differences on correct antisaccade lantency for the inhibition as well as switching function. The lack of effects on inhibition and switching is in contrast with previous studies where cognitive control impairments have been related to depression (Joormann et al., 2007). However, impairments in cognitive control have been mainly observed when processing

emotional material, whereas in our study non-emotional material was processed. Moreover, our findings do not rule out the possibility that general impairments in cognitive control are present in severely depressed individuals. Additional research should be conducted with clinically diagnosed individuals. Our results did show an interaction between Block Type and Group for saccade error rates. Further exploration of this interaction effect showed that there was a difference in error rates between single and mixed blocks for both groups with this effect being greater in the non-dysphoric compared to the dysphoric individuals. The lack of depression-related differential effects on antisaccade accuracy is inconsistent with previous findings (Derakshan et al., 2009). Derakshan et al. (2009) used the antisaccade task with emotional and neutral facial expressions as cues and found that dysphoric individuals had higher error rates in response to emotional faces. One possible explanation for the lack of effects on error rates in our study could be the absence of emotional information. The findings on error rates require further investigation.

An important aim of the present study was to investigate the relation between rumination, a specific core cognitive feature of depression, and cognitive control. Several theorists have argued that deficits in cognitive control are related to individual differences in the tendency to ruminate (Davis & Nolen-Hoeksema, 2000) and this cognitive feature of depression could be more proximally related to cognitive control impairments than the broad construct of depression (Koster et al., in press). As hypothesized, high ruminators showed slower antisaccade latencies than low ruminators, indicating impaired inhibitory control. On the other hand, in contrast to hypotheses, antisaccade error rates were the same for both groups. Importantly, inhibition impairments were predicted by ruminative brooding, but not reflective pondering. Consistent with Whitmer and Banich's (2007) findings, switching impairments were not associated with depressive rumination in the present study. However, it is noteworthy that depressive rumination was associated with a switching impairment in a

recent study that included emotional material (De Lissnyder et al., 2010). Clearly, future studies should further examine this discrepancy in relation to emotional and non-emotional tasks.

Interestingly, our findings imply that there is an association between a specific core cognitive feature of depression, rumination, and impaired inhibition. The present study improves over the previous literature which has mainly relied on manual reaction time tasks that can be influenced by retarded responding in depression (Joomann et al., 2007). There are several interesting theoretical and clinical implications of these findings. First, the data provide empirical evidence to support the idea that reduced inhibition is associated with rumination. This study adds to a growing literature showing that cognitive control plays an important role in rumination (see Koster et al., in press, for a review). In addition, the finding underscores the importance of fine-grained analysis of facets of cognitive control. At a broader level these data show the link between information processing characteristics and thinking styles. Second, the observation that rumination but not dysphoria is related to impaired inhibition provides interesting information about the role of cognitive control in depression. That is, if cognitive control would serve as a proximal risk factor for depression one would predict the presence of impaired control in relation to depressive symptoms in general. Instead, our data indicate that cognitive control is linked to rumination, a specific cognitive symptom in depression. Cognitive control and rumination may contribute to the affective core symptoms of depression such as anhedonia, sustained negative affect and problems in emotion regulation (Joormann et al., 2007). Thus, cognitive control impairments and rumination may in interaction lead to the development of depressive episodes (De Raedt & Koster, 2010). Third, from a neuroscientific point of view there is research that elucidates the neural circuitry function of emotion regulation (e.g. Ochsner, Bunge, Gross & Gabrieli, 2002), with an important role of the DLPFC to initiate emotion regulation leading to the

inhibition of emotion producing systems, via other frontal regions such as the ventromedial prefrontal cortex (Davidson et al., 2002), regions that are also implied in suppressing saccadic eye movements (Hutton & Ettinger, 2006). Impaired cognitive regulation of emotion may be associated with prolonged rumination (Koster et al., in press). However, a correlation between the cognitive control impairments and rumination cannot be used to infer the causal relation between both factors and further research should specify the functional relationship between both constructs. Finally, an improved understanding of the cognitive control mechanisms affected in rumination can be important clinically, as it allows the development of therapeutic interventions that focus on underlying cognitive control impairments when trying to improve depression.

In conclusion, the results of this study offer new insights into the association between cognitive control and depressive symptoms. The findings indicate that depressed rumination is related to impaired inhibition of non-emotional material. Future research is required to further specify the role of cognitive control and rumination in the aetiology, maintenance and recurrence of depression.

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Footnotes

[1] This distractor task consists of blotting out the letters E and F on a paper full with letters and took only five minutes. This task was administered to prevent potential emotional responses to performance difficulties on the antisaccade task (as hypothesized) to influence the questionnaires.

[2] Switching can also be examined within the mixed block by performing a trial-by-trial analysis by comparing switch (trials preceded by a different trial type) to repeat (trials preceded by the same trial type) trials (Ansari et al., 2008). The analysis within the mixed block revealed the same results as the single to mixed block comparisons but due to length considerations these analysis were not included in this manuscript.

Figure Caption

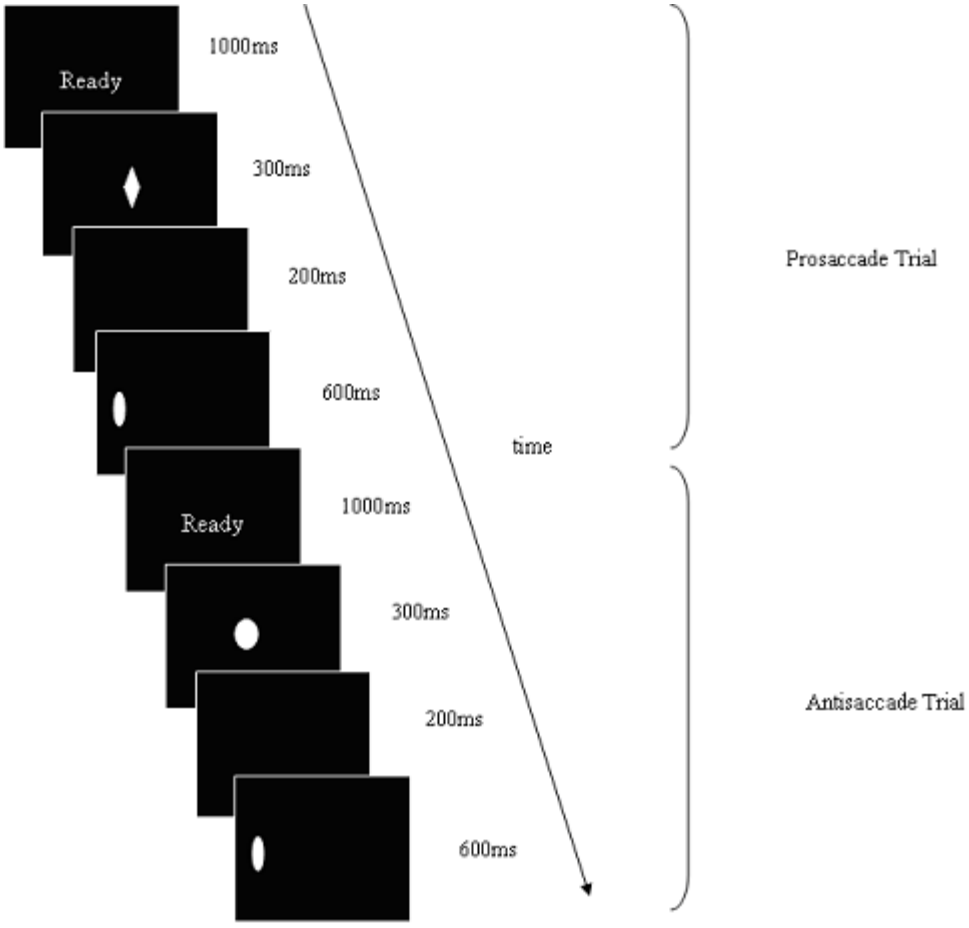

Figure 1. Anti- and prosaccade trials in the mixed block. The diamond indicates a prosaccade and the circle an antisaccade trial.

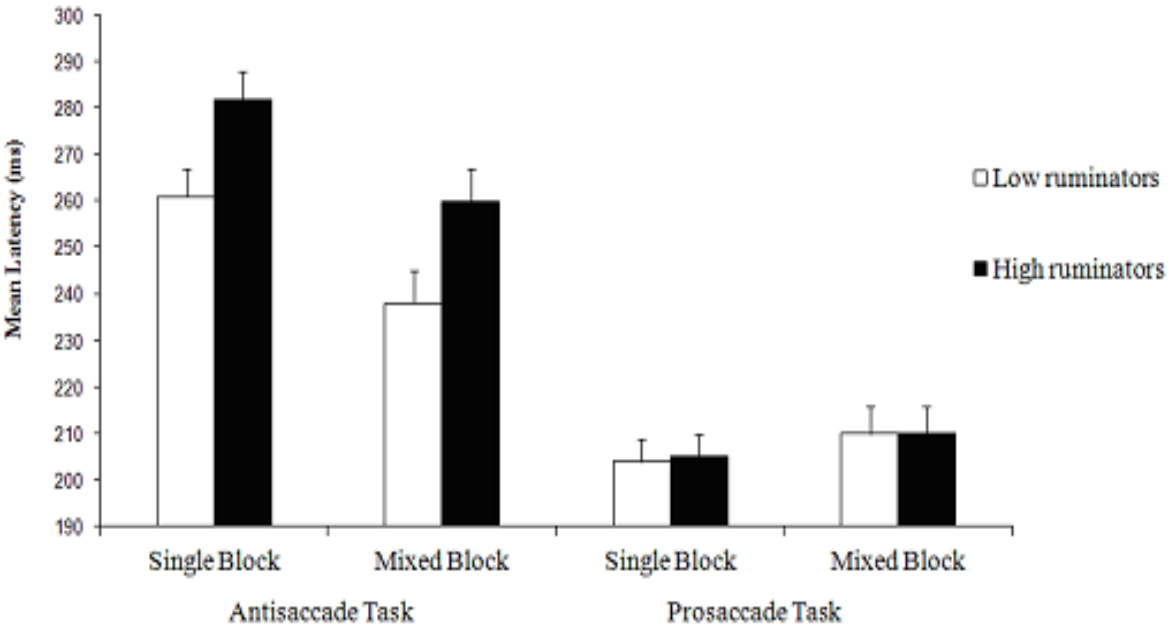

Figure 2. Mean correct saccade latencies and standard errors for the anti- and prosaccade tasks in each of the single blocks and mixed blocks as a function of rumination group.