The Role of Executive Control Deficits in Cognitive Correlates of Dysphoria

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The Role of Executive Control Deficits in Cognitive Correlates of Dysphoria

A thesis submitted in partial fulfillment of the requirement for the degree of Bachelor of Science in Psychology from The College of William and Mary

by

Christian Alexander Ledwin Bean

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The Role of Executive Control Deficits in Cognitive Correlates of Dysphoria

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College of William & Mary
Abstract

Recent research has suggested that deficits in executive control, especially impairments in cognitive inhibition, as well as rumination, negative involuntary memories, and reduced autobiographical memory specificity could play key roles in the development and exacerbation of depressive symptoms. In the present study, participants completed the Negative Affective Priming (NAP) task, the Ruminative Responses Scale (RRS), the Continuous Word Association Task (CWAT), the Autobiographical Memory Task (AMT), and the Center for Epidemiologic Studies Depression Scale Revised (CESD-R) to examine the relationship between deficits in executive control and dysphoria that may be mediated by ruminative thinking, negative involuntary memory retrieval, and autobiographical memory specificity. Executive control deficits and greater ruminative tendencies were found in the dysphoric sample relative to controls, although there was no evidence to support differences in involuntary memory retrieval or memory specificity. Furthermore, rumination, especially brooding rumination, was found to mediate the relationship between executive control deficits and dysphoria. Although the NAP task seems to measure some aspect of executive control, the results suggested that the task itself warrants further scrutiny.

*Keywords*: dysphoria, executive control, rumination, involuntary memory, autobiographical memory specificity
The Role of Executive Control Deficits in Cognitive Correlates of Dysphoria

Depression is often characterized by abnormal cognitive processes such as increased elaboration of negative emotional material and difficulties inhibiting responses to such material (Gotlib & Joormann, 2010). This increased focus on negative material often takes the form of rumination, wherein individuals focus repetitively on their negative emotional state and its potential causes and implications without actively attempting to relieve their distress (Joormann, 2006). In addition to rumination, depression has been associated with increased negative involuntary memory salience and intrusiveness (Newby & Moulds, 2011; Smets, Wessel, Schreurs, & Raes, 2012) and with a lessened ability to recall specific autobiographical memories when prompted (Brittlebank, Scott, Williams, & Ferrier, 1993; Mackinger, Loschin, & Leibetseder, 2000). Evidence has accumulated in recent years that these cognitive phenomena are sometimes associated with each other independently of depressive symptomology (e.g., Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Watkins, 2004; Watkins & Teasdale, 2001), which suggests that there may be a common causal mechanism underlying rumination, a reduced ability to manage involuntary memories, and diminished autobiographical memory specificity. It is plausible that this common mechanism may be some form of impaired cognitive control, such as a deficit in cognitive inhibition (see Brewin & Beaton, 2004; Dalgleish et al., 2007; Koster, De Lissnyder, Derakshan, & De Raedt, 2011). However, as far as I am aware, no study has yet sought to explicitly tie these phenomena together through a common cause in inhibitory dysfunction in a dysphoric (i.e., subclinically depressed) sample. The current study has two goals: firstly, to replicate these cognitive phenomena associated with depression in a dysphoric sample, and secondly, to test a mediation model whereby impaired cognitive inhibition
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contributes to dysphoria indirectly by encouraging or exacerbating rumination, negative involuntary memories, and reduced autobiographical memory specificity (see Figure 1).

Figure 1. Proposed mediation pathway in which cognitive inhibition deficits contribute to dysphoria indirectly by promoting the experience of rumination, negative involuntary memories, and less specific autobiographical memories, which in turn leads to depressive symptoms.

Rumination

Rumination is often defined as repetitive thinking about the causes, symptoms, meaning, and implications of a negative mood state (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Ruminative responses to negative events and material have been shown to prolong depressive episodes (Nolen-Hoeksema, 1991) and predict depressive disorders, including new onsets of depressive episodes (Nolen-Hoeksema, 2000). People who engage in ruminative responses to depressive symptoms have higher levels of depressive symptoms over time, even after
accounting for baseline levels of depressive symptoms (Nolen-Hoeksema & Morrow, 1991). Despite the association between depression and rumination, there is evidence to suggest that the propensity to engage in rumination is not merely a symptom of depression but rather a stable tendency that may increase one’s vulnerability to depression. Roberts, Gilboa, and Gotlib (1998) found elevated levels of rumination not only in individuals with current dysphoria, but also in individuals with previous depressive episodes compared to individuals who have never been depressed, although these individuals did not differ on concurrent depressive symptomology. Nolen-Hoeksema (2000) also showed that previously (but not currently) depressed participants have scored significantly higher on measures of rumination than never-depressed participants.

To account for the etiology of ruminative responses, Koster et al. (2011) proposed the “Impaired Disengagement” hypothesis of depressive rumination, whereby prolonged processing of negative self-referential material is due in large part to an inability to disengage attention from it, rather than, e.g., an attempt to come to a new, helpful insight about one’s current situation. Rumination involves automatic as well as controlled processes, and although rumination might involve goal-relevant material, the onset of rumination largely occurs automatically (Linville, 1996), which suggests that engaging in rumination may be the result of a failure to inhibit ruminative impulses.

**Inhibition Deficits**

Inhibitory processes are involved in limiting the access of irrelevant information to working memory and are thus important for attention, memory, and engaging in focused, goal-oriented behavior, which may be impaired in depressed or dysphoric populations. Indeed, depression is associated with deficits in cognitive inhibition and executive control more broadly (Gotlib & Joormann, 2010; Joormann, 2006). In a study by Hertel and Gerstle (2003), dysphoric
students exhibited increased recall from sets of words they had practiced suppressing relative to controls, demonstrating inhibitory deficits, and the degree of inhibitory dysfunction was significantly correlated with self-report measures of depression. Dysphoric participants and participants with a history of depressive episodes have exhibited reduced inhibition of negative material (Joormann, 2004), which suggests that reduced inhibitory ability can potentially function as a stable marker or predictor of depression. Further evidence for a predictive role of inhibition deficits was provided when reduced interference or inhibitory control predicted dysphoric symptoms and rumination longitudinally over a period of six months in a nonclinical student sample (Zetsche & Joormann, 2011).

A recent meta-analysis by Yang, Cao, Shields, Teng, and Liu (2016) showed a significant negative association between rumination and cognitive inhibition that was not accounted for by depressive status or symptomology. Cognitive inhibition deficits may therefore encourage depression indirectly by making ruminative responses more likely or perseverative and contributing to common symptoms of depression such as anhedonia, mood dysregulation, and sustained negative affect (Joormann, Yoon, & Zetsche, 2007).

**Involuntary Memories**

Diminished executive control in healthy participants was associated with higher numbers of intrusive (unwanted and involuntary) thoughts during the White Bear Suppression Task (Wegner & Zanakos, 1994), wherein participants are told to consciously suppress thoughts of a white bear while verbally expressing their thoughts stream-of-consciousness-style (Brewin & Beaton, 2004). These results suggest that proper inhibitory function may play an important role in suppressing goal-irrelevant cognitive content that could in turn provoke ruminative responses and depressive episodes. Goal-irrelevant cognitive content may include involuntary memories of
negative autobiographical events, which are a factor in the maintenance of depressive symptoms (Brewin, Reynolds, & Tata, 1999). As people ruminate more about negative involuntary thoughts and memories, they experience more such involuntary cognitive content and have higher levels of depression (Smets, Wessel, Schreurs, & Raes, 2012). Watkins (2004) proposed that engaging in rumination following a negative event prevents successful emotional processing and thus has an impact on the recurrence of the event (via the experience of involuntary memories) and on the distress associated with those memories.

Converging evidence points to a complex relationship between involuntary memories and rumination that lends credence to the possibility of a common etiological source. The relationship between the experience of negative involuntary memories and depression may be moderated by rumination, as ruminating on such memories may act to amplify their negative effects on mood and affect (Williams & Moulds, 2010). Involuntary memories can trigger ruminative thinking, and in turn ruminating on a specific involuntary memory can make additional involuntary memories more accessible (Newby & Moulds, 2011). There is evidence to suggest that rumination may trigger further involuntary memories indirectly by increasing negative affect, which makes the recollection of negative involuntary memories more likely (Ehlers & Clark, 2000). In a study by Lyubomirsky, Caldwell, and Nolen-Hoeksema (1998), dysphorics induced to ruminate subsequently recalled more negative involuntary autobiographical memories and recalled negative events as happening relatively frequently in their lives (and positive events as infrequent) as compared to nondysphoric subjects or dysphorics who engaged in the distraction control. Rumination and negative involuntary memories can reciprocally influence each other, which may plausibly be attributed to deficient cognitive inhibition. Once depressed or dysphoric individuals have begun ruminating, they may
have difficulty inhibiting negative involuntary memories, and after experiencing such memories spontaneously it may be harder to inhibit ruminative responses.

**Reduced Autobiographical Memory Specificity**

When prompted to recall a specific autobiographical memory, people with depression and other clinical syndromes such as posttraumatic stress disorder and eating disorders often demonstrate a robust tendency to recall more general memories than nonclinical populations (Dalgleish et al., 2007; Williams & Broadbent, 1986). This tendency, known as reduced autobiographical memory specificity (AMS), is clinically significant, as it predicts poorer long-term outcomes in individuals with major depressive disorder (e.g., Brittlebank et al., 1993; Hipwell, Reynolds, & Pitts Crick, 2004). In addition to depression, reduced AMS is also associated with higher levels of rumination (Watkins & Teasdale, 2001) and intrusive thoughts (Kuyken & Brewin, 1995). Brewin, Reynolds, and Tata (1999) found that reduced AMS was associated with greater levels of spontaneous intrusion of stressful memories. Just as rumination is highly correlated with depression but a separable entity, participants in a study by Brittlebank et al. (1993) who recovered from depression still demonstrated reduced AMS relative to control participants, suggesting that reduced AMS may a stable trait and not merely a function of depressive symptoms. Mackinger, Loschin, and Leibetsder (2000) compared women who had never been depressed with women who had recovered from depression and found relatively reduced AMS in the recovered sample, supporting the conclusion made by Brittlebank et al. (1993).

Williams, Stiles, and Shapiro (1999) argue that to access a specific memory and progress beyond the categorical descriptor stage during memory search to a more refined interrogation of the specific memory database, one must inhibit unneeded categorical descriptors in some way.
Failure to inhibit these descriptors, it is proposed, will lead to the generation of overly general responses (reduced AMS) to cue words. Thus, reduced AMS may be explained by difficulties in searching the self-memory system that stem from inhibitory failures. Dalgleish et al. (2007) provided evidence to suggest that reduced executive control, which includes cognitive inhibition, is to a significant extent driving the relationship between depressed mood and reduced AMS when they found that a tendency to recall overgeneral memories is negatively associated with performance on a variety of measures of executive control independent of current depressed mood.

It is hypothesized that the dysphoric sample in the present study will demonstrate reduced cognitive inhibitory abilities, greater ruminative tendencies, a greater propensity to experience negative involuntary memories, and reduced autobiographical memory specificity relative to controls. As illustrated in Figure 1, it is further hypothesized that the relationship between cognitive inhibition deficits and depressive symptoms will be mediated by rumination, negative involuntary memory retrieval, and AMS.

**Method**

**Participants**

The participants in this study were 82 currently enrolled undergraduate psychology students at a small, mid-Atlantic liberal arts college (58 females, 24 males, $M_{\text{age}} = 19.1$, $SD = 1.35$). Participants received course credit for their participation, and all provided informed consent.

**Materials**

The computer task was programmed using SuperLab 5.0 and performed on a Dell Inspiron 530 desktop computer. Participants made their responses on a RB-830 Response Pad to
ensure precision timing.

Measures

**CESD-R.**

Participants were recruited for this study based on their scores on the Center for Epidemiologic Studies Depression Scale Revised (CESD-R; Eaton, Smith, Ybarra, Muntaner, & Tien, 2004) that was taken an average of five weeks prior ($M = 5.06$, $SD = 1.60$) to the rest of data collection as part of an hour-long battery of self-report measures given to most students in introductory psychology classes. The 20 items on the CESD-R ask participants to rate how frequently in the past two weeks they have experienced depressive symptoms, both physical (e.g., “My appetite was poor”) and emotional (e.g., “I could not shake off the blues”), on a scale of 1 (“Not at all or less than 1 day last week”) to 5 (“Nearly every day for two weeks”). The CESD-R has been shown to possess high internal consistency, strong factor loadings, and theoretically consistent convergent and divergent validity with anxiety, schizotypy, and positive and negative affect (Van Dam & Earleywine, 2011). In the present study, the CESD-R again demonstrated excellent internal consistency (Cronbach’s $\alpha = 0.97$). Recruitment of participants resulted in two groups: a dysphoric group of participants who scored highly on the CESD-R ($n = 41$) with scores equal to or greater than 30 ($M = 42.07$, $SD = 10.47$), therefore meeting the criteria for the probable presence of a depressive episode in the past two weeks (Eaton et al., 2004) and dysphoric status, and a control group of participants ($n = 41$) with scores less than or equal to 3 ($M = 1.42$, $SD = 1.20$) who reported almost no depressive symptoms in the past two weeks.

**RRS.**

Rumination was assessed via the Ruminative Responses Scale (RRS) of the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991; Treynor, Gonzalez, & Nolen-
The RRS is a popular measure of ruminative tendencies (Yang et al., 2016) that consists of 22 items that assess the intensity and extent to which individuals engage in rumination. Participants rate on a scale from 1 (almost never) to 4 (almost always) the degree to which they endorse each statement as being self-descriptive when experiencing negative affect. The items capture various aspects of rumination, including thoughts about symptoms, causes, and reactions to negative mood. The RRS shows good test-retest reliability (Nolen-Hoeksema et al., 1994; Bagby, Rector, Bacchiochi, & McBride, 2004) and acceptable convergent and predictive validity (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema & Morrow, 1991). In the current study, the RRS demonstrated excellent internal consistency (Cronbach’s $\alpha = 0.94$). The RRS has also been shown to be relatively stable over the long term (14+ weeks) in depressed individuals (Bagby et al., 2004).

Treynor et al. (2003) propose that the RRS contains two subscales of five items each, reflection and brooding, that capture different subcomponents of rumination. Reflective rumination is characterized by an attempt to understand and gain some insight into the nature of one’s negative mood (e.g., “Analyze recent events to try to understand why you are depressed”), while brooding rumination consists of a passive consideration of how one’s current situation could be better (e.g., “Think, ‘Why do I have problems other people don’t have?’ ”). The two-dimensional model of the RRS has been demonstrated to possess good reliability and good convergent and discriminant validity in nonclinical samples (Schoofs, Hermans, & Raes, 2010). Both the reflective (Cronbach’s $\alpha = 0.83$) and brooding (Cronbach’s $\alpha = 0.83$) subscales possessed good internal consistency in this sample. The other 12 items that comprise the composite RRS score are depression-related and therefore, unlike the subscales, are hypothesized to be unable to capture aspects of ruminative thinking independent of general depressive
symptoms (e.g., “Think about how sad you feel,” or “Think about your feelings of fatigue and achiness”).

**NAP Task.**

The ability to inhibit irrelevant negative material was assessed by the Negative Affective Priming task (NAP task; Daches & Mor, 2015; Joormann, 2006; Zetsche & Joormann, 2011). Each trial of the NAP task is comprised of a prime display followed by a probe display, with the presentation of a central fixation cross for 1000 ms between each display. Both displays are made up of two words, one red and one blue, presented one above the other. On each display, participants were instructed to press a key to indicate whether the target word, the red one, was “negative” or “neutral” while ignoring the blue distractor word.

There were two main types of trials: inhibition and inhibition control. In the prime display of inhibition trials, the target word was neutral while the distractor word was negative. In the probe display, the target word was a different negative word while the distractor was a different neutral word. Thus, in the inhibition trials, the participants had to respond to a negative word on the probe display immediately after ignoring a negative word on the prime display (see Figure 2 for an example). Inhibition control trials possessed the same probe display (negative target and neutral distractor) as the inhibition trials, although the prime display of inhibition control trials consisted of a target and distractor that were both neutral words. Thus, in the inhibition control trials, participants responded to a negative word on the probe display without previously ignoring one on the prime display (refer to Figure 2).
Figure 2. Sample inhibition and inhibition control trials. The labels on the inhibition trial apply exactly to the inhibition control trial as well. While the distractor on the prime display of the inhibition trial is negative, the distractor on the prime display of the inhibition control trial is neutral.

The measure of inhibition on the NAP task is the NAP bias score, which is calculated by subtracting the response latency on inhibition control probe displays from the response latency on inhibition probe displays. If participants are properly inhibiting the negative distractors, a small delay will be manifested in responding to inhibition probe displays due to interference from the internal representation of the previously inhibited material (Tipper, 1985; Zetsche & Joormann, 2011), as they must respond to a negative word immediately after inhibiting one. Therefore, higher NAP bias scores represent a greater delay in responding to negative words following an attempt to ignore negative words, which has been suggested to indicate a better ability to inhibit irrelevant emotional material, since the inhibitory response to negative material
on the prime trial carries over to the probe trial (Joormann, 2006). Conversely, lower NAP bias scores would denote a relative inhibitory deficit.

Filler inhibition and filler inhibition control trials were also presented so that participants would not learn to expect negative target words immediately following a negative distractor (Daches & Mor, 2015). The prime displays of the filler inhibition and filler inhibition control trials were the same as the prime displays of the inhibition and inhibition control trials, respectively. The difference came on the probe displays, as both the target and distractor words were neutral for both types of filler trials. The NAP task consisted of 96 total trials, made up of three blocks that each contained eight trials of each of the four types (inhibition, inhibition control, filler inhibition, and filler inhibition control). The 32 trials appeared in a random order within each block in addition to a practice block of 16 trials made up of four trials of each type.

The negative and neutral words were repeated across blocks but not within them, and the words in each block did not differ in length. The location of the target and distractor words at the top or bottom of each display was randomized between and within blocks. Participants were unaware of the division of trials into prime and probe displays due the identical response required for every display and the fixation cross that occurred between every display, not just between those connected in a trial. Both the participant’s responses and reaction times on the probe displays were recorded.

The negative and neutral words used for the NAP task were drawn from the list of nouns contained in the Affective Norms for English Words (ANEW; Bradley & Lang, 1999) that had been pre-rated by impartial judges on a scale of 1 (most unpleasant) to 9 (most pleasant) to produce a valence score for each word. In the present study nouns were considered negative if they had a valence score below 4 ($M = 2.37, SD = 0.61$), while neutral words had a valence score
between 4 and 6 ($M = 5.30, SD = 0.38$). As expected, negative words had significantly lower valence scores ($t(126) = -32.08, p < .001$) than neutral words.

**CWAT.**

The Continuous Word Association Task (CWAT; Ball, 2007; Brewin & Soni, 2011; Jones & Steel, 2014) was used as a measure of the frequency of involuntary memory recall. It was made up of six trials that each began with the spoken presentation of a cue word by the experimenter. The cue words, taken from Brewin and Soni (2011), consisted of three positive (thrill, affection, and salary) and three negative cues (misery, emergency, and failure). After hearing the cue word, participants were asked to speak aloud the first word or phrase that comes to mind. As quickly as possible after providing the first word or phrase, the participants were instructed to speak aloud the next word or phrase they thought of associated with the response they had just provided, and to continue this pattern until the experimenter asked them to stop. In this way, participants created a continuous chain of words or phrases each associated in their minds with the previous one. The experimenter ended the trial after the participant had provided a random number of associations between 10 and 15.

After each trial, the sequence of associations was read aloud to the participant, who was instructed to stop the experimenter when an association was reached that had triggered an autobiographical memory. The presence of a memory so recalled was recorded, as well as the position of the word that had triggered it in the sequence. Participants provided a brief description of the recalled memory, which was used to categorize it as specific, defined as memories of events that occurred at a particular place and time and lasted less than a day, or general, which includes nonspecific memories that extend over time as well as general categories of similar events. They also gave a rating of how important the memory is to their self-identity.
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(on a scale of 0 to 10) to indicate the personal importance of the memory and a rating of the emotional content of the memory on a scale from -3 (very negative) to 3 (very positive).

Memories triggered earlier in the sequence indicate a relatively increased propensity to experience involuntary, goal-irrelevant memories in response to emotionally induced semantic cue categories (see Figure 3 for examples of CWAT responses).


Memory: “I thought of going to my aunt and uncle’s house every Thanksgiving.”


Memory: “I thought about getting bad grades.”


Memory: “When I found out my brother was in the hospital.”


Memory: “I thought of going home at the beginning of last winter break.”

*Figure 3.* Sample CWAT associations with involuntary memories. The cue word is in bold, and every association is separated by a dash. The circled word or phrase is the one that triggered the memory. The first two examples demonstrate general memories, while the latter two examples show specific memories.

**AMT.**

The Autobiographical Memory Test (AMT; Williams & Broadbent, 1986) was used as the primary measure of reduced AMS (autobiographical memory specificity). In each of the
AMT’s 10 trials, participants were presented with a cue word and asked to recall and briefly describe aloud a specific memory, defined using the same criteria as specific memories in the CWAT above. After two practice trials, participants were presented with each of the 10 cue words drawn from Williams and Broadbent’s (1986) original study consisting of five negative words (hurt, clumsy, sorry, angry, and lonely) and five positive words (interested, happy, successful, safe, and surprised) in a random order. The measure of reduced AMS was the proportion of specific memories recalled, with a lower proportion indicating a relatively reduced ability to recall specific autobiographical memories. Failure to recall a memory within 30 seconds was coded as an omission but only occurred on 2% of trials. The participants’ descriptions of the recalled memory were used by the experimenter to encode the specificity of the memory (see Figure 4 for sample AMT responses). Two experimenters ran participants separately and demonstrated high inter-rater reliability in coding the memories for specificity (agreement on a random sample of memories (n = 140) was 99.3%, κ = 0.97).

**Happy:** “I feel happy whenever I am with my roommate.”

**Hurt:** “I had a kitten when I was younger who always used to bite me.”

**Clumsy:** “I woke up in the middle of the night and knocked my phone off the bed, shattering the screen.”

**Safe:** “I felt safe being with my friend last night.”

*Figure 4.* Sample AMT memories. The cue word is in bold. The first two memories are general, while the latter two memories are specific.
Procedure

Upon entering the room, participants signed an informed consent form. Next, they were taken to a smaller adjoining room where they sat approximately two feet in front of a desk with a computer on it while the experimenter sat roughly three feet to the participants’ right. The participants then completed both the CWAT and the AMT. The order of these two memory tasks was counterbalanced to test for an effect of memory task order on the specificity of participant’s memories. After completing the CWAT and the AMT, participants completed the NAP task. Following the NAP task, participants were moved back into the larger testing room to complete the RRS questionnaire by hand. Finally, participants were debriefed and given contact information for the college counseling center and other local mental health services.

Results

Rumination

One participant omitted an item on the brooding rumination subscale, so while 82 participant responses were used for the reflective rumination subscale, only 81 participant responses were used for the composite rumination score (as measured by the total score on the RRS) and the brooding rumination subscale.

As expected, the dysphoric group showed significantly higher scores on all rumination measures ($M_C = 57.78, SD_C = 10.39; M_R = 12.02, SD_R = 3.73; M_B = 12.35, SD_B = 3.68$) than the control group ($M_C = 36.27, SD_C = 12.25; t_C(79) = 8.51, p < .001; M_R = 8.24, SD_R = 3.75; t_R(80) = 4.57, p < .001; M_B = 8.76, SD_B = 3.56; t_B(79) = 4.47, p < .001$). Please refer to Table 1 for an overview of group differences on the experimental and self-report measures.
Table 1. Group Differences on Self-Report and Experimental Measures

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<tr>
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<th>Control Group (n = 41)</th>
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<td>Memory Specificity</td>
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</table>

CESD-R = Center for Epidemiologic Studies Depression Scale Revised; RRS = Ruminative Responses Scale; NAP = Negative Affective Priming; CWAT = Continuous Word Association Task; AMT = Autobiographical Memory Test

NAP Task

Only the probe trials of the NAP task were analyzed. Trials were not used if the response was incorrect or if the reaction time for the trial was less than 250 ms or greater than 3000 ms. In addition, eight participants were removed from the NAP data analysis for having an overall error rate on inhibition probe trials exceeding 25%, leaving 74 participants’ responses on the NAP task for data analysis.

Contrary to expectations, the dysphoric group demonstrated a significantly higher NAP bias score ($M = 44.87$, $SD = 92.37$) than the control group ($M = 6.11$, $SD = 115.60$; $t(73) = 2.08$, $p <.05$). This unexpected result was nevertheless consistent with the strong association between depression and rumination, as there were positive correlations between NAP bias scores and scores on the CESD-R ($r(74) = .26$, $p <.05$), composite rumination ($r(73) = .30$, $p = .01$), and brooding rumination ($r(73) = .33$, $p <.01$), and a smaller correlation between NAP bias scores and reflective rumination that was nonsignificant but approaching significance ($r(74) = .20$, $p = .08$).
The NAP bias effect was successfully elicited, but not in the direction predicted based on previous research.

A simple linear regression analysis indicated that NAP bias scores were a significant predictor of CESD-R scores ($b = .05, t(73) = 2.26, p < .05$), composite rumination ($b_C = .04, t_C(73) = 2.64, p = .01$) and brooding rumination ($b_B = .01, t_B(73) = 2.99, p < .01$) but not reflective rumination ($b_R = .01, t_R(73) = 1.76, p = .08$). NAP bias scores ceased to predict CESD-R scores when any form of rumination was taken into account in a multiple linear regression analysis ($b_C = .01, t_C(73) = 0.63, p > .05; b_B = .02, t_B(73) = 1.02, p > .05; b_R = .03, t_R(73) = 1.60, p > .05$), which supports the view that rumination mediates the link between executive dysfunction and dysphoria. Sobel tests for mediation (Preacher & Hayes, 2004) were significant with composite rumination ($z = 2.45, p < .05$) and brooding rumination ($z = 2.52, p < .05$) but not reflective rumination ($z = 1.50, p > .05$) as the mediator between NAP bias scores and CESD-R scores, formally establishing the mediating role of composite rumination and brooding rumination.

**CWAT**

There were no significant effects of order (all $ps > .05$) when the CWAT was taken before or after the AMT, so order of the memory tests was not included in further analysis. The CWAT was successful in eliciting involuntary memories on 93% of trials. However, there was no significant difference in the total number of trials when involuntary memories were recalled between the dysphoric group ($M = 5.68, SD = 0.61$) and control group ($M = 5.46, SD = 0.92$; $t(80) = 1.27, p > .05$). There was also no significant difference in the number of word associations made during the CWAT before an involuntary memory was elicited when comparing the dysphoric group ($M = 5.56, SD = 1.67$) and control group ($M = 5.19, SD = 1.92$; $t(80) = 0.94, p$
>.05) (refer to Table 1). Somewhat surprisingly, there was no significant effect of cue valence on performance between groups (see Table 2 for a summary).

**Table 2. Group Differences on the CWAT and AMT by Cue Valence**

<table>
<thead>
<tr>
<th>Cue Type</th>
<th>Dysphoric Group (n = 41)</th>
<th>Control Group (n = 41)</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>CWAT Total Memories</td>
<td>Pos 2.88 0.33</td>
<td>2.68 0.61</td>
<td>1.80</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>Neg 2.80 0.46</td>
<td>2.78 0.52</td>
<td>0.22</td>
<td>ns</td>
</tr>
<tr>
<td>Memory Position</td>
<td>Pos 5.17 2.08</td>
<td>5.09 2.14</td>
<td>1.61</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>Neg 5.26 2.24</td>
<td>5.26 2.82</td>
<td>0.15</td>
<td>ns</td>
</tr>
<tr>
<td>Memory Specificity</td>
<td>Pos 0.52 0.33</td>
<td>0.57 0.31</td>
<td>0.64</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>Neg 0.54 0.35</td>
<td>0.60 0.33</td>
<td>0.83</td>
<td>ns</td>
</tr>
<tr>
<td>AMT Memory Specificity</td>
<td>Pos 0.83 0.20</td>
<td>0.87 0.17</td>
<td>0.85</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>Neg 0.82 0.21</td>
<td>0.84 0.16</td>
<td>0.45</td>
<td>ns</td>
</tr>
</tbody>
</table>

CWAT = Continuous Word Association Task; AMT = Autobiographical Memory Test

There were some notable differences in the characteristics of the involuntary memories recalled. The dysphoric group reported memories that were significantly more negative on positive cue trials (M = 0.92; SD = 0.99) than the control group (M = 1.50; SD = 1.21; t(80) = 2.36, p < .05) as well as significantly more personally important memories on negative cue trials (M = 4.90; SD = 2.01) than the control group (M = 3.98; SD = 1.80; t(80) = 2.18, p < .05).

AMS (defined as the proportion of specific memories recalled) on the CWAT was significantly correlated with AMS as measured by the AMT (r(82) = .22, p <.05). Thus, a correlation was found between the specificity of involuntary memories and the specificity of consciously recalled memories. However, there was no significant difference in AMS as measured by the CWAT between the dysphoric group (M = 0.53, SD = 0.24) and the control
group \((M = 0.59, SD = 0.23; t(80) = 1.10, p >.05)\). A mediation analysis was not conducted with any of the CWAT measures because there were no differences in memory retrieval between the dysphoric and control groups.

**AMT**

There was no significant difference in AMS as measured by the AMT between the dysphoric group \((M = 0.83, SD = 0.17)\) and the control group \((M = 0.85, SD = 0.13; t(80) = 0.77, p >.05)\). There was also no significant correlation between NAP bias scores and AMS \((r(74) = -.09, p >.05)\). Thus, no evidence for reduced AMS was found in the current dysphoric sample, and so no mediation analysis involving AMS was conducted.

**Discussion**

The first goal of the present study was to replicate in a dysphoric sample several cognitive characteristics frequently associated with depression: deficient cognitive inhibition, increased ruminative tendencies, abnormalities in the characteristics and elicitation of involuntary memories, and reduced AMS (autobiographical memory specificity). The second goal was to test for evidence that these cognitive characteristics may mediate the relationship between executive control deficits and dysphoria. Perhaps the most surprising result was that participants scoring highly on measures of depressive symptomology possessed higher NAP bias scores, indicating that they took longer to respond to negative words immediately after ignoring one than control controls. Some recent interpretations of the NAP task would expect the results to be reversed; since dysphoric individuals are unable to successfully inhibit the negative distractor on the prime display, they should not show a delay in responding to a negative target presented immediately afterwards (Zetsche & Joormann, 2011). Rather, NAP bias scores should be higher for the healthy controls that can successfully inhibit the negative distractor, thereby
producing an interference effect that accounts for the response latency on inhibition trials relative to inhibition control trials.

It seems unlikely that healthy controls are relatively less able to inhibit negative emotional material, as the current results might suggest according to Joormann’s (2006) theory. However, there are three possible alternative explanations for my results. The first is that the NAP task is not merely measuring cognitive inhibition. Executive control is viewed by many researchers to consist of three executive functions: inhibition, the monitoring and updating of working memory, and mental set shifting (De Lissnyder, Koster, Derakshan & De Raedt, 2010; Miyake, Friedman, Emerson, Witzki, & Howarter, 2000). While cognitive inhibition is the ability to inhibit the processing of irrelevant and distracting material, set shifting refers to the ability to switch between different tasks and mental sets and alter behavior accordingly (Monsell, 1996). Set shifting impairments have been observed in dysphoric and depressed individuals (Harvey et al., 2004; Merriam, Thase, Haas, Keshavan, & Sweeney, 1999; Rogers et al., 2004) and in high ruminators (Davis & Nolen-Hoeksema, 2000; De Lissnyder et al., 2010). Similar to rumination, set shifting has been suggested to be a stable vulnerability factor for depression, as impairments have been found in previously depressed individuals (Paelecke-Habermann, Pohl, & Leplow, 2005).

The inhibition trials of the NAP task require the participant to immediately shift from ignoring a negative distractor to responding to a negative target word, which may be an index of set shifting ability. If the NAP task more accurately measures set shifting ability, then one would expect the dysphoric groups to have relatively higher NAP bias scores, indicating a reduced ability to quickly shift from ignoring a negative word to responding to one that manifests as a response latency on the inhibition trials (Monsell, 2003). Indeed, the current results support such
an interpretation. Although there is also set shifting involved on the inhibition control trials as the participant switches from ignoring a neutral word to focusing on a negative word, one would expect the set shifting cost (as manifested in a response latency) to be greater when shifting between different modes of relating to negative stimuli, as some studies suggest that depressed individuals show the largest impairment on set shifting when stimuli are negatively valenced (Murphy et al., 1999; Deveney & Deldin, 2006).

The second possible explanation for the unexpected results is that the negative priming paradigm is able to assess cognitive inhibition, and a reduced inhibitory ability should be manifested as a higher NAP bias score rather than a lower one, contrary to Joormann’s hypothesis (see Joormann, 2006; Zetsche & Joormann, 2011). In his original negative priming research, Tipper (1985) argued that negative priming, manifested as a response latency, will only occur if the ignored cue on the prime trial is sufficiently attended to—otherwise, there will be no internal representation of the cue to cause interference on the following probe trial. Perhaps the dysphoric group demonstrated a negative priming effect because they were relatively more attentive to the negative distractor words on the inhibition prime trials than the control group was. If this is the case, then dysphorics demonstrated a response latency due to a reduced ability to inhibit processing of the negative distractor words, and one would expect higher NAP bias scores to actually represent unsuccessful inhibition, because the participant is less able to suppress processing of the distractor word and thus possesses a more salient internal representation to interfere with responding on the probe trial. Future research should examine the NAP task to determine exactly which function of executive control it measures and the mechanisms by which it does so.

The third possible explanation is that dysphoric participants might demonstrate different
response patterns on the NAP task than clinically depressed participants. If executive control impairments contribute to depressive symptoms, then it would be reasonable to expect a dysphoric group to have smaller inhibitory impairments than a clinically depressed group relative to controls. However, it seems more plausible that a dysphoric group would demonstrate a weaker but still similar trend to a clinically depressed group rather than a trend in the completely opposite direction. The selection of participants for the current study ensured that the dysphoric group, while lacking in clinical diagnosis, nevertheless reported levels of depressive symptoms that qualified as indicating a probable depressive episode in the past two weeks, which suggests that the dysphoric group was more similar than not to a clinically depressed group.

In addition to differences in executive control between the dysphoric and control groups, the current study also succeeded in eliciting differences between the groups in ruminative tendencies, with the dysphoric group scoring higher on the RRS and both the brooding and reflective rumination subscales (refer to Table 1). Executive control deficits have been associated with both the brooding and reflective rumination subscales (Whitmer & Banich, 2007; Zetsche, D'Avanzato, & Joormann, 2012), although brooding rumination has been especially linked to impaired inhibition of negative information (Daches & Mor, 2015; De Lissnyder et al., 2010). It is not surprising, then, that both rumination as a whole and brooding rumination, but not reflective rumination, mediated the relationship between executive control as measured by NAP bias scores and depressive symptomology. This is important clinically because, of the two rumination subscales, brooding is relatively more maladaptive (Schoofs et al., 2010). The current results suggest that it may be fruitful in the treatment of depression or dysphoria to attempt to reduce brooding rumination indirectly by bolstering executive control.
No difference was found in the total number of trials in which involuntary memories were recalled on the CWAT by dysphoric status, nor was there a difference in the propensity to recall involuntary memories as measured by the number of associations made on the CWAT before a memory was recalled between dysphoric and control participants. Future studies should include neutral cue words on the CWAT, as dysphorics and nondysphoric controls may differ in aspects of involuntary memory retrieval in response to non-emotional stimuli, especially if dysphorics may be more inclined to negatively interpret neutral stimuli. Some differences were found in the characteristics of memories, as the dysphoric group recalled memories that were more negative in response to positive cue words and more personally important memories in response to negative cue words. As such, although there were limited differences found in this study (refer to Table 2), the effects of cue valence on memory recall in dysphoria or depression may yet warrant further study. Furthermore, there was no indication of differences in AMS in either voluntary or involuntary memories between groups. Although reduced AMS has been posited to be a stable trait independent of current depressive status (Brittlebank et al., 1993; Mackinger, Loschin, & Leibetseder, 2000), it may nevertheless be more characteristic of depression than of dysphoria, or subclinical depression.

Although the current study successfully found reduced executive control and increased ruminative tendencies in dysphoric participants and provided evidence supporting the theory that one of the ways diminished executive functioning increases depressive symptomology is indirectly by encouraging rumination (see Figure 5), no differences were found between the dysphoric and control groups in the accessibility of involuntary memories or AMS. It is possible that differences in memory recall and characteristics may be observed at clinical levels of depression, but without replicating these effects in a dysphoric sample no comment can be made
on their possible mediating role between reduced executive function and depressive symptomology. Replication of the current study with a clinically depressed population would possibly give a clearer picture of the relationship between executive control, the autobiographical memory system, and depressive symptomology.

![Figure 5: Supported mediation pathway. The results showed that rumination mediated the relationship between executive control dysfunction and depressive symptoms.](image)

The NAP task did not attempt to examine the inhibition of neutral or positive stimuli, so it is possible that the results do not represent an executive control deficit exclusively in relation to negative material but could also represent a general executive control deficit or a deficit in handling emotional material, regardless of valence. The unexpected results of the NAP task warrant further scrutiny into the nature of the task, although it seems to measure some form of executive control.

It is worth noting that the supported mediation pathway does not preclude other forms of relationships between these phenomena in dysphoria or depression. It seems less plausible that dysphoria might also contribute to reduced executive control indirectly by encouraging rumination, reduced AMS, and the experience of negative involuntary memories, but that pathway was not tested in the current study. However, depressed mood could plausibly increase the likelihood of engaging in rumination, experiencing negative involuntary memories, and
recalling less specific memories. The reciprocal relationship between rumination and involuntary memories (Newby & Moulds, 2011; Williams & Moulds, 2010) is just one example of how potential mediators of the relationship between executive control and depressive symptoms could also influence each other.

In sum, the current study found reduced executive control and higher levels of rumination in dysphorics compared to controls, but no evidence for differences in involuntary memory retrieval or accessibility or in autobiographical memory specificity. The results support the theory that the relationship between executive control deficits and depressive symptoms are mediated by an increase in ruminative tendencies, especially the tendency to engage in brooding rumination.
References


