EXECUTIVE DYSFUNCTION, MEDIATED BY RUMINATION, ANTICIPATES INCREASES IN DEPRESSIVE SYMPTOMS

BY

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THESIS

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ABSTRACT

Depression is associated with disruptions in cognitive processes as well as in affect. Executive function (EF) deficits are among the most common cognitive disruptions associated with depression, but the causal relationships between EF and depression are unclear. EF deficits may exacerbate symptoms and hence may play a role in the development, maintenance, or recurrence of depression. In turn, depression may disrupt EF. The present study examined whether EF prospectively predicted worsening of depression symptoms. Shifting, inhibition, and working memory (WM) aspects of EF were assessed in relation to anhedonic depression change scores in 51 participants with a range of risk for depression. Since rumination has previously been associated with worse EF and depressive symptoms, rumination was tested as a mediator of the association between EF and depression change scores. Taken together, analyses indicated that poorer WM at time 1, but not shifting or inhibition, predicted an increase in depressive symptoms. When a mediation analysis was conducted with rumination as a mediator, the association between WM and depression change scores was no longer significant, indicating that rumination fully mediated the relationship between WM and depressive symptoms. Findings suggest that EF influences the occurrence of depressive symptoms. In addition, rumination plays an important role in the link between WM deficits and depressive symptoms. Efforts aimed at preventing rumination or targeting ruminative processes in treatment may reduce risk for depression.

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INTRODUCTION

Investigations of cognition-emotion interactions have revealed that cognitive deficits, such as problems with memory and attention, are common during depressive episodes (Gotlib & Joormann, 2010; Levin et al., 2007). Furthermore, cognitive deficits have been found to persist beyond depressive episodes and may even contribute to relapse (Austin et al., 2001). Levin et al. (2007) suggested that at least some of the cognitive impairments and biases associated with depression are likely due to specific executive function (EF) deficits. EF has been defined generally as cognitive control and the set of processes by which individuals "effortfully guide behavior toward a goal" (Banich, 2009).

Although not always considered in studies of depression and anxiety, EF is not a unitary phenomenon. Current theorizing suggests that it is comprised of multiple related but separable components, including (at minimum) the ability to shift attention from one task or mental set to another (shifting), the ability to override or suppress dominant or automatic responses (inhibition, possibly not separate from a more general component accounting for variance in common across all types of EF tasks), and the ability to actively maintain and/or manipulate information during a brief time period (working memory, WM: Miyake et al., 2000; Miyake & Friedman, 2011). Deficits in each of these EF components have been associated with depression using neuropsychological measures (Snyder, 2012) and behavioral tasks (Joormann & Gotlib, 2008; Rose & Ebmeier, 2006). Additionally, depression has been associated with abnormal neural activity in brain regions that implement these EF components (Engels et al., 2010; Herrington et al., 2010; Rogers et al., 2004).

EF deficits not only may contribute to cognitive deficits in depression (Levin et al., 2007) but may interfere with recovery from negative mood (Gotlib & Joormann, 2010). An inability to disengage from negative material may play a role in the chronicity and severity of depression. For example, clinically depressed individuals had greater switch costs during a mental counting task that incorporated affective words than did healthy controls (Lo & Allen, 2011). Difficulties switching attention away from affective material may lead to a more prolonged focus on negative material, which may increase or maintain negative affect and thus may be associated with the maintenance of or increases in depressive symptoms. Individuals with depression have also been found to have difficulties preventing or inhibiting mood-congruent material (e.g., negative material) from entering WM (Gotlib & Joormann, 2010). Problems inhibiting negative material might lead to the maintenance of or increases in depressive symptoms over time as well. Furthermore, deficits in maintaining and manipulating information in WM have been found in individuals with major depressive disorder (Rose & Ebmeier, 2006). An inability to maintain and manipulate task-relevant information may interfere with the ability to complete tasks in daily life, which could be distressing and hence foster depression.

In individuals at risk for depression, EF deficits could play a role in precipitating a depressive episode or in increasing cognitive and emotional processes that contribute to depression risk. Crocker and colleagues (2012) found that trait negative affect, a well-established risk factor for depression, was associated with decreased activity in brain regions involved in top-down control of attention in individuals with a range of depressive symptoms when confronted with emotional distraction. In addition, trait negative affect disrupted top-down control of attention on a non-emotional, color-word Stroop task in a nonclinical sample but only in a negative affective context (Hur et al., submitted).

Because the relationship between EF deficits and depressive symptoms has typically been investigated cross-sectionally, it is unclear whether EF deficits are associated with an increase in depressive symptoms over time and which precedes and may cause the other. The present study investigated whether executive dysfunction anticipates increases in depressive symptoms. EF deficits were assessed with a well-established self-report measure that provides information on a variety of functional impairments that individuals experience in their everyday lives. Laboratory behavioral EF tasks can be limited in terms of their ecological validity by providing more structure and support for focus on a particular task than occurs in typical real-life settings. In the present study, executive function and depressive symptoms were both assessed at an initial screening session (T1), and depressive symptoms were measured again 3 months later (T2). It was hypothesized that worse EF in each component (shifting, inhibition, WM) at T1 would be associated with increases in depressive symptoms from T1 to T2.

Finally, although deficits in EF may contribute directly to increases in depressive symptoms, it is also possible that an intervening variable mediates the relationship between EF deficits and depressive symptoms. One likely candidate is rumination, as deficits in EF have been associated with higher levels of rumination (Davis & Nolen-Hoeksema, 2000; Joormann & Gotlib, 2008). Rumination is defined as a repetitive, passive focus on one's "symptoms of distress" and has been associated with maintenance and exacerbation of depressive symptoms (Nolen-Hoeksema, 1991). Difficulties shifting attention away from negative ruminative thoughts, preventing ruminative thoughts from entering WM, or clearing ruminative thoughts from WM could increase depressive symptoms. Since increased rumination has been linked to EF deficits, it was hypothesized that rumination would mediate the relationship between EF deficits and in depressive symptoms.

METHODS

Participants

Fifty-two participants (27 female) were recruited based on the Positive Affect and Negative Affect scales from the Positive and Negative Affect Schedule-X (PANAS-X; Watson & Clark, 1994) from a pool of 756 undergraduates at the University of Illinois at Urbana-Champaign. Participants were contacted if they 1) scored at or above the 80th percentile on one of the dimensions and at or below the 50th percentile on the other dimension, or 2) scored at or below the 50th percentile on both dimensions. One recruited participant was excluded from the present study due to missing data. Of the 51 subjects included in this study, 16 had high positive affect, 20 had high negative affect, and 15 had positive and negative affect scores at or below the 50th percentile. For the purposes of the present study, the goal of the recruitment was to obtain a range of individuals at varying risk for depression. Positive and negative affect scores on the PANAS were not targets of interest in present analyses, and PANAS grouping was not utilized. Participants were compensated with course credit for the initial screening session (T1) and were financially compensated for the follow-up session (T2) approximately 3 months later (M= 96 days, SD=60 days).

Questionnaires

At T1 and T2, participants completed the 22-item Anhedonic Depression subscale of the Mood and Anxiety Symptom Questionnaire (MASQ-AD; Watson, Clark et al., 1995; Watson, Weber et al., 1995). The MASQ-AD allows both categorical and dimensional analytic strategies. For the present study, a dimensional approach was used to assess changes in depressive

symptoms. Change scores were calculated by subtracting T1 from T2 MASQ-AD scores. A positive change score represented an increase in depressive symptoms over time.

Participants also completed the 75 -item Behavior Rating Inventory of Executive Function - Adult Version (BRIEF-A; Roth, Isquith, & Gioia, 2005) at T1. The BRIEF-A is intended to assess EF over the past 6 months in an ecologically valid manner. The measure provides scores for 9 subscales measuring different EF components. For the present study, the Shift (6 items), Inhibit (8 items), and Working Memory (8 items) subscales were used to assess the three EF components discussed above. Higher Shift, Inhibit, and Working Memory scores represent worse EF.

Rumination was measured at T2 using the Rumination subscale of the Rumination-Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999). According to the RRQ, rumination is characterized as chronic self-focus, often involving recurrent negative thinking about the past (e.g., “I spend a great deal of time thinking back over my embarrassing or disappointing moments”; Trapnell & Campbell, 1999).

RESULTS

Table 1 reports means and standard deviations for the EF components and depressive symptoms at T1 and T2. Depressive symptoms increased for 10 individuals, stayed the same for 1 individual, and decreased for 40 individuals. Individuals whose MASQ-AD score increased vs. decreased did not differ in MASQ-AD score at T1 (t (50) = 0.69).

Zero-order correlations revealed that the Shift, Inhibit, and Working Memory scores were each positively correlated with MASQ-AD scores at T1 and T2 (see Table 2). Thus, as predicted, higher depression was associated with worse self-reported EF. Correlations between the MASQ-AD change scores and Shift, Inhibit, and Working Memory scores revealed that Working Memory and MASQ-AD change scores were positively correlated (r (51) = 0.35, $p < 0.02$), indicating that worse self-reported WM was associated with an increase in anhedonic depression over time (Figure 1). The correlations between Shift and MASQ-AD change scores and between Inhibit and MASQ-AD change scores were also in the expected direction though not significant ($p = 0.07$ and $p = 0.12$ respectively).

In order to assess whether the zero-order relationship between WM scores and depression change scores was specific to WM, a hierarchical linear regression was conducted with all three EF scores (Shift, Inhibit, and WM) as predictors. WM remained a marginally significant predictor of MASQ-AD change scores (t (50) = 1.68, $\beta = 0.33$, p = 0.10).

Taking another approach, a hierarchical regression was conducted predicting T2 MASQ-AD scores with T1 MASQ-AD scores entered in the first step and WM entered in the second step. WM scores accounted for significant variance even after taking into account initial depression levels (total $R^2 = 0.60$, $\Delta R^2 = 0.10$, *F-change* (1, 48) = 11.67, $\beta = 0.34$, $p = 0.001$).

In order to assess whether WM remained a significant predictor of T2 MASQ-AD with all three EF scores entered as predictors, three hierarchical linear regressions were run to examine the incremental contribution of each of the three EF scores withT1 MASQ-AD scores and the other two EF scores already entered. WM remained a marginally significant predictor of T2 MASQ-AD scores (t (50) = 1.89, β = 0.25, p = 0.065). In contrast, neither Shift nor Inhibit was a significant predictor (t (50) = 1.28, β = 0.16, p = 0.21 and t (50) = 0.23, β = 0.03, p = 0.82, respectively).

Next, a mediation analysis was conducted to test the hypothesis that rumination would mediate the relationship between WM and MASQ-AD change scores. Using the Process SPSS macro for testing mediation (Hayes, 2012), the direct and indirect effects were calculated (note: the Process macro provides unstandardized coefficients). As illustrated in Table 2, the zero-order effect of WM on the MASQ-AD change scores and the zero-order effect of WM on rumination were significant, which are common preconditions for establishing mediation. The effect of rumination on the MASQ-AD change scores, with WM variance removed, was also significant (B = 3.55, t (50) = 2.09, $p < 0.05$). After rumination variance was removed, the effect of WM on the MASQ-AD change score no longer approached significance (B = 0.55, t (50) = 1.26). Since the sample was relatively small, the bootstrapping method was used to measure the total indirect effect, which does not assume that the data are normally distributed. Using 5,000 bootstraps, the total indirect effect of WM on MASQ-AD change scores via rumination was significant at the 95% confidence level [0.04, 1.14] (Figure 2). This analysis was repeated, using T2 MASQ-AD scores with T1 MASQ-AD scores partialed out rather than T2-T1 MASQ-AD change scores. All results were the same as with change scores.

DISCUSSION

The present study demonstrated that executive dysfunction anticipates increases in depressive symptoms and furthermore that rumination fully mediates that relationship. Not only were EF deficits associated with higher levels of depression at an initial screening session, worse self-reported WM (a specific EF component) was associated with an increase in depressive symptoms over time, above and beyond the effects of initial depression. While prior studies have revealed associations between EF deficits and depression cross-sectionally, the present study showed that EF disruption anticipates subsequent increased depression using a prospective design. This suggests that EF deficits play an active a role in the course of depressive symptoms.

The present study also adds to previous literature by indicating that rumination is a mechanism by which executive dysfunction anticipates increases in depressive symptoms. Based on prior cross-sectional evidence that EF deficits are associated with higher levels of rumination (Davis & Nolen-Hoeksema, 2000; Joormann & Gotlib, 2008) and that rumination is associated with higher levels of depression (Nolen-Hoeksema et al., 2008), rumination was hypothesized to mediate the relationship between EF deficits and increases in depression. Using two alternate analytical approaches, rumination fully mediated the relationship between WM and depression change. Present findings suggest that WM may be the key component of EF in the relationship between EF deficits and increases in depressive symptoms and furthermore that rumination is the route by which WM exerts its effects.

Overall, rumination appears to play a central role in the relationship between EF deficits and depressive symptoms. Impairments in WM associated with depressive symptoms may be due to deficient control of attention resulting from a focus on material that is of personal concern

(Hertel, 1997), as is the case with rumination. Since rumination is an iterative focus on one's symptoms of distress and negative material, material that is being ruminated upon may consume WM resources, already associated with a limited capacity system (Miyake & Shah, 1999). An impaired ability to maintain task-oriented material in WM due to rumination may prevent non-negative material from entering WM and may also impair task completion, which could promote increases in depressive symptoms over time for individuals at risk.

It is also possible that impairments in WM disrupt other coping efforts, such as behavioral or cognitive and emotional strategies, that serve to ameliorate ruminative thoughts and depressive symptoms (Gotlib & Joormann, 2010). Although the present study did not test whether WM is associated with increases in rumination, because rumination was not measured at T1, according to Nolen-Hoeksema and colleagues (2008) rumination appears to be relatively stable in individuals. Thus, it is likely that rumination is a precursor to WM impairment, although its relationship with impaired WM could also be reciprocal (e.g., rumination leads to impaired WM, and impaired WM leads to more rumination). The nature of causality in the relationship between WM and rumination should be examined in future prospective studies.

A limitation of the present study is the use of self-report only. Although self-report can provide an indication of EF deficits in individuals' daily lives, it is subject to biases including social desirability (Furnham, 1986) or inaccurate recollection. The impact that perceived EF deficits have on individuals' daily function should be confirmed by informant reports and compared with neuropsychological and psychophysiological assessments to determine which components of EF are most predictive of depressive symptoms. In the meantime, present data suggest that there can be a causal relationship between EF deficits and subsequent symptoms of depression.

Although WM impairments are linked with increases in depressive symptoms, ruminative processes account for this relationship, at least statistically. This is important because providing people with skills to prevent rumination before negative events occur as well as strategies to cope with negative events that have occurred may reduce both WM impairment and increases in depressive symptoms. In a study by Lyubomirsky and Nolen-Hoeksema (1995), dysphoric individuals were less likely to use effective problem-solving strategies. Teaching individuals to problem-solve may prevent life challenges from becoming debilitating. Other intervention techniques such as mindfulness meditation and mindfulness-based cognitive therapy or stress reduction have been shown to be effective in reducing rumination and depressive symptoms (Jain et al., 2007; Kuyken et al., 2008) as well as increasing EF (Zeidan et al., 2011) and could be an important therapeutic technique to implement before the onset of a depressive episode, particularly in those with a ruminative style of thinking. Finally, early intervention may help prevent or reduce the development of a ruminative style of thinking, which may in turn prevent many of the cognitive deficits and other symptoms associated with depression from developing.

FIGURES

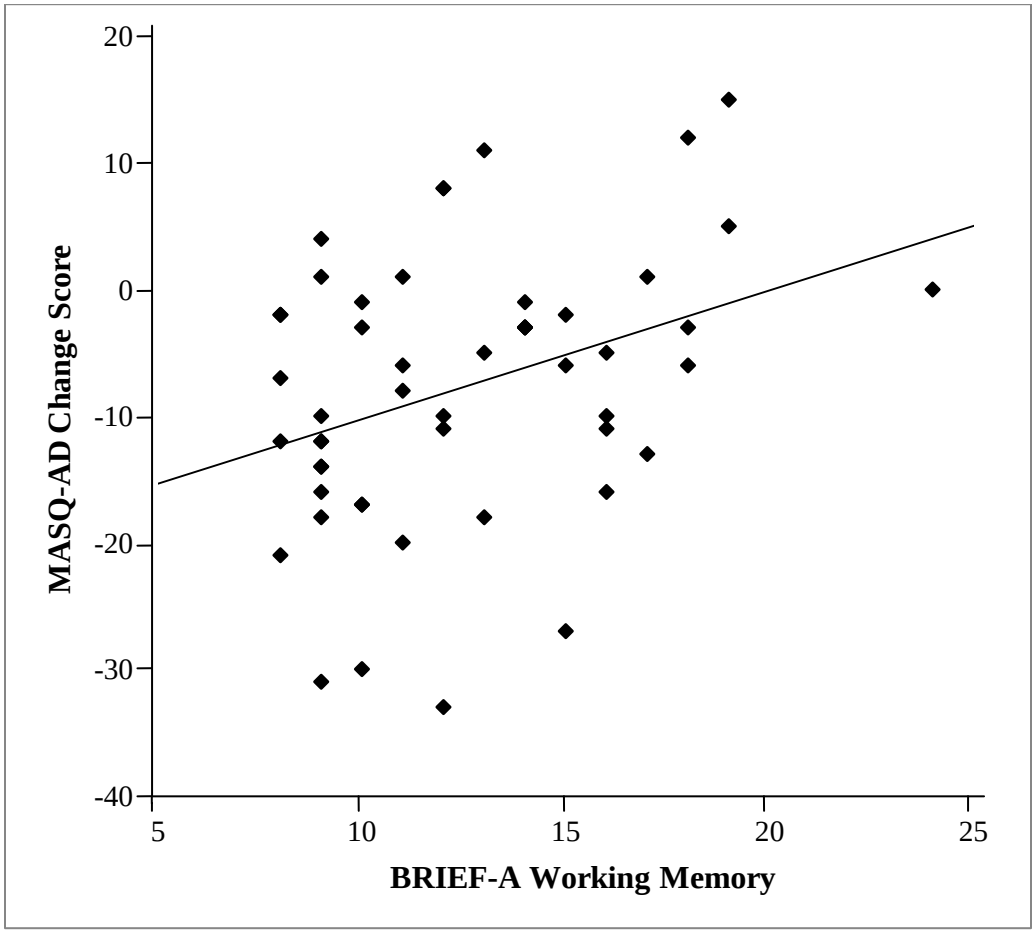
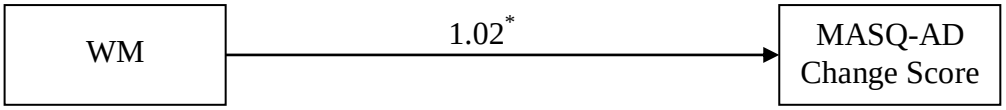


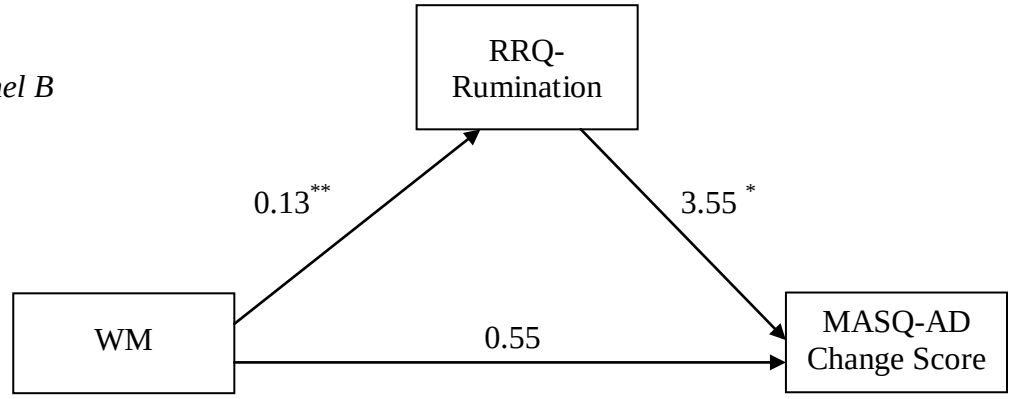
Figure 1. Scatterplot showing the association between working memory scores and anhedonic depression change scores. Higher WM scores represent worse self-reported WM, and positive MASQ-AD change scores represent an increase in depressive symptoms from Time 1 to Time 2.

Figure 2. Mediation Model of Working Memory (WM) on the MASQ-AD change score through RRQ-Rumination. Unstandardized coefficients are provided.

Panel A



Panel B



Indirect Effect: (0.13)*(3.55) = 0.46*
Note: *$p < 0.05$, **$p < 0.01$

TABLES

Table 1. Questionnaire means and standard deviations for the full sample, those who decreased in depressive symptoms from time 1 (T1) to time 2 (T2), and those who increased from time 1 to time 2.

Mean (SD)	Total (N=51)	Decreased in Depression (N=40)	Increased in Depression (N=10)	t value
Shift	9.41 (2.68)	8.9 (2.24)	10.7 (2.95)	-1.81
Inhibit	13.47 (3.03)	13 (2.64)	14.6 (3.47)	-1.36
Working Memory	12.51 (3.72)	11.88 (3.12)	13.9 (3.99)	-1.50
MASQ-AD (T1)	59.84 (12.02)	59.43 (11.92)	60.7 (13.33)	-0.28
MASQ-AD (T2)	52.14 (15.07)	47.95 (12.36)	67.3 (15.44)	3.68**
MASQ-AD Change Score	-7.71 (10.73)	-11.48 (8.56)	6.6 (5.02)	-8.67***
MASQ-AD (T2 Residuals)	0.00 (10.64)	-3.82 (8.25)	14.40 (5.60)	-8.28***
RRQ-Rumination	3.22 (0.96)	3.03 (0.90)	4.02 (0.82)	-3.34**

Note: equal variances not assumed; t-tests are between the N=40 and N=10 samples

* $p < 0.05$

** $p < 0.01$

*** $p < 0.001$

Table 2. Correlations between executive function components, MASQ-AD, and RRQ-Rumination for the full sample (N=51).

	Shift	Inhibit	Working Memory	MASQ-AD (T1)	MASQ-AD (T2)	MASQ-AD Change Score	MASQ-AD (T2 Residuals)	RRQ-Rumination
Shift	1	0.54***	0.58***	0.52***	0.59***	0.24	0.31*	0.49***
Inhibit	-	1	0.68***	0.41**	0.50***	0.24	0.29*	0.45**
Working Memory	-	-	1	0.40**	0.57***	0.35*	0.41**	0.52***
MASQ-AD (T1)	-	-	-	1	0.71***	-0.13	0.00	0.54***
MASQ-AD (T2)	-	-	-	-	1	0.61***	0.71***	0.72***
MASQ-AD Change Score	-	-	-	-	-	1	0.99***	0.41**
MASQ-AD (T2 Residuals)	-	-	-	-	-	-	1	0.49***
RRQ-Rumination	-	-	-	-	-	-	-	1

*$p < 0.05$
**$p < 0.01$
***$p < 0.001$
Note: MASQ-AD = Mood and Anxiety Symptom Questionnaire – Anhedonic Depression

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