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Rumination Mediates the Relationship between Impaired Cognitive Control for Emotional Information and Depressive Symptoms: A Prospective Study in Remitted Depressed Adults

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Abstract

Impaired cognitive control may be an important vulnerability factor for depression. Moreover, impairments in cognitive control have been proposed as a crucial process underlying ruminative thinking. The present study investigates the influence of impaired cognitive control for emotional information on rumination and depressive symptoms in a prospective design with a 1 year follow-up in a clinical sample. Thirty remitted depressed adults completed the Internal Shift Task (IST), a measure of cognitive control of emotional information, at baseline. Moreover, questionnaires measuring rumination (RRS) and depressive symptoms (BDI-II) were administered. One year later participants were contacted again and asked to complete the BDI-II and RRS. Mediation analyses showed a significant influence of impaired cognitive control for emotional information at baseline on depressive symptoms one year later, which was fully mediated by rumination. These findings underscore the importance of cognitive control abilities as a process underlying rumination and as a vulnerability factor for depression. They can stimulate translational research to improve the effectiveness of interventions that aim to decrease vulnerability by targeting cognitive control.

*Key words***:** cognitive control, rumination, depression, remission

Rumination Mediates the Relationship between Impaired Cognitive Control for Emotional Information and Depressive Symptoms: A Prospective Study in Remitted Depressed Adults

Depression is a common burdensome disorder which tends to be chronic as it is characterized by a high relapse rate. No less than 50% of depressed patients will experience more than one depressive episode even when treated with first line pharmacological and/or psychotherapeutic interventions (Kessler & Wang, 2009). Given this high relapse rate, an important research goal is to identify and understand the functional role of specific vulnerability factors underlying the recurrence of depressive episodes after remission, to support the development of new therapeutic avenues. Cognitive theories have been highly important in this regard, emphasizing both the role of negative content of thinking as well as information-processing biases as stable risk factors for depression and its recurrence (Clark, Beck, & Alford, 1999).

Cognitive control impairments are considered an important vulnerability factor for depression (for a review, see Joormann & D'Avanzato, 2010). Several researchers have already associated depression with deficits in cognitive control (Hertel, 1997; Gotlib & Joormann, 2010). Moreover, it has been stated that these deficits are not only present during depression, but that impairments in cognitive control after remission would be predictive of future depression (Joormann & D'Avanzato, 2010). The idea is that because of cognitive control problems, vulnerable people can’t disengage from negative thoughts, causing depressive rumination, which in turn enhances depressive symptoms (De Raedt & Koster, 2010). This study will define cognitive control as the ability to switch between and update information in working memory. Joormann (2010), for instance, proposed that cognitive inhibition is a key mechanism in the ability to regulate emotions. In her view, not the initial activation of negative cognitions, but the ability to regulate these negative cognitions in order

to repair negative mood determines the risk of becoming depressed. Specifically, efficient functioning of attentional control over emotional information held in working memory could be impaired, which may explain the inability to regulate negative thought content and cause rumination (see Koster, De Lissnyder, Derakshan, & De Raedt, 2011). With regard to recurrent depression, there are indeed some indications that cognitive control is reduced with experiencing one or more depressive episode(s) (Vanderhasselt & De Raedt, 2009). However, most of the work in this area is cross-sectional and there is little longitudinal research about the dynamic interplay between cognitive control and negative cognitions in the prediction of recurrent depression.

With regard to negative cognitions, the concept of rumination is highly important in depression. Rumination has been put forward as one of the important underlying vulnerability factors for depression (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). According to the response styles theory (Nolen-hoeksema, 1991), rumination can be considered as a response strategy that consists of persistent focusing of attention on depressive affect, symptoms, and their consequences. Previous studies have shown that this trait-like response style has several detrimental effects (for a review, see Nolen-Hoeksema et al., 2008). Among others, rumination has been associated with onset, severity, as well as duration of depression. These detrimental effects necessitate a better understanding of the underlying cognitive processes of rumination.

There is emerging insight into the relation between cognitive control and rumination. According to Watkins and Brown (2002), repetitive negative thoughts deplete cognitive resources. Research has provided support for this idea, showing that rumination diminishes cognitive resources and affects task performance and problem solving (Watkins, 2008). However, several authors proposed that impairments in cognitive control may also contribute to the tendency to ruminate (De Raedt & Koster, 2010). Cognitive control functions serve as

an important mechanism determining the content of working memory and removing information that is no longer relevant. Accordingly, decreased cognitive control mechanisms lead to difficulties in disengaging internal attention away from irrelevant negative information, which contributes to depressive rumination (Koster et al., 2011). A recent study confirms the association between rumination and impairments in cognitive control functions, especially concerning negative information in working memory (De Lissnyder, Koster, & De Raedt, in press).

Recently, a new paradigm was developed for measuring impaired cognitive control, the Internal Shift Task (IST; Chambers, Lo & Allen, 2008). The benefit of this task is that it allows investigating *internal attention,* in contrast to other tasks which mainly examined cognitive control for externally presented stimuli (cf. Chun, Golomb, & Turk-Browne, 2011). The IST was modified to investigate cognitive control by examining the ability to switch internal attention between both emotional and non-emotional mental representations in working memory (De Lissnyder et al., in press). In this task, a larger switch cost between mental representations reflects impaired cognitive control. The IST has shown to have high internal consistency as well as test-retest reliability (De Lissnyder, Koster & De Raedt, 2011). As depression is characterized by interpersonal difficulties (Gotlib & Hammen, 2002), faces were selected as stimuli instead of words. More specifically, angry faces were chosen because of their direct relevance to depression, which is characterized by fear of social rejection (Barnett & Gotlib, 1988). Previous research has shown an association between depression and attentional bias towards angry faces (for a review see Bistricky, Ingram & Atchley, 2011). The use of these stimuli is in line with our former studies measuring attentional control (e.g. De Lissnyder, et al., in press). The IST was used in this study to investigate the predictive power of impairments in cognitive control in a remitted depressed sample. This study adds to the existing literature, investigating the crucial question whether

cognitive control during an euthymic state can predict future levels of depressive symptoms. Therefore we selected a high-risk sample of recovered depressed individuals (Kessler & Wang, 2009), in which the results of the cognitive control task cannot be caused by a negative mood state during a current depressive episode. In this way, we can test whether underlying cognitive control problems might be a vulnerability factor that can also be measured after recovery.

The aim of the current study was to examine whether impaired cognitive control predicts future depressive symptoms in a remitted depressed sample, mediated by rumination. In non-clinical samples, there are two recent prospective studies showing that individual differences in controlling interference from negative information predict rumination (De Lissnyder, et al., 2012) and the maintenance of depressive symptoms (Zetsche & Joormann, 2011). However, in remitted depressed adults such studies are lacking. Based on cognitive theories of rumination and recurrent depression (De Raedt & Koster, 2010; Joormann, 2010), we hypothesized that impaired cognitive control, as measured by the IST, would be able to predict depressive symptoms at a one year follow up. We specifically expected that the relationship between cognitive control and depressive symptoms would be mediated by rumination. Moreover, we expected to find this only for impairments in switching away from emotional information, more specifically in switching from angry to neutral faces, as previous research suggests specific difficulties to disengage from negative information (e.g. Leyman, De Raedt, Schacht & Koster, 2007).

Method

Participants

Participants were recruited through advertisement in community papers. Based on a screening using the MDQ and the MINI (see further), which allowed excluding participants with a current depressive episode or other axis I disorders (except for anxiety disorders), 30

eligible remitted depressed adults were invited to participate in the study. At baseline, participants had, on average, experienced 2.26 depressive episodes (*SD*= 1.39). The screening showed that 43% of our sample was taking anti-depressant medication and 38% had a comorbid anxiety disorder. One year after initial participation, 23 participants (inclusion rate 76%; 16 women, 7 men) ranging in age from 25 to 56 (*M*= 47.22, *SD*= 8.14) completed the follow up.

Materials

Depressive symptoms. First, all participants were screened using the Dutch version of the Mini International Neuropsychiatric Interview (MINI). This structured interview assesses current and lifetime psychiatric disorders based on DSM-IV criteria in a valid and reliable way (Sheehan et al., 1998).

The Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996) was used to measure the presence and the degree of depressive symptoms. This 21-item questionnaire assesses the severity of a range of affective, somatic and cognitive symptoms of depression on a scale ranging from 0 to 3. Participants completed the Dutch version (BDI-II-NL), which has acceptable reliability and validity (Van der Does, 2002).

The Major Depression Questionnaire (MDQ; Van der Does, Barnhofer, & Williams, 2003) is a questionnaire based on the DSM-IV criteria of depression which investigates both current as well as past depressive symptoms. The MDQ is a validated questionnaire (Williams, Van der Does, Barnhofer, Crane, & Segal, 2008) which allows indexing past depressive episodes.

Rumination. The Ruminative Response Scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) is a self-report questionnaire to investigate rumination. The validated Dutch adaptation, the RRS-NL, consists of 26 items (Raes & Hermans, 2007). Participants indicate how often they engage in responses to a depressed mood that are focused on the self,

symptoms, or consequences of the depressed mood using a four-point Likert scale ranging from 1 (*almost never*) to 4 (*almost always*).

The Internal Shift Task. The IST was modified to examine the ability to switch attention between items in working memory in response to emotional and non-emotional facial expressions (De Lissnyder et al., in press). The Internal shift task was programmed using E-prime 2.0 software package.

The stimuli were colored pictures of faces selected from the Karolinska Directed Emotional Faces (KDEF). This selection was based on intensity and arousal ratings from a validation study by Goeleven, De Raedt, Leyman and Verschuere (2008) and included 24 neutral (Intensity: *M*= 5.15, *SD*= .37; Arousal: *M*= 2.48, *SD*= .23) and 24 angry (Intensity: *M*= 6.36, *SD*= .71; Arousal: *M*= 3.87, *SD*= .58) faces. All pictures were cut to exclude background interference (hair, clothing) and were adjusted to the same size (326 x326 pixels).

All participants performed a non-emotional task version in which they were instructed to focus on *gender* (male or female categorization) and an emotional task version, in which they were asked to focus on *emotion* (neutral or angry categorization). Importantly, the stimuli of both tasks were identical: angry and neutral faces. Each task version consisted of 3 practice blocks and 12 consecutive experimental blocks. The order of both task versions was counterbalanced (N= 13 for gender first, N= 10 for emotion first).

Faces were presented at the centre of the screen (a 75Hz, 17-inch colour monitor) one at a time. Participants were instructed to keep a silent mental count of the number of items in each category (male versus female in the gender task version/angry versus neutral in the emotion task), presented over a block of items (with at random 10 to 14 items). Each time a face was presented, they were asked to press the spacebar as quickly as possible to indicate that they had updated their mental count and had retained the correct number of items of each category in working memory. Upon responding, the next face appeared after a 200ms inter-

trial interval. At the end of each block, participants were asked to report the number of items in each category in a fixed order to encourage a consistent counting strategy. Figure 1 depicts an example of a block of items during the task. The reaction times (in ms) for the key-presses after each picture were recorded and used as the main variables in the analyses. Due to the sequence of the faces, each block of items contained both switch and no-switch trials (random order).

Procedure

At baseline, participants completed the IST. Subsequently, self-report questionnaires measuring depressive symptoms and rumination were administered. This order of testing prevented priming of mood by the questionnaires that may subsequently influence IST performance. One year later participants were contacted again through e-mail and asked to complete the questionnaires online.

Data-analytic Strategy

All analyses were based on the 23 participants who completed the whole experiment. To investigate mediation, the indirect effect of the initial variable (cognitive control measures) on the outcome variable (BDI-II) through the mediating variable (RRS-NL) was tested using a stepwise approach with multiple regression analysis. To assess the indirect effect, a bootstrapping method was used with 5000 estimates, as suggested by Preacher and Hayes (2008). This procedure helps to circumvent statistical problems of testing for mediation in smaller sample sizes.

Results

Data Preparation

The median reaction times per category over all blocks of the IST were calculated and used for the analyses. Note that for the emotion task, we performed analyses on the specific

shift trials (angry-neutral, neutral-angry) because previous research suggests specific difficulties to disengage from negative information (Koster et al., 2011). Table 1 gives an overview of the mean, standard deviations and range of all measures of the task. The use of median scores reduces the influence of outliers on the data. Both correct and incorrect blocks of items were included in the analyses (cf. Chambers et al., 2008). A recent study showed that reliability of the IST remains very high when both blocks with a correct and incorrect count are included (De Lissnyder, et al., 2011). Overall, participants were very accurate in categorising the number of faces per block, *M*= 83.04%, *SD*= 15.89. Moreover, participants were as accurate in the emotion task version as in the gender task version, $t(22)=.03$, $p=.98$.

Depressive Symptoms and Zero-order Correlations

At initial assessment, participants reported a mean RRS score of 51.78 (*SD*= 9.99, range 32 to 73) and a mean BDI-II score of 7.52 (*SD*= 6.58, range 0 to 20). One year later, participants reported a mean RRS-NL score of 46.57 (*SD*=15.64, range 26 to 76) and a mean BDI-II score of 11.61 (*SD*= 10.85, range 0 to 35). Whereas only one participant scored above the clinical cut-off score of 19 on the BDI-II at initial assessment, 34.8% scored higher than this cut-off one year later.

To check whether we should control for correlations between variables at baseline, Pearson correlations between IST performance and initial depressive symptoms and rumination respectively were calculated, but no significant correlations were found (all *p*s>.11). Table 2 shows zero-order correlations between all variables.

Given that all participants were selected on low BDI-II and that BDI-II scores at time 1 were not correlated with the other measures at time 1, BDI-II at time 1 was not used as a covariate in the analyses.

Rumination as Mediator between Cognitive Control and Depressive Symptoms at Follow Up

First, we tested the direct effect from indexes of cognitive control on depressive symptoms. Shift angry-neutral predicted depressive symptoms at follow up at a marginally significant level, β=.38, $t(22)= 1.86$, $p=.08$. Similar but significant results were yielded for shift neutral-angry and later depressive symptoms, β=.46, $t(22)= 2.34$, $p=.03$. It has been argued that this first step is not a necessary requirement for mediation (Kenny, Kashy & Bolger, 1998), so we proceeded with mediation analyses.

In the second step, crucial for mediation, we tested if the two shift indices predicted rumination. The analysis showed that both shift angry-neutral, β=.41, $t(22)= 2.07$, $p=.05$ and shift neutral-angry, β=.58, $t(22)= 3.22$, $p< .01$ were significantly related to rumination at follow up.

The third step in the mediation analyses showed that rumination was significantly associated with depressive symptoms, when controlling for both shift angry-neutral, β=.74, $t(22)= 4.78$, $p< .01$, as shift from neutral to angry, β=.76, $t(22)= 4.39$, $p< .01$.

A final step showed that the association between shift angry-neutral and BDI-II was not significant, β=.07, $t(22)= .45$, $p=.66$, when controlling for the mediating RRS-NL, which is indicative of full mediation. Similar results, β=.02, $t(22)= .10$, $p=.92$, were obtained for the mediation model with shift neutral-angry as initial variable.

To further examine whether the indirect effect was significantly different from 0, an accelerated-bias-corrected bootstrapping (Preacher & Hayes, 2008) was used. This method produces a confidence interval for the indirect effect, which may not include 0 to indicate a significant mediation path. Results indicated that the indirect effect of both shift angry-neutral, 95% CI[.001, .017], S.E.=.004, $p<.05$, as shift neutral-angry, 95% CI[.004, .021], S.E.=.004, $p< .05$, on depressive symptoms were significant. These results showed full mediation of the relationship between cognitive control and depressive symptoms by rumination.

The investigated meditational model was performed using longitudinal data, which was only partially structured to be temporally consistent with the hypothesized process, because follow up RRS-NL and BDI-II were measured at the same time. To control for the alternative mediation path, the data were subjected to an analysis in which the depressive symptoms acted as the mediator between cognitive control and rumination. If the results of this reversed model are also significant, caution is warranted. The indirect effect of the alternative model with shift angry-neutral was not significant as the confidence interval for the indirect effect produced by the bootstrapping method did include zero, 95% CI[-.002, .026], S.E.=.008, p>.05. However, the alternative model with shift neutral-angry did show a significant mediation of BDI-II for RRS-NL, 95% CI[.001, .023], S.E.=.006, p< .05.

Emotion specificity

To investigate the emotion specificity effect, we also analysed the mediation model using shift between gender information in the gender task as initial variable. It is important to mention that exactly the same stimuli were used in this task, but the response criterion was gender as compared to emotion. This alternative model did not meet the requirements of the second step of mediation analysis (p> .05), so this model was not investigated further. These results indicated that not cognitive control in general, but specifically cognitive control over emotional information has an important influence on rumination and depressive symptoms.

Discussion

This study used a prospective design with a one year follow up to examine the hypothesis that impaired cognitive control predicts depressive symptoms in a remitted depressed sample. More specifically, we investigated if this relation was mediated by rumination. In line with theoretical predictions, the main findings show that cognitive control was predictive of depressive symptoms one year later, and rumination plays an important role as it fully mediates the relation between cognitive control and depressive symptoms.

The finding that impairments in cognitive control at baseline were predictive of the level of depressive symptoms one year later, mediated by rumination, are in line with the hypothesis based on the theory by De Raedt and Koster (2010) which states that reduced cognitive control leads to increased vulnerability for depression after recurrent episodes. Previous research using cross-sectional designs had already shown evidence for an association between diminished cognitive control and depression (for a review, see Joormann & D'Avanzato, 2010). The current study adds to this literature in showing that impairments in cognitive control in a sample of remitted depressed adults are predictive of later levels of depressive symptoms. Provided the important problem of recurrence after treatment, insight into the risk factors predicting recurrent symptoms of depression is of crucial importance.

The most crucial finding is that, based on the mediation analyses, rumination plays a crucial role as mediator between cognitive control and depressive symptoms. Research already provided some indication that cognitive control is predictive of rumination in healthy students under stress (De Lissnyder et al., 2012) and the maintenance of depressive symptoms in a healthy population (Zetsche & Joormann, 2011). Although we mainly expected to find an influence of shift from angry to neutral information, the other emotion condition from neutral to angry was also significant. This might indicate that both impaired shifting from and towards emotional information can be predictive of depressive symptoms, which would point to valence a-specificity of switching. However, only the effect of the shift from angry to neutral was no longer significant in the alternative analysis in which the mediator and outcome variable are reversed. In a mediation model where the mediator and the outcome variable are measured at the same moment in time, it is important to ascertain whether the model is only valid in the hypothesised direction. The unexpected effect of shift from neutral to angry was still significant for the reversed model, showing that we need to be cautious with statements about the valence a-specificity. Moreover, results showed that in the prediction of

later levels of depressive symptoms, emotion specific cognitive control was more important than general cognitive control. That is, rumination did not mediate the relationship between shifting from gender information and depressive symptoms in the gender task, whereas it did mediate for shifting between emotional information and depressive symptoms in the emotion task.

These findings are both of theoretical as well as of clinical importance. At the theoretical level, these results provide partial confirmation of the model proposed by Koster et al (2011), although there are some indications that shifting from neutral to negative information may also play a role. This model states that impairments in cognitive processing create difficulties in disengaging attention away from negative information which contributes to rumination and ultimately leads to depression. Our results are also in line with the notion that especially emotion specific impairments in cognitive control are predictive of recurrent depressive symptoms rather than general cognitive control (cf. De Raedt & Koster, 2010; Joormann, Yoon, & Zetsche, 2007). Clearly, given the correlational data obtained here, we cannot rule out the possibility of third variables and therefore cannot provide conclusive evidence for a causal relation between cognitive control and later levels of depressive symptoms. However, the current study provides an important impetus to conduct experimental studies manipulating cognitive control (see below).

At the clinical level, relapse into depression provides an important challenge as current pharmacological and therapeutic treatments seem to be insufficiently capable to prevent recurrent depression. The findings that impairments in cognitive control play a crucial role in predicting later level of depression leads to a promising prospective for targeting cognitive control in clinical interventions. Possible procedures for strengthening cognitive control are already being used. For example, Mindfulness based cognitive therapy (MBCT) has been demonstrated to be effective in prevention of relapse into depression and research has

suggested that this protective effect might be based on changes in cognitive control (Chambers et al., 2008). Also procedures that more directly target underlying cognitive processing, like cognitive training and cognitive bias modification (Baert, Koster & De Raedt, 2011), where cognitive control can be strengthened through computerized training programs can potentially reduce depressive symptoms as well as rumination (Siegle, Ghinassi, & Thase, 2007).

Some limitations to the current study can be noted. First, due to specific inclusion criteria and the long term follow up, the sample size is rather small. Nevertheless, the fact that this mediation effect could already be observed with this sample is indicative of strong associations between the different constructs. Moreover, it is noteworthy that the bootstrapping procedure has been shown to be reliable in small samples (Preacher & Hayes, 2008). Unfortunately, this sample size did prevent more fine- grained analyses on other potentially interesting variables (e.g., the role of the number of episodes). Second, distinct components of rumination (i.e., brooding and reflection) could not be examined in this small sample. Third, the lack of correlations between cognitive control, rumination, and depressive symptoms at baseline measurement might be surprising. However, it is likely that this results from our selection procedure, as only participants without a current depressive episode were invited to participate. Subsequently, this selection limited the variability in our self report measurements, which might have diminished chances of finding significant correlations at baseline. Moreover, Zetsche and Joormann (2011) were also unable to find significant correlations at initial assessment.

To summarize, this study found that cognitive control is predictive of later level of depressive symptoms, mediated by rumination. This study is, to our knowledge, the first to show this in a remitted depressed sample. Our results underscore the crucial role of cognitive

control in the relapse of depression which offers a promising avenue for improving clinical interventions.

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Figure 1. An example of a block of items during the Internal Shift Task.

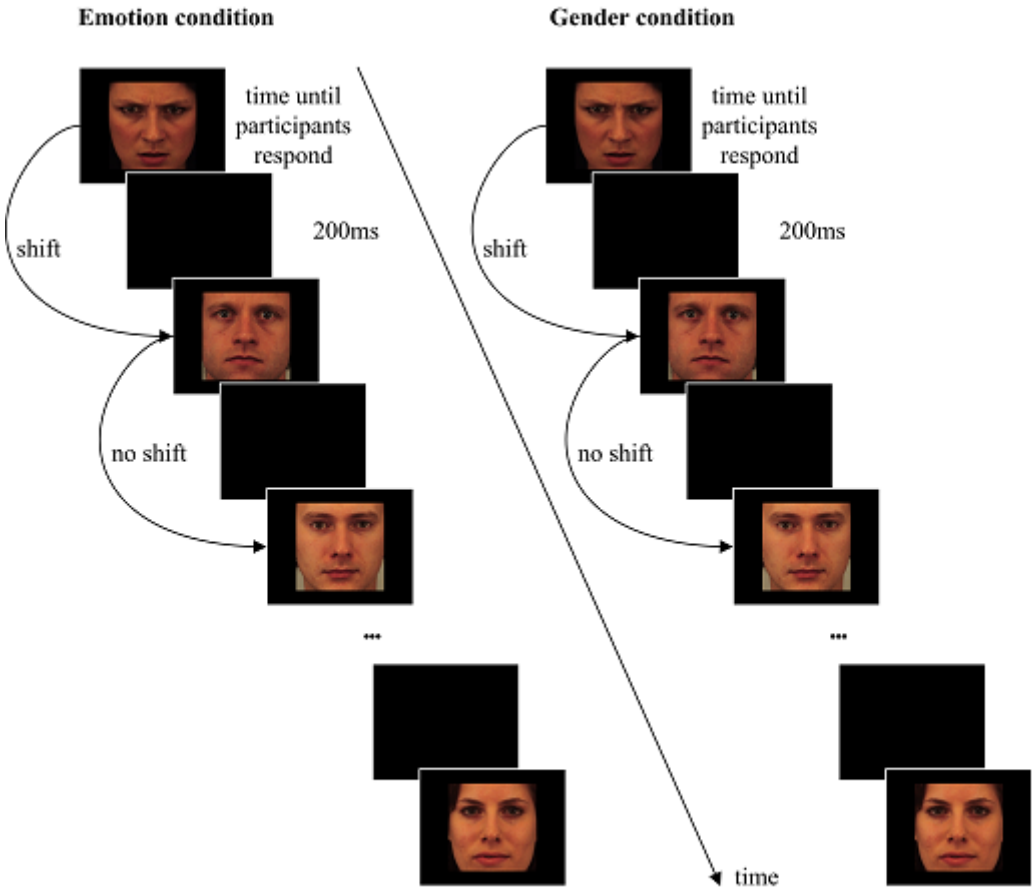

Table 1.
Mean scores, standard deviations and range for the measures of the Internal Shift Task.

	Mean	SD	Minimum	Maximum
Emotion task				
Angry-angry	1303	360	709	1814
Neutral-neutral	1354	367	659	1999
Angry-neutral	1698	442	643	2491
Neutral-angry	1562	420	687	2598
Gender task				
No shift gender	1264	302	542	1739
Shift gender	1542	403	544	2308

Table 2.
Zero-order correlations.

	BDI (time 1)	RRS (time 1)	shift cost angry to neutral	shift cost neutral to angry	shift cost gender	BDI (time 2)	RRS (time 2)
BDI (time 1)	-						
RRS (time 1)	-.16	-					
shift cost angry to neutral	.24	-.07	-				
shift cost neutral to angry	.04	-.04	.80**	-			
shift cost gender	.26	-.17	.51**	.23	-		
BDI (time 2)	.61**	.01	.38	.45*	-.04	-	
RRS (time 2)	.47**	-.12	.41*	.57**	-.07	.77**	-

* Correlation is significant at the .05 level (2-tailed).
** Correlation is significant at the .01 level (2-tailed).