

DECREASED COGNITIVE FUNCTIONING IN DEPRESSION: A RESULT OF  
INHERENT DEFICITS OR A BY-PRODUCT OF EMOTION REGULATION?

A Thesis  
by  
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## **Abstract**

### **DECREASED COGNITIVE FUNCTIONING IN DEPRESSION: A RESULT OF INHERENT DEFICITS OR A BY-PRODUCT OF EMOTION REGULATION?**

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Past research has found that depression is associated with both increased rumination and decreased memory ability. Some researchers think that rumination may increase cognitive load and consequently might impair memory ability directly. The purpose of the present study, therefore, was to determine if rumination might cause the memory deficits that are found in depression. In this study, 100 young adult participants were first asked to verbally describe a recent emotionally upsetting negative event to the experimenter. After telling the story, participants were randomly assigned to either ruminate (rumination condition) or were given no further instruction (control condition). All participants then completed parts of the Wechsler Memory Scale – Fourth Edition (WMS-IV) to measure verbal and visual memory. Participants also completed several questionnaires, including the Beck Depression Inventory-II to measure depressive symptoms and the Ruminative Response Scale to measure habitual rumination. Contrary to the hypothesis, there were no significant differences in visual or verbal memory scores between the rumination and control

conditions, and depressive symptoms did not moderate the effect. In addition, and in contrast to previous literature, there was no relationship between depression and memory performance, and a small positive correlation between memory and habitual rumination. These results suggest that rumination may not be as cognitively harmful as previously theorized.

*Keywords:* cognition, memory, depression, emotion regulation, rumination

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Decreased cognitive functioning in depression: A result of inherent deficits or a  
by-product of emotion regulation?

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### **Abstract**

Past research has found that depression is associated with both increased rumination and decreased memory ability. Some researchers think that rumination may increase cognitive load and consequently might impair memory ability directly. The purpose of the present study, therefore, was to determine if rumination might cause the memory deficits that are found in depression. In this study, 100 young adult participants were first asked to verbally describe a recent emotionally upsetting negative event to the experimenter. After telling the story, participants were randomly assigned to either ruminate (rumination condition) or were given no further instruction (control condition). All participants then completed parts of the Wechsler Memory Scale – Fourth Edition (WMS-IV) to measure verbal and visual memory. Participants also completed several questionnaires, including the Beck Depression Inventory-II to measure depressive symptoms and the Ruminative Response Scale to measure habitual rumination. Contrary to the hypothesis, there were no significant differences in visual or verbal memory scores between the rumination and control conditions, and depressive symptoms did not moderate the effect. In addition, and in contrast to previous literature, there was no relationship between depression and memory performance, and a small positive correlation between memory and habitual rumination. These results suggest that rumination may not be as cognitively harmful as previously theorized.

*Keywords:* cognition, memory, depression, emotion regulation, rumination

Decreased cognitive functioning in depression: A result of inherent deficits or a by-product of emotion regulation?

Depression is one of the most common and debilitating mental illnesses. The World Health Organization (2016a) reported that depression affects 350 million people of all ages globally, making it the leading cause of disability worldwide and a significant contributor to the international burden of disease. The National Institute of Mental Health estimates that approximately 6.6% (15.7 million) of adults 18 years old and older in the United States experienced at least one major depressive episode in the last year (National Institute of Mental Health, n.d.). The prevalence of depression is particularly pronounced in adolescents; in 2010-2011, the National Survey on Drug Use and Health reported that the lifetime prevalence of a major depressive episode was 12.8% for adolescents aged 12-17, and 8.1% of adolescents aged 12-17 experienced a major depressive episode in the last year based on self-report data (Center for Disease Control and Prevention, 2013). Other reports suggest the lifetime and 12-month prevalence rates for adolescents may be slightly lower (e.g., the National Survey of Children's Health and the National Health Interview Survey reported a 7.1% lifetime prevalence rate and a 5.1% rate for the past year; Center for Disease Control and Prevention, 2013).

According to the DSM-5, major depressive episodes are marked by a significant decrease in mood, interest, and/or pleasure lasting a minimum of two weeks (American Psychiatric Association, 2013). Depressive episodes may also include changes in weight and appetite, disrupted sleep, psychomotor agitation or retardation, loss of energy, feelings of worthlessness or guilt, impaired concentration, and reoccurring thoughts of death (American Psychiatric Association, 2013). Thoughts of death may include suicidal ideation without a

specific plan, a plan for committing suicide, or a suicide attempt. Globally, more than 800,000 people commit suicide every year (World Health Organization, 2016b). Among 15 to 29 year olds, suicide was the second leading cause of death worldwide in 2012 (World Health Organization, 2016b).

Due to its prominence across the world, research has focused on better understanding depression and developing effective treatments to effectively minimize or eradicate depressive symptoms. Past research has found evidence supporting two relationships relevant to the current study.

First, Major Depressive Disorder (MDD) has been associated with cognitive deficits, particularly in attention, memory, and executive function (Baune, Fuhr, Air, & Hering, 2014; Rock, Roiser, Riedel, & Blackwell, 2013; Trivedi & Greer, 2014). However, the origin of these deficits is a topic of debate. It is not clear if cognitive deficits are a core symptom of depression or if cognitive deficits are a byproduct of other symptoms.

Second, depressive symptoms positively correlate with ruminative thoughts. Multiple research studies have found that individuals who ruminate more exhibit more depressive symptoms (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Nolen-Hoeksema & Davis, 1999; Nolen-Hoeksema, Larson, & Grayson, 1999), more extreme depressive symptoms (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema & Morrow, 1991), and longer-lasting depressive symptoms (Nolen-Hoeksema, 2000) than individuals who ruminate less.

In most of the existing research, these two relationships (between depression and cognitive deficits, and between depression and rumination) have been studied independently. Recently, researchers have proposed potential interrelationships among these three variables.

For example, Joormann and colleagues (Joormann, 2010; Joormann & D'Avanzato, 2010; Joormann & Gotlib, 2010) argued that depression is associated with inherent deficits in cognitive inhibition. This hinders the ability to remove irrelevant negative content from working memory, thus leading to ruminative thoughts and maintenance or worsening of negative mood. Conversely, Williams and colleagues (2007) argued that deficits in executive function and deleterious rumination are separable problems, but may interact in predicting the course and/or severity of depression.

A third, less studied possibility is that rumination may mediate the relationship between depression and cognitive functioning, such that cognitive deficits are a byproduct of increased rumination in depression. One small study has found initial support for this theory (Watkins & Brown, 2002), but the possibility has not been otherwise pursued.

The present research aimed to disentangle these relationships using combined experimental and correlational methods. The experimentally induced effects of rumination on auditory memory (immediate and delayed) and visual memory (immediate and delayed) were examined in people with varying levels of depressive symptoms. Based on the research reviewed below, I hypothesized that experimentally induced rumination would impair verbal memory and that depressive symptoms would be more highly correlated with memory ability in the control conditions.

### **Emotions and Emotion Regulation**

Before understanding emotion regulation, it is necessary to formally define emotions. Though the term is used colloquially, the field of psychology has long debated the proper conceptualization of emotions (e.g., Izard, 2010; Kleinginna & Kleinginna, 1981). In this

paper, emotions will be discussed in terms of the *modal model of emotions*, as described by Gross (2014).

According to the modal model of emotions, the emotion generation process begins when a situation arises. Situations can be external events (e.g., being yelled at by a boss) or internal events (e.g., worrying about a future event). Once a situation arises, a person attends to it, appraises it, and responds accordingly. The response impacts the situation and the cycle repeats itself. It is important to note that emotions are differentiated from moods. Emotions are the result of a sequential process following an event while moods are longer lasting and generally lack a specific trigger. Thus, sadness is an emotion, whereas depression is a mood.

Emotion regulation is the process through which individuals are able to influence the content, timing, and expression of their emotions, both intentionally and unintentionally (Gross, 1998a). The *process model of emotion regulation* (see Figure 1) expands on the modal model of emotions by identifying five types of emotion regulation based on where in the emotion generation process they occur: situation selection, situation modification, attentional deployment, cognitive change, and response modulation (Gross, 2014). More broadly, these five types of emotion regulation can be categorized as either antecedent focused strategies (situation selection through cognitive change) or response-focused strategies (response modulation).

Antecedent-focused emotion regulation strategies occur early in the process model of emotion regulation, prior to an emotional response (Gross, 1998b). For example, one may choose which situations to engage with or attend to during the first three steps of the process model (situation selection, situation modification, and attentional deployment). In the fourth stage of the process model of emotion regulation (cognitive change) one may engage in

*reappraisal* by construing an emotional situation in a less emotional or upsetting way prior to the emotional response (Richards & Gross, 2000).

Alternatively, response-focused emotion regulation strategies occur at the end of the process model of emotion regulation, once the initial emotion has already been evoked. Response-focused emotion regulation strategies attempt to manipulate the resulting output of the emotion, potentially by prolonging or diminishing the emotion (Gross, 1998a).

For example, yesterday, Fred was laid off due to job cuts from the company he had worked at for 10 years. When he wakes up this morning, he remembers he is out of work. Before developing an emotional reaction, Fred could employ an antecedent-focused emotion regulation strategy, such as reappraisal, to change his cognitions about the event. In this case, Fred would frame being fired as a more positive event and focus on the opportunity to explore new career options. Conversely, if Fred used a response-focused emotion regulation strategy, he would have an initial negative reaction to remembering he was fired and would then try to alter his emotions. In this case, Fred may ruminate about the event by persistently thinking about his feelings of sadness and failure that arose from losing his job.

### **Rumination as Emotion Regulation**

*Rumination* is characterized by the repetition of negative thoughts and feelings after negative stimuli and, thus, is a response-focused emotion regulation strategy. Ruminative thoughts are passive, rather than active, and typically lack productive problem solving (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Instead, ruminative thoughts focus primarily on emotional reactions to negative stimuli (Nolen-Hoeksema, 1991). Ruminative thoughts can persist in the absence of environmental stimuli for the cognitions (Martin &



Tesser, 1996). As a result, rumination can also trigger subsequent negative emotions that would not otherwise occur.

Ruminative thoughts have been compared to negative cognitive styles and automatic thoughts, which have been well studied by cognitive psychologists (Nolen-Hoeksema et al., 2008). However, ruminative thoughts differ from negative cognitions in that the primary concern with negative cognitions is the *content* of thought (Beck, Rush, Shaw, & Emery, 1979), whereas the primary concern in ruminative thoughts is the *style* of thought. A ruminative response style is a set of thoughts and behaviors that inhibit an individual's ability to improve their mood by focusing attention inward on emotions.

Prior research has identified two components of rumination: reflection and brooding (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). A growing body of research confirms that brooding and reflection are independent factors of rumination and that reflection has adaptive components while brooding does not (e.g., Burwell & Shirk, 2007; Joormann, Dkane, & Gotlib, 2006; Schoofs, Hermans, Raes, 2010). Reflection is hypothesized to assist in problem solving and has been described as the “purposeful turning inward to engage in cognitive problem solving to alleviate one's depressive symptoms” (Treynor et al., 2003). Similarly, the analytic rumination hypothesis (Andrews & Thomson, 2009) proposes that depression occurs because of a problem in someone's life, and reflective rumination is aimed at solving this problem. According to this hypothesis, depression is like a fever, which temporarily sidelines a person so that they can engage in the needed reflective problem solving.

Conversely, brooding is maladaptive, not goal-oriented, and passively compares one's situation to an unachieved outcome (Treynor et al., 2003). Brooding correlates with increased depression, both concurrently and at a one-year follow-up (Treynor et al., 2003). Currently

depressed participants score more highly on the brooding subscale of the Ruminative Response Scale than formerly depressed, socially anxious, and health control participants (Joormann et al., 2006).

Correlational studies have found a negative relationship between rumination and both active problem solving and coping. For example, individuals with high scores on ruminative items in a coping measure (the COPE) were significantly less likely to engage in active, structured problem solving (Carver, Scheier, & Weintraub, 1989). Ruminating while in a depressed mood has been shown to interfere with generating solutions to life problems (Morrow, 1990 as cited in Nolen-Hoeksema, 1991). These findings corroborate the theory that rumination interferes with problem solving.

### **Rumination and Depression**

The use, and resulting effects, of various emotion regulation strategies in the development and maintenance of psychopathology is of increasing interest to mental health professionals (e.g., Berking & Wupperman, 2012). As previously suggested, one of the most concerning facets of rumination may be its close relationship to depression. Extensive research has explored the style, content, and effects of rumination in people with depression.

For example, one meta-analysis of 114 studies examined the relationship between emotion regulation strategies and four psychopathological disorders (anxiety, depression, eating, and substance-related disorders; Aldao et al., 2010). Increased psychopathology was associated with increased usage of maladaptive emotion regulation strategies and decreased usage of adaptive strategies. A correlation with a large effect size was found between psychopathology and scores on self-report rumination measures ( $r = .49$ ); the effect size increased when psychopathology was narrowed to depressive symptoms ( $r = .55$ ). Increased

psychopathology correlated with two other maladaptive emotion regulation strategies with medium to large effect sizes: avoidance ( $r = .38$ ) and suppression ( $r = .34$ ). Increased psychopathology negatively correlated with two adaptive emotion regulation strategies: problem solving ( $r = -.31$ ) and reappraisal ( $r = -.14$ ). Problem solving negatively correlated with depression ( $r = -.33$ ), providing further evidence that rumination may interfere with effective problem solving.

Interestingly, the positive relationship between maladaptive strategies and psychopathology is consistently stronger than the negative relationship between adaptive strategies and psychopathology (Aldao & Nolen-Hoeksema, 2012). This suggests that it is the use of maladaptive strategies, rather than the “disuse” of adaptive ones, that is the larger problem for people with depression. Strategies with maladaptive features, such as rumination, may have the dual effect of both directly influencing mood, and indirectly influencing it by occupying resources that would otherwise be used for more adaptive strategies. For example, Nolen-Hoeksema (1991) theorizes that rumination augments and prolongs depression by allowing thinking to be negatively biased by a depressed mood, initiating a spiral of negative thoughts. Negative thoughts then capture the depressed person’s attention and prevent its use for more productive behavior, such as problem solving. As such, people who respond to depression with rumination by focusing on depressive symptoms, their causes, and their consequences have longer episodes of depression than people who do not ruminate about depression (Nolen-Hoeksema, 1991).

The correlation between rumination and depression remains stable over time and has been well documented in short-term longitudinal studies (e.g., lasting 1-1.5 years), and may indicate a bi-directional causal relationship. One study measured levels of rumination and

depressive symptoms at two time points: baseline and a one-year follow-up (Nolen-Hoeksema et al., 1999). Results indicated that at both time points, higher levels of rumination correlated with increased depressive symptoms and that depressive symptoms at the first time point predicted rumination levels at the second time point. Another study found that participants who engaged in more rumination after the loss of a loved one reported increased depressive symptoms over 18 months than those who engaged in less rumination (Nolen-Hoeksema & Davis, 1999). Similarly, a separate study measured levels of rumination and depressive symptoms one and six months after the loss of a loved one. At both time points, more depressed participants reported higher levels of rumination. Ruminative coping styles were associated with higher levels of depression after six months (Nolen-Hoeksema, Parker, & Larson, 1994). In conjunction with other similar studies, these findings demonstrate the stable positive relationship between rumination and depressive symptoms over time, signifying that a ruminative coping style is a prominent feature of depression.

Experimental research supports Nolen-Hoeksema's (1991) theory that rumination can both prolong and deepen depressed moods. Participants induced to feel sad report feeling significantly more depressed after ruminating than after completing a distraction task (Morrow & Nolen-Hoeksema, 1990), evidencing that rumination can deepen a sad mood. This pattern is mirrored in people with depression. In a similar paradigm, participants with depression who experienced no mood induction reported feeling significantly more depressed after ruminating than participants who completed a distraction task (Nolen-Hoeksema & Morrow, 1991). These findings imply that rumination adversely affects preexisting depressed moods. Rumination and depressed affect are likely a vicious cycle; depressive thoughts spark passive rumination, which fails to improve affect, which activates more passive rumination.

Ruminative response styles can also predict the presence and onset of depressive symptoms. Individuals who reported ruminating in response to a depressed mood prior to a traumatic event were more likely to be depressed 10 days and 7 weeks after the event (Nolen-Hoeksema & Morrow, 1991), suggesting that preexisting ruminative response styles can interact with environmental events and lead to depression.

Furthermore, a longitudinal study by Nolen-Hoeksema (2000) investigated the relationship between rumination and depression. In the study, a clinician conducted two 90-minute sessions with participants, approximately one year apart. The study resulted in three important findings. First, participants who were diagnosed with Major Depressive Disorder during the initial interview had higher scores on a rumination measure at both time points. Second, higher rumination scores during the first interview significantly predicted the onset of Major Depressive Disorder at the second time point, even after controlling for baseline depressive symptoms. This implies that rumination increases the likelihood of depression. Third, individuals who were diagnosed with depression during the first interview but not during the second interview had significantly lower scores on the rumination scale than participants who remained depressed, before controlling for baseline levels of depressive symptoms. Together, these findings establish that rumination is an integral part of depression; increased rumination may lead to depression and decreased rumination in a depressed individual may assist in recovery from a depressive episode.

### **Cognitive Effects of Rumination**

Research investigating emotion regulation independent of psychopathology demonstrates that rumination (and other maladaptive strategies) can have other detrimental effects, not just on mood, but also on cognitive ability. The theory that emotion regulation

can have cognitive consequences is rooted in Baumeister's ego-depletion model (Baumeister, Bratslavsky, Muraven, & Tice, 1998; Richards & Gross, 2000). The ego-depletion model postulates that executive functions such as choice, active responses, and self-regulation require underlying, limited cognitive resources (Baumeister et al., 1998). According to this model, self-regulation uses a portion of executive functioning resources. The amount of resources consumed is dependent upon the difficulty of the self-regulation task. The analytical rumination hypothesis also suggests that attentional control allocates limited cognitive resources to problem-solving, which compromises other goals (such as performance on laboratory tasks; Andrews & Thomson, 2009).

Several studies have investigated the effects of emotion regulation on executive functioning. These studies generally find that rumination has adverse effects on working memory and other executive functions. For example, one correlational study examined the relationship between ruminative tendencies (measured by a shortened version of the Ruminative Responses Scale) and 1) working memory (measured by the Backward Digit Span) and 2) cognitive flexibility (measured by the Wisconsin Card Sorting Test) in college students (Davis & Nolen-Hoeksema, 2000), with depression (as measured by the Beck Depression Inventory) as a covariate. Individuals with high scores on the Ruminative Response Scale (ruminators) made more perseverative errors on the Wisconsin Card Sort than individuals with low scores on the Ruminative Response Scale (nonruminators), indicating that ruminators have less cognitive flexibility than nonruminators. There was no significant effect of ruminative tendencies on working memory (Davis & Nolen-Hoeksema, 2000). One limitation of this study was that participants' ruminative behavior was not measured during cognitive tasks. Thus, Davis and Nolen-Hoeksema's (2000) study presents

interesting findings about the potential long-term association of rumination and cognitive flexibility, but does not address what effects rumination may cause at the time of occurrence. In addition, participants' scores on the Beck Depression Inventory were included as covariates in their analyses, which may have reduced the relationship between rumination and working memory capacity.

One recent quasi-experimental study addressed some of these (Curci, Lanciano, Soleti, & Rimé, 2013). In the study, researchers placed participants into a high or low working memory capacity group based on performance on a Random Number Generation task. They then presented participants with either a negative or neutral mood induction prior to their completing a second working memory task. Ruminative behaviors were measured with the Rumination Response Scale (RRS) after the task. Scores on the RRS were higher in the negative mood condition than the neutral mood condition, a finding that was exacerbated for participants with a low working memory capacity. Individuals with high rumination scores and low working memory capacity were less successful on the working memory task than individuals with high rumination scores and high working memory capacity. These findings imply that exposure to a negative emotion induction creates a competition for cognitive resources between rumination and working memory tasks. That is, because people with low working memory capacity have fewer cognitive resources available, they cannot simultaneously ruminate and perform a cognitive task.

Worrying, like rumination, is the repetition of verbal, negative, intrusive thoughts (Nolen-Hoeksema et al., 2008). The difference between rumination and worrying is subtle; rumination concentrates primarily on past events, whereas worrying often centers on future events. Rumination and worrying have similar ramifications; both have been associated with

increased negative affect and have been observed in depression. Furthermore, the cognitive tolls of worrying and rumination are similar and rely on a similar set of cognitive processes. Like rumination, worrying relies on the phonological loop, which maintains verbal material, such as words and stories, typically through subvocal thought rehearsal.

One study examined the effects of worrying on verbal and visual memory (Moreno, Ánvila-Souza, Gomes, & Gauer, 2015). Participants were divided into high-worriers and low-worriers based on their scores on the Penn State Worry Questionnaire. Participants were categorized as high-worriers if they scored from the third quartile and low-worriers if they scored up to the first quartile. High-worriers were less accurate than low-worriers on a verbal memory task. Additionally, high-worriers were less efficient (as measured by slower reaction times) than low-worriers on a visuospatial memory task. The authors argued that typical thoughts of worry were the major occupant of working memory in the high-worry group, leaving fewer mental resources available for the verbal and visuospatial tasks in the study.

### **Cognitive Deficits in Depression**

Cognitive deficits similar to those found in rumination research are also commonly found in people with depression. Although depression is primarily conceptualized as a mood disorder, many past studies have found associations between depression and deficits in cognition. Most of these studies have found evidence for neuropsychological deficits in several domains, including attention, working memory and executive function, processing speed, and episodic memory. These deficits are reflected in the DSM-5 criteria for a major depressive episode, which includes “diminished ability to think and concentrate” and “psychomotor agitation or retardation” as potential symptoms (American Psychiatric Association, 2013).



Although there is ample research documenting cognitive deficits in depression, the heterogeneity of dependent measures and participant samples (e.g., in age, timecourse of disease, medication, etc.) makes it difficult to draw strong conclusions about *which* cognitive functions are impaired and *why*. Several recent reviews highlight this difficulty and make progress towards developing a more nuanced understanding of cognition in depression (Baune et al., 2014; Rock et al., 2013; Trivedi & Greer, 2014).

Rock et al. (2013) investigated cognitive functioning in participants ranging from approximately 12-80 years old with depression during symptomatic and remitted states. The meta-analysis included only studies that used the Cambridge Neuropsychological Test Automated Battery (CANTAB). Including studies that measure cognitive ability with a single neuropsychology test battery ensures interstudy homogeneity and may help elucidate which cognitive abilities are most impaired. This meta-analysis included four tests of executive function (One Touch Stockings of Cambridge, Spatial Working Memory, Intra-Extra Dimensional Set Shift, and Spatial Span), four tests of memory (Delayed Matching to Sample, Paired Associates Learning, Pattern Recognition Memory, and Spatial Recognition Memory), one test of attention (Rapid Visual Information Processing), and one test of reaction time (Reaction Time) from the CANTAB. The executive function tests predominately measure working memory, cognitive flexibility, and spatial planning. The memory section primarily targets visual memory and pattern recognition. The Rapid Visual Information Processing test is a measure of sustained attention and Reaction Time measures motor and mental response speeds.

Compared to healthy controls, participants with depression displayed impaired attention (Cohen's  $d = -0.65$ ), executive function (Cohen's  $d$  ranged from  $-0.34$  to  $-0.54$ ), and

memory (Cohen's  $d$  ranged from -0.40 to -0.50). The authors concluded that depression significantly correlates with moderate deficits in executive function, memory, and attention (Rock et al., 2013). Though these conclusions are valuable, it is important to consider reviews and meta-analyses that use heterogeneous dependent measures.

A more diverse review focused on cognition in early/first episode Major Depressive Disorder (Trivedi & Greer, 2014). Pooled effect sizes found significant deficits in attention (effect size 0.36) and visual learning/memory (effect size 0.53). Trivedi and Greer concluded that cognitive impairments are present at the onset of depression and remain relatively stable as the illness progresses. Two studies included in this review highlight this conclusion.

One study compared brain activation of young adults, who were either at a high or low risk for depression, during a working memory task (Mannie, Harmer, Cowen, & Norbury, 2010). There were no significant differences in accuracy or response latency between the high-risk and low-risk groups, indicating that high- and low-risk individuals perform equally on a standardized test of working memory (n-back). However, high-risk participants showed greater activation in regions of the brain associated with working memory. The authors postulate that differing neural responses of high-risk participants to a working memory task might be a vulnerability marker of depression. The second study found that non-depressed participants with low episodic memory scores on a free + cued recall memory task were at a higher risk for having a diagnosis of depression three years later. The authors concluded that low episodic memory performance may be a premorbid marker of depression (Airaksinen, Wahlin, Forsell, & Larsson, 2007).

Because much of the past research on cognitive deficits in depression focused on adult or older-adult samples, a recent review centered on neuropsychological functioning in

Major Depressive Disorder occurring in adolescence and early adulthood. Baune et al. (2014) reviewed seven studies to better understand the impairment of individual cognitive domains in depression. Four studies included a measure of working memory; two of these studies found working memory impairments with medium to large effect sizes (Klimkeit, Tonge, Bradshaw, Melvin, & Gould, 2011; Matthews, Coghill, & Rhodes, 2008). Two other studies found no relationship between working memory deficits and depression (Baune, Czira, Smith, Mitchell, & Sinnamon, 2012; Korhonen et al., 2002). Visual memory was measured in two studies; one found significant differences between depressed and control groups with large effect sizes (Matthews et al., 2008) and the other found no significant relationship (McClure, Rogeness, & Thompson, 1997). Only one study included a measure of attention and found no significant differences between depressed and nondepressed participants (Kyte, Goodyear, & Sahakian, 2005). Three studies measured verbal learning and memory, however none found significant differences between non-depressed and depressed groups.

Because other meta-analyses have found significant impairments in attention, verbal memory, and working memory, further research is necessary to understand these cognitive abilities in depression. For example, it is possible that verbal and episodic memory deficits may not appear until later in adulthood, while working memory and processing speed are more impacted in young adulthood and adolescents.

Across reviews, there is convincing evidence for processing speed and attention deficits in people who are depressed, and some evidence for analogous working memory deficits. Because researchers define working memory in multiple ways, some of which overlap with other cognitive domains, it is difficult to determine the strength of the relationship between working memory and depression. In contrast to other domains, evidence

for depression-related episodic memory deficits is less consistent and less studied. The association between episodic memory deficits and depression does appear to be stronger in adult and older-adult samples. This may be, however, a result of episodic memory being measured more frequently in adult and older-adult samples.

Though impaired cognitive functioning is often observed in people with depression, research has been unable to conclusively determine whether cognitive deficits are a core feature of depression. Across the literature, the cognitive domains studied, effect sizes, and methodologies vary, sometimes resulting in conflicting findings for which domains are impaired. The variability of outcomes may be in part a result of inconsistent diagnosis conditions, treatment status, and severity of depression (McClintock, Husain, Greer, & Cullum, 2010).

Further complicating the issue, some studies have found medication improves cognitive deficits observed in people with depression (Herrera-Gurzmán et al., 2010; Wagner, Doering, Helmreich, Lieb, & Tadic, 2012), whereas other studies have found an association between antidepressant use and poor cognitive abilities such as working memory and verbal learning and ability (Lee, Hermens, Porter, & Redoblado-Hodge, 2012). It is difficult to determine the effects of antidepressant medication on cognitive functioning in depression because medication use covaries with other variables, such as treatment status and severity of symptoms.

Finally, determining the causes for cognitive deficits in people with depression is further complicated by other symptoms of the disorder (such as amotivation and reduced ability to cope with interference). Thus, despite a large body of research demonstrating a

relationship between depression and cognitive deficits, there is little certainty about the nature of these deficiencies.

### **Relationship Between Rumination, Depression, and Cognitive Deficits**

There are at least two potential theories connecting rumination, depression, and cognitive deficits. The first and more heavily researched theory suggests that cognitive deficits in depressed people, specifically decreased cognitive inhibition, result in increased rumination (Joormann, 2010; Joormann & D'Avanzato, 2010; Joormann & Gotlib, 2010). A second and less examined theory postulates that increased rumination (which is cognitively taxing) in people with depression leads to cognitive deficits (Watkins & Brown, 2002).

Joormann (2010) conceptualizes depression as a disorder of impaired emotion regulation due to deficits in inhibitory function. Under this conceptualization, inhibition is an executive function that affords control over working memory by enabling the selection and updating of its contents. Optimal working memory performance relies on high levels of inhibitory control; if inhibitory control is weakened, other cognitive processes such as learning, retrieval, and comprehension may be negatively affected (Hasher, Zacks, & May, 1999). Joormann postulates that poor cognitive inhibition exacerbates depressive symptoms through its impact on rumination (Joormann & D'Avanzato, 2010).

According to this theory, in the presence of negative stimuli, effective cognitive inhibition blocks negative thoughts and accesses mood-incongruent material, which decreases negative thoughts and increases positive thoughts in working memory. This enables recovery from a negative mood. In contrast, ineffective cognitive inhibition fails to decrease negative material in working memory and often accesses mood-congruent (negative) material. Negative thoughts in working memory increase, resulting in mood

maintenance or intensifying. Joormann and D'Avanzato (2010) suggest that poor cognitive inhibition may result in rumination and prevent the use of more adaptive emotion regulation strategies, such as reappraisal.

As discussed previously, there is some evidence that rumination is associated with increased perseveration on cognitive tasks, which is in line with Joorman's theory. To more specifically test whether rumination is associated with reduced inhibition of negative information, Joormann and Gotlib (2010) had depressed and non-depressed participants complete a negative affective priming task in order to measure their ability to cognitively inhibit negative stimuli. Compared to non-depressed participants, depressed participants exhibited a lack of inhibition for negative stimuli when processing negative material during the negative affective priming task. Participants without depression and participants with remitted depression more successfully inhibited negative material during the negative affective priming task than depressed participants. Furthermore, reduced inhibition of negative material in depressed participants correlated with increased rumination. These findings support Joormann's proposed hypothesis that depression is associated with decreased cognitive inhibition and increased rumination.

Although this study is consistent with the hypothesis that cognitive deficits cause rumination (e.g., they are associated), it cannot rule out alternative causal explanations for the association. In particular, it is also possible that rumination causes cognitive deficits. To my knowledge, little research has investigated this theory. One study investigated the possibility of rumination causing or exacerbating impairments in executive tasks in people with depression (Watkins & Brown, 2002). A 2 (group: depressed, non-depressed) x 2 (condition: rumination, distraction) design compared participants' accuracy on a random

number generation (RNG) task. All participants were induced into a sad mood by thinking about a recent personal difficulty for five minutes. Participants then completed the RNG task in a rumination condition (“think about what your feelings might mean”) and a distraction condition (“think about the shape of a large black umbrella”). The depressed group demonstrated impaired performance on the RNG task when ruminating. However, in the distraction condition, the depressed and non-depressed groups did not significantly differ on their performance on the RNG task. This shows that rumination reduces executive capacity, suggesting that executive functioning is not inherently impaired in depression. These findings support the present theory that rumination will mediate the relationship between depression and cognitive deficits.

There are a few limitations to Watkins and Brown’s (2002) study. The first is that the study had a small sample ( $n = 28$ , 14 depressed), which may lead to decreased generalizability. The second is that executive functioning was measured with only one measure (RNG). Arguably, the RNG task is a poor representation of general executive functioning (Engle, Tuholski, Laughlin, & Conway, 1999) as it primarily measures executive subfunctions, such as inhibition, updating, and monitoring (Peters, Giesbrecht, Jelicic, & Merckelbach, 2007). Other facets of cognition, such as memory, that are commonly linked with depression and rumination were not measured. As a result, the study did not comprehensively measure executive functioning and lacks the ability to validly make claims about larger cognitive functioning. Finally, rumination was not compared to a true control condition; it was compared to an alternative emotion regulation strategy (distraction).

**Present Research**

The current study sought to extend the understanding of the relationship between rumination, depressive symptoms, and cognition. The primary hypothesis was derived from three themes in the literature: depression correlates with both 1) increased rumination and 2) with decreased cognitive functioning. Additionally, 3) increased rumination correlates with decreased cognitive functioning. Together, these findings suggest that the relationship between depression and cognitive deficits may be caused by ruminative behaviors.

In this study, all participants were first asked to recall a recent negative event. After recalling the event, half of the participants were asked to ruminate on the event and half were not given further instructions. Then, all participants completed a cognitive battery, including tests from the Wechsler Memory Scale – Fourth Edition that individually test auditory and visual memory, as well as an additional computerized task of visual working memory. Participants also filled out questionnaires to measure their depressive symptoms and habitual rumination techniques.

In accordance with previous findings, I hypothesized that participants who were induced to ruminate during the memory tasks would perform worse than participants in the control condition. Importantly, I expected that this effect would be larger in participants with low levels of depression. I based this prediction on the assumption that people with high levels of depressive symptoms would be ruminating regardless of the provided instructions. In addition, because verbal memory and rumination rely on the same cognitive processes (e.g., the phonological loop), I expected the hypothesized effects to be larger on verbal memory than on visual memory.



## Method

This study was approved by the Appalachian State University IRB on October 27, 2016 (IRB #17-0009) and adheres to all ethics principles. See Appendix A for IRB approval page and consent form.

### Participants

Participants were 100 students recruited from Appalachian State University who participated in this study for partial course credit. Prior to beginning the study, it was determined that participants would be between 18-23 years of age. Four participants were excluded from analyses due to exceeding this designated age range (26, 27, 36, and 54 years old), reducing the final sample to 96 participants. Participants ranged from 18-22 years old ( $M = 19.20$ ,  $SD = 1.14$ ). Of the 96 participants, 71 were female.

### Materials

**Questionnaires.** Participants were asked to complete a total of five questionnaires: the Brief COPE, the Beck Depression Inventory-II, the Rumination Response Scale, the Beck Anxiety Inventory, and a demographics questionnaire.

**Demographics Questionnaire.** The demographics questionnaire collected basic demographic information including: age, gender, and program of study. Additionally, the questionnaire asked participants to self-report current and past mental health diagnoses, treatments, and medication use. See Appendix B.

**Beck Depression Inventory-II.** The Beck Depression Inventory-II (BDI-II; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) is a common measure of evaluating the presence and severity of depressive symptoms. The BDI-II contains 21 self-report statements. Participants respond on a scale of 0-3, with higher scores representing greater severity of

depressive symptoms. Potential scores range from 0-63. Scores from 0-13 reflect minimal depression, scores from 14-19 indicate mild depression, scores from 20-28 indicate moderate depression, and scores about 29 indicate severe depression. Normative data from the BDI-II interpretation manual indicates that the mean score for college students is 12.56 ( $SD = 9.93$ ). Reliability for the BDI-II in the current sample was  $\alpha = .73$ .

***Rumination Response Scale.*** The Rumination Response Scale (RRS; Treynor et al., 2003) contains 22 items to measure how often respondents habitually engage in ruminative behaviors. Respondents rate the frequency they engage in each statement on a 1-4 Likert scale, with higher responses indicating more frequent behavior. The RRS includes behaviors such as “think about a recent situation, wishing it had gone better”, “think about how sad you feel”, and “think about all your shortcomings, failings, faults, and mistakes”. Potential scores range from 22-88, with higher scores indicating increased rumination. The RRS can also be divided into three subcomponents: Depression, Brooding, and Reflection. Depressive symptoms are more strongly related to Brooding than to Reflection. Reliability for the overall RRS in the current sample was  $\alpha = .92$ . For the subscales, reliability was  $\alpha = .87$  for Depression,  $\alpha = .80$  for Brooding, and  $\alpha = .77$  for Reflection.

***Beck Anxiety Inventory.*** The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1998) asks respondents to indicate how much they have been bothered by 21 symptoms of anxiety in the last month. Responses are given on a 0-3 scale, ranging from 0 = “Not at all to” 3 = “Severely: it bothered me a lot”. The scale includes mental items (“fear of losing control”) and physical items (“heart pounding/racing”). Potential scores range from 0-63, with higher scores indicating increased anxiety. Reliability for the BAI in the current sample was  $\alpha = .90$ .

**Brief COPE.** The Brief COPE (Carver, 1997) is a shortened measure of the original COPE inventory, which assesses a broad range of coping mechanisms individuals may utilize to manage their stress. The Brief COPE contains 28 statements about coping responses. Respondents indicate how frequently they engage in the coping behavior on a 1-4 Likert scale, with higher ratings indicating more frequent use of the coping response. The COPE includes a variety of potential responses, such as ‘venting’ (“I’ve been expressing my negative feelings”), planning (“I’ve been trying to come up with a strategy about what to do”), and behavioral disengagement (“I’ve been giving up the attempt to cope”). The Brief COPE was included for exploratory purposes.

**Memory Battery.** Participants completed the adult battery from the Wechsler Memory Scale – Fourth Edition (WMS-IV; Wechsler, 2009). The WMS-IV is a standardized measure of memory and cognition normed for ages 16-69. It contains seven subtests, four of which measure both immediate and delayed performance. The subtests can be used to form five indexes: auditory memory, visual memory, visual working memory, immediate memory, and delayed memory.

The Wechsler Memory Scale was selected as the primary dependent variable for three reasons. First, the measured domains are impaired in people with depression with relative consistency and robust effect sizes. Second, past research has demonstrated deficits in working memory, visual memory, and verbal memory while engaging in rumination or similar processes. Finally, the Wechsler Memory Scale has been used in past depression research, (Baune et al., 2014; Trivedi & Greer, 2014).

All WMS-IV subtests were administered except Spatial Addition, which was replaced with Automated Symmetry Span (Oswald, McAbee, Redick, & Hambrick, 2015). This was

done to shorten the length of time it took to complete the battery, as pilot testing indicated that Spatial Addition took considerably longer than the remaining subtests. Like Spatial Addition, Symmetry Span is a test of visuospatial working memory. Although this substitution meant that the visual working memory index of the WMS-IV could not be calculated, it did not impact the calculation of the Auditory or Visual memory scales, which were the primary dependent variables.

***Visual Reproduction I & II.*** Visual Reproduction assesses memory for nonverbal visual stimuli. Visual Reproduction I is a test of immediate visual memory and Visual Reproduction II is a test of delayed visual memory. In Visual Reproduction I, participants are shown a series of five designs. Designs are presented for 10 seconds individually. After each design is presented, the participant is asked to draw the design from memory. Reproductions are scored after the testing session has ended. Each potential criterion in the five designs is scored independently with a score of 0 or 1, with 1 indicating that the participant correctly recalled the criterion. For both Visual Reproduction I and II, the maximum score is 43. Visual Reproduction II is administered approximately 20-30 minutes after Visual Reproduction I and assesses long-term visual-spatial memory through both free recall and recognition tasks. In the recall task, the participant is asked to draw the designs previously shown during Visual Reproduction I. Participants may reproduce the designs in any order. Reproductions are scored identically to Visual Reproduction I. In the recognition task, participants are asked to identify which designs match the original design shown during Visual Reproduction I. In the recognition task, answers are scored as 0 or 1, with scores of 1 indicating a correct response. The maximum score for the recognition task is 7 points.

***Logical Memory I & II.*** Logical Memory is a measure of narrative auditory memory. Logical Memory I assesses immediate auditory memory and Logical Memory II assesses delayed auditory memory. During Logical Memory I, participants hear two short stories. Each story contains 25 details. Immediately after hearing each story, participants are asked to recall the story to the best of their ability. Participants receive one point per detail correctly remembered according to a standard scoring guide. Logical Memory II is administered 20-30 minutes after Logical Memory I and has both recall and recognition task. During the recall task, participants are first asked to recite each story, again being scored by the amount of details correctly remembered. In the recognition task, participants answer 15 yes/no questions per story. Answers are scored as 0 or 1, with scores of 1 indicating a correct response. The maximum score for the recognition task is 30 points. For the free recall conditions in Logical Memory I and II, the maximum score is 50 points.

***Verbal Paired Associates I & II.*** Verbal Paired Associates assesses auditory memory for associated word pairs. Verbal Paired Associates I assesses immediate auditory memory and Verbal Paired Associates II assesses delayed auditory memory. During Verbal Paired Associates I, the experimenter orally reads a list of 14 word pairs to the participant. Some word pairs are intuitive (e.g., city, town), while others are random (e.g., day, box). The experimenter then reads the first word from each word pair and asks the participant to provide the associated word. There are four trials of the same 14 word pairs in different orders. Answers are scored on a 0 or 1 scale, with a score of 1 indicating a correct response. The maximum score for each trial is 14 points. The maximum score for the total subtest is 56 points. Verbal Paired Associates II is administered approximately 20-30 minutes after Verbal Paired Associates I and assesses delayed memory for verbally paired information through

both cued recall and recognition tasks. In the delayed recall task, the experimenter reads the first word of each pair and asks the participant to provide the associated word. The scoring for the delayed recall task is identical to the immediate recall condition; the maximum score is 14 points. In the recognition task, the participant is read a list of word pairs and asked to identify which word pairs are part of the original list. Scores in the recognition task are scored as either 0 or 1, with 1 indicating a correct response; the maximum score is 40 points.

*Designs I & II.* Designs is a measure of visual memory for unfamiliar visual material. Designs I is a measure of immediate visual memory and Designs II is a measure of delayed visual memory. During Designs I, participants see a grid containing 4-8 designs for 10 seconds. Once the grid is removed, the participants recreate the grid by selecting designs from a set of cards and placing the cards in the appropriate position. This process is repeated four times. Participants receive a content, spatial, and bonus score. Content scores range from 0-2 per card. If the participant fails to identify either the target or distracter card, they receive zero points. If the participant identifies the distracter card, they receive 1 point. If the participant identifies the target card, they receive 2 points. Spatial scores are either 0 or 1 per cell location. A score of 1 indicates that the participant placed any card (correct or incorrect) in an appropriate cell. A score of 0 indicates that a participant failed to place a card in a correct cell. Bonus points are awarded when the target card is placed in the correct location. Designs II is administered approximately 20-30 minutes after Designs I and has both free recall and recognition tasks. First, the participant is asked to recreate the designs shown in the immediate recall condition. Scoring for the Designs II is identical to Designs II. In the recognition portion of Designs II, the experimenter shows the participant a series of grids. Participants are asked to identify which two designs are correct and in the same place as in

Designs I. Thus, there are two correct responses per item. One point is awarded for each correct item, with a maximum of 2 points per item.

***Symbol Span.*** Symbol Span assesses visual working memory for novel visual stimuli. The participant is shown a series of abstract symbols on a page. Then, the participant is asked to identify the symbols in order from a greater number of symbols. Answers are scored on a 0-2 point scale. A score of 0 reflects incorrect answers. A score of 1 indicates that the participant has correctly recalled the symbols but in the incorrect order. A score of 2 indicates that the participant has correctly recalled both the symbols and the order. The maximum score is 50 points.

***Symmetry Span.*** Symmetry Span (Oswald et al., 2015) is an automated computer task. Participants are asked to judge whether 8x8 matrices are symmetrical down an imaginary vertical axis. In between each matrix, participants are shown a single red square in a 4x4 grid to be remembered at the end of the set. Sets range from 3-5 with two administrations for each set size. The task takes approximately 10 minutes to complete.

### **Procedure**

After giving informed consent, participants were tested individually in a quiet room by a single experimenter. Prior to the experimental portion of the study, the participants filled out a portion of the questionnaires (BDI-II, BAI, and demographics). These questionnaires were presented at the beginning of the study so that the rumination induction did not influence responses and because the IRB protocol required that the experimenter check the participants' responses to the suicidality question of the BDI-II early in the procedure. Though there was protocol in place for handling suicidal responses, no participants scored highly on the suicidality question and, thus, no further action was necessary.

After completing the questionnaires, all participants were asked to describe a recent negative event in detail for three to five minutes. If the participant did not speak for at least three minutes, then the experimenter asked general prompting questioning (e.g., “what else happened during the event?”) All narratives were audio-recorded. Participants were given the following instructions:

I would like you to describe a recent emotionally upsetting negative event. This event must be something that occurred to you and should have lasted at least a few minutes, but less than one day. For example, an ongoing fight with a friend would not be sufficient, but a specific confrontation would work well. As you describe the event, I would like you to concentrate on what things happened during the event, including what people might have said or did. I would like you to talk about how this made you feel and what the consequences of the event may be.

After the event description, participants in the control condition received no further instructions. Participants in the rumination condition received the following instructions:

While we complete the rest of the study, I would like you to think about your feelings about this event, what they might mean, and what might have caused them. After the study is over, I will ask you to retell the event. When you are retelling the story, I would like you to include the same details as you did now and also include any new emotions that may arise while you are thinking about the event.

Participants then completed the WMS-IV. Approximately halfway through the battery, between Logical Memory II and Verbal Paired Associates I, participants completed a manipulation check. The manipulation check asked participants to rate “how much they had been thinking about their story” on a scale of 1 (not at all) to 6 (constantly).



After the cognitive battery, participants were asked to repeat their original memory using the following instructions: “I would like you to retell the story you told me at the beginning of the study. As you repeat your story, please try your best to include the same details as you did before.” Finally, participants completed the RRS and Brief COPE. These questionnaires were presented at the end so as to not influence participants’ emotion regulation strategies throughout the study.

## **Results**

### **Score Calculation**

Prior to conducting analyses, scaled scores for the Auditory Memory Index (AMI) and Visual Memory Index (VMI) were calculated. The AMI included scores on Logical Memory I and II and Verbal Paired Associates I and II. The VMI score included scores on Visual Reproductions I and II and Designs I and II. Neither index included scores from the recognition part of delayed recall tests. AMI and VMI scores are derived from scores scaled for age from the relevant subtest. Scaled scores are summed and converted into a single index score for each index. For both indexes, scores range from 40-160, with 100 being the 50<sup>th</sup> percentile. In the current sample, VMI scores ranged from 82 to 136 ( $M = 104.36$ ,  $SD = 11.62$ ) and AMI scores ranged from 58 to 126 ( $M = 98.06$ ,  $SD = 11.33$ ).

### **Participant Characteristics**

Descriptive statistics for BDI-II, RRS, and BAI scores in the sample are located in Table 1. Two participants did not complete the full BDI-II inventory and one participant opted to not complete the measure. Of the participants that responded to the measure, 84.9% did not meet the cutoff for any levels of depression ( $n = 79$ ), 8.6% met the criteria for mild

depression (14-19 points;  $n = 8$ ), 5.4% met the criteria for moderate depression (20-28 points;  $n = 5$ ), and 1.1% met the criteria for severe depression (29-63 points;  $n = 1$ ).

In addition to completing the BDI-II, participants were asked to self-report whether they had a prior or current diagnosis of depression and whether they were currently taking antidepressant medication. The majority of participants ( $n = 76$ ) reported no history of depression, though one of those participants reported taking antidepressant medication. Seven participants reported a diagnosis of depression and current symptoms, and six of those were taking antidepressants. An additional 12 participants reported a previous diagnosis of depression but no current symptoms, and three of those were taking antidepressants. Finally, one participant chose not to disclose mental health information.

Finally, Table 2 presents the average BDI-II scores of the groups described in the prior paragraph. Of particular note, among the participants who reported remission of depression, those who were taking antidepressant medication had BDI-II scores closer to participants with a current diagnosis of depression. Those who were not taking antidepressants had scores closer to those with no history of depression.

### **Manipulation Check**

An independent samples t-test was conducted to compare responses on the manipulation check across the two conditions (rumination instructions vs. control instructions conditions). As expected, individuals in the rumination condition reported thinking more about their narrative during the memory tests ( $M = 2.37$ ,  $SD = 1.04$ ) than individuals in the control condition ( $M = 1.92$ ,  $SD = 1.04$ ),  $t(93) = 2.12$ ,  $p = .037$ ,  $d = .43$ . These results indicate that the rumination manipulation effectively induced participants in the

rumination condition to ruminate more during the study than individuals in the control condition.

### **Planned Analyses**

**Auditory Memory.** To test the primary hypothesis that depression would moderate the impact of the rumination manipulation on auditory memory, I conducted an ANCOVA on AMI scores with condition (rumination, control) entered into the model as a factor, BDI-II scores entered as a covariate, and an additional Condition x BDI-II interaction term. In this analysis, main effects indicate the effects of rumination and depressive symptoms on auditory memory independent of each other. The interaction effect tests whether depressive symptoms and rumination affect auditory memory together.

As may be seen in the left portion of Figure 2, the ANCOVA indicated no significant main effect of BDI-II,  $F(1,89) = 0.16, p = .686, \eta_p^2 = .002$ , no significant main effect of Condition,  $F(1,89) = 0.32, p = .575, \eta_p^2 = .004$ , and no significant interaction effects between BDI-II and Condition,  $F(1,89) = 0.72, p = .399, \eta_p^2 = .01$ .

**Visual Memory.** The ANCOVA described above was also conducted using the VMI as a dependent variable. As was the case with the AMI, the ANCOVA indicated no significant main effects of BDI-II,  $F(1,89) = .08, p = .78, \eta_p^2 = .001$ , no significant main effects of Condition,  $F(1,89) = 0.99, p = .32, \eta_p^2 = .01$ , and no significant interaction effects between BDI-II and Condition,  $F(1,89) = 0.83, p = .36, \eta_p^2 = .01$ .

**Verbal vs. Visual Memory.** Finally, to directly test the hypothesis that both Condition and BDI-II scores would impact auditory memory more than visual memory, I conducted a repeated-measures ANCOVA on memory scores, with one within-subjects variable (Memory Domain: Auditory vs. Visual), one between-subjects variable (Condition:

Rumination vs. Control), BDI-II as a continuous covariate, and an additional Condition x BDI-II interaction term.

The ANCOVA indicated a significant main effect of Memory Domain,  $F(1,94) = 21.6, p < .001, \eta_p^2 = .11$ , but no other significant main effects or interactions (all  $F$ 's  $< 1.5$ , all  $p$ 's  $> .25$ , all  $\eta_p^2 < .01$ ). Most importantly for the current hypothesis, there was no interaction between Memory Domain, Condition, and BDI-II scores,  $F(1,89) = 0.0002, p = .95, \eta_p^2 = .000$ . Unexpectedly, as reported in the score calculation section above, participants' VMI scores were significantly higher than their AMI scores. Single sample t-tests comparing the scores to 100 (the WMS-IV median) found that VMI scores were significantly higher than 100,  $t(95) = 3.86, p < .001$ , but the AMI scores were not significantly lower than 100,  $t(95) = 1.54, p = .13$ . This suggests that the difference in performance across memory domains is due to our participants performing better than average on the VMI, rather than being impaired in the AMI.

### Supplementary Analyses

**Experimental Effects Alone.** To verify that there were no effects of the manipulation, the prior analyses were also conducted without depression as a covariate. The rumination manipulation did not significantly affect AMI scores,  $t(94) = -.312, p = .756$ . Individuals in the rumination condition,  $M = 97.15, SD = 14.9$ , did not perform significantly different than individuals in the control condition,  $M = 98, SD = 11.7$ . The rumination manipulation did not significantly affect VMI scores,  $t(94) = -.82, p = .414$ . Individuals in the rumination condition,  $M = 103.6, SD = 11.47$ , did not perform significantly differently than individuals in the control condition,  $M = 105.55, SD = 11.87$ .

**Correlational Analysis.** In addition, I conducted correlational analyses to better understand the relationship between depression, rumination, and memory (See Table 3). Consistent with past research, depression was significantly positively correlated with RRS total scores, as well as all three subsections (reflection, depression, and brooding). Surprisingly, and in line with the ANCOVA analyses above, I did not replicate past findings showing that depressive symptoms were associated with reduced auditory or visual memory.

There was also a consistent, small positive relationship between RRS scores and the two memory measures, though only the relationship between RRS-Reflection and the AMI index was statistically significant. To confirm this unexpected positive association between RRS scores and memory, I re-ran the repeated measures ANCOVA with RRS scores as a covariate, rather than BDI-II scores. In this analysis, the only significant effect was that of RRS on overall memory ability,  $F(1,92) = 4.88, p = .03$ .

**Self-Reported Depression Status.** A second set of analyses investigated the effects of self-reported depression status on Memory and RRS total scores. First, a 2 (Memory Domain: Auditory vs. Visual) x 3 (Depression Status: Current Diagnosis vs. Remitted vs. No Diagnosis) ANOVA on memory scores indicated that Depression Status was associated with overall memory performance, though the effect was just below statistical significance,  $F(2,92) = 2.95, p = .06$ . As may be seen in Table 4, this effect was due to the Remitted group having better memory scores than the No Diagnosis group,  $F(1,86) = 5.81, p = .02$ . Though the Remitted group also had numerically higher memory scores than the Current Diagnosis group, the difference did not reach statistical significance,  $F(1,17) = 1.93, p = .18$ , likely in part due to low power to detect the effect. The No Diagnosis and Currently Depressed group did not differ from each other,  $F(1,82) = 0.21, p = .65$ .

In addition, there were significant effects of self-reported depressed status on RRS scores,  $F(2,92) = 9.07, p < .001$ . Specifically, post-hoc comparisons using Tukey's HSD test indicated that total scores on the RRS were significantly higher in the both Currently Depressed group ( $M = 55.43, SD = 11.46$ ) and the Remitted Depression group ( $M = 47.58, SD = 7.95$ ) than in the No Diagnosis group ( $M = 39.25, SD = 11.32$ );  $p = .001$ , and  $p = .04$ , respectively. There were no significant differences between the Currently Depressed and Remitted Depression group,  $p = .295$ .

Self-reported depression status had significant effects on the Brooding,  $F(2,91) = 4.49, p = .014$ , Reflection,  $F(2,90) = 4.88, p = .01$ , and Depression,  $F(2,90) = 9.12, p < .001$ , subcomponents of the RRS. Post-hoc comparisons using Tukey's HSD test were conducted; all significant differences are reported. For the Brooding subsection, the Currently Depressed group ( $M = 13.14, SD = 4.34$ ) exhibited significantly more brooding rumination than the No Diagnosis group ( $M = 9.48, SD = 3.16$ ),  $p = .012$ . For the Reflection subsection, the Currently Depressed ( $M = 12.43, SD = 3.10$ ) group exhibited significantly more reflective rumination than the No Diagnosis group ( $M = 8.99, SD = 3.40$ ),  $p = .024$ . For the Depression subsection, the Currently Depressed group ( $M = 30.00, SD = 6.26$ ) exhibited significantly more depressive rumination than the No Diagnosis group ( $M = 20.92, SD = 6.15$ ),  $p = .002$ . The Remitted Depression group also ( $M = 26.25, SD = 5.86$ ) exhibited significantly more depressive rumination than the No Diagnosis group,  $p = .017$ .

### **Discussion**

This study tested a novel theory about the relationship between rumination, cognitive deficits, and depression: namely, that chronic rumination may be a source of memory deficits in people with depression. This theory was tested experimentally by asking people to either

ruminate (or not) on a personally relevant story while performing a series of memory tests. The primary hypothesis was that experimental condition and depressive symptoms would have an interactive effect on auditory memory, such that asking people to ruminate would impact cognitive performance with increasing negative strength as depressive symptoms decreased. Thus, the rumination induction would decrease cognitive performance more for individuals with low depressive symptoms than for individuals with high depressive symptoms.

Although the manipulation check was significant, there was no interaction effect between depressive symptoms and rumination for either auditory memory or visual memory. Even when depression was not included as a covariate, there was no impact of condition on participants' memory scores. Therefore, the primary hypothesis was not supported. Moreover, supplementary analyses suggested that there was no relationship between depression and memory in the current sample, which contradicts prior research. If anything, having a chronic ruminative response tendency was associated with *better* memory. This relationship between chronic rumination and better memory supports the analytical rumination hypothesis proposed by Andrews and Thomson (2009).

The only finding of the current study that replicated prior research was that rumination was associated with both depressive symptoms and self-reported diagnostic status. Not only was there a strong positive correlation between depressive symptoms and chronic rumination, people with any history of depression (current or remitted) had higher levels of chronic rumination than people with no history of depression.

### **Depression and Cognition**

As reviewed in the introduction, although there is evidence for a relationship between depression and cognitive impairments, the exact nature of the deficits is unclear and likely moderated by study- and participant-related variables. There is relatively consistent evidence that people with depression show deficits in attention, memory, and executive functioning (e.g., Baune et al., 2014). Most meta-analyses and reviews that report memory deficits, however, do not further specify which domains of memory are measured (e.g., Rock et al., 2013). Given these findings, this study hypothesized that depressive symptoms would correlate with decreased auditory and visual memory. This hypothesis was not supported. Not only were depressive symptoms uncorrelated with visual and verbal memory, the supplementary analyses indicated that people with remitted depression actually had better memory than people with no history of depression. There are at least three potential explanations for this hypothesis not being supported.

First, it is possible that this study did not have enough participants with depression to detect differences in cognitive abilities. Given that the correlation between the BDI-II and AMI scores was both small and positive ( $r = .04$ ) and that there appears to be a non-linear relationship between diagnostic status and memory, it is unlikely that statistical power is the underlying problem. It is, however, important to remember that both depressive symptoms and diagnostic status were based on self-report data. It is possible that a more rigorous measurement (e.g., a multi-measure, multi-perspective, or multi-setting paradigm) of depressive status would detect more cognitive differences.

Second, it is possible that the test used to measure memory was not sufficiently sensitive to detect depression-related cognitive deficits. As previously discussed, there are



inconsistent findings about which cognitive domains are impaired in people with depression and how large the deficits are. Many factors complicate the relationship, including heterogeneity of measures. Even when the same measures are used, conflicting results have been found. For example, several studies have used the WMS-IV (Logical Memory in particular) to study verbal memory in people with depression and have found mixed results. Reppermund, Ising, Lucae, and Zihl (2008) found significant differences between patients with depression and healthy controls aged 22-58 years old ( $M = 43.5$ ) on Logical Memory, but Korhonen et al. (2002) found no significant memory differences between adolescents with MDD ( $M = 18.9, SD = 2.0$ ) and healthy participants on Logical Memory ( $M = 16.0, SD = 1.9$ ). Inconsistent results using the WMS suggests that the measure is capable of detecting differences, however differences may not be reliably present. It is possible that Logical Memory is less capable of detecting memory differences in younger adults or that memory deficits are more common in older adults with depression, but definitive conclusions cannot be drawn because Reppermund et al. (2008) did not provide separate analyses for younger and older adults, although age was controlled for in their analyses.

Finally, it is possible that there is simply not a strong relationship between depressive symptoms and memory. The present study is not the first or only one to find these null effects; many other studies have failed to find a relationship between depression and cognitive deficits. For example, some studies have found evidence for impaired visual learning and memory (Matthews et al., 2008; Trivedi & Greer, 2014), but others have not (McClure, Rogness, & Thompson, 1997). Thus, rather than being flawed due to sample size or test selection, it is possible that the current results support a notable subset of literature suggesting that depression is not marked by inherent cognitive deficits.

It is also possible, as suggested by some of the supplementary analyses, that the relationship between depressive symptoms and memory deficits may be more complex than was previously thought. In particular, it appears that although a ruminative response style is positively associated with depressive symptoms, it is also associated with better overall memory.

### **Rumination and Cognition**

The research reviewed previously suggests that rumination, and its future-oriented counterpart “worry” are cognitively demanding. This research focused on investigating how these processes impact working memory. For example, findings suggest that some ruminating individuals (measured by high scores on the RRS) exhibit working memory deficits while ruminating (Curci et al., 2013). Separate research has demonstrated verbal working memory deficits in high-worriers (Moreno et al., 2015). Nolen-Hoeksema et al. (2008) presented findings linking rumination and worrying, suggesting that while rumination and worrying are distinct processes, they share many underlying characteristics (such as repetitive and self-focused perseverative thoughts) and are associated with similar cognitive impairments (such as concentration and attention).

The present research theorized that rumination may be characterized by similar deficits in verbal memory due to the shared characteristics presented by Nolen-Hoeksema, Wisco, and Lyubomirsky (2008). However, the experimentally induced rumination manipulation showed no impact on memory scores. In addition, the RRS scores showed a positive relationship with memory ability, indicating that individuals who report higher levels of habitual rumination actually have better overall memory than individuals who report lower levels of habitual rumination. One possibility for this finding is that rumination and worrying

may not be as related as previously theorized. It is also possible that rumination is not uniformly deleterious.

Beyond the obvious difference in time orientation between rumination (past focused) and worry (future focused), there are several factors that differentiate worry and rumination that may explain why worrying correlates with impaired verbal memory but rumination correlates with improved verbal memory. When worrying, one is contemplating an event with uncertain outcomes but when ruminating, one is rehashing a past event (Nolen-Hoeksema et al., 2008). It is possible that worrying requires more cognitive resources because individuals are required to generate possible outcomes, whereas rumination does not require such creative thinking, as the outcome has already been determined. As a result, rumination would not have the same harmful effect on verbal memory as worrying. Moreover, research on past vs. future thinking suggests that thinking about the future is more strongly related to working memory capacity than thinking about the past (Hill & Emery, 2013).

A second related possibility is that, rather than rumination either reducing cognitive resources or competing with access to the phonological loop, a ruminative response style serves as practice for rehearsing verbal information. It is also possible that individuals with better memory abilities are more prone to rumination. After all, a person cannot ruminate on a past that they cannot remember.

One piece of evidence in support of these ideas is the correlations between the subsections of the RRS and AMI scores. Of the three subsections of the RRS, the reflection subsection is most highly correlated with AMI scores. Reflection items on the RRS are neutrally valenced and operationalize rumination as the engagement in contemplation (i.e.,

“write down what you are thinking and analyze it”). Neutral contemplation of past events, rather than brooding on the negative feelings associated with the event, may simply be a productive method of rehearsal.

One puzzling contradiction in this study’s findings is that although the rumination manipulation was successful, and although RRS scores were positively associated with memory ability, there were no significant differences in memory ability between the experimental conditions. The lack of relationship between condition and memory scores suggests that the rumination manipulation, despite inducing participants to think more about their personal narrative, may tap into something different than habitual rumination. It is also possible that the rumination manipulation does successfully induce rumination, but in-the-moment rumination has different effects than long-term rumination. A third possibility is that the rumination induction was not strong enough, and people in the rumination condition weren’t thinking enough about their memory to make a difference; even in the rumination condition, scores on the manipulation check were low ( $M = 2.37$ ).

If the reason habitual rumination is correlated with higher memory scores is due to prior practice rehearsing verbal information, then it logically follows that the rumination induction should not necessarily result in better AMI scores. Participants in the rumination condition are not all high habitual ruminators, thus they have different levels of practice rehearsing verbal information. This indicates that it is the past experience with rumination that is associated with higher AMI scores, and not active rumination in the moment.

### **Future Analyses**

Within this dataset, two additional analyses may help to understand the observed patterns in the data. First, experimenters anecdotally noted that there was a wide variance of

the intensity of the negative stories. It is possible that differences in the severity of negative stories told in participants' narratives may be impacting the results. For example, some subgroups (such as women or individuals with increased depressive symptoms) may consistently give more negative narratives than others. Although analyzing the narratives was beyond the scope of this thesis, I plan to transcribe and analyze the narratives in two ways. First, I will process the narratives using the Linguistic Inquiry and Word Count (LIWC) software, which can provide counts for negative and positive emotion words. This allows for an object quantification of the negativity of the narratives. In addition, the transcripts will be subjectively scored by raters to estimate differences in emotional significance.

In addition, narratives can be analyzed to create a secondary, indirect manipulation check. All narratives were recorded during the first and second recitations. If the rumination manipulation was effect, it is possible that participants in the rumination condition would be more accurate in the retelling of their stories than participants in the control condition. To test this, raters blind to condition could compare the first and second versions of the narratives and calculate a percentage of details participants accurately provide in the second telling.

### **Limitations**

One major limitation of this study is that no direct measure of affect was administered. Thus, it is not possible to know how the negative mood induction affected participants or if there were systematic differences in participants' experiences of negative affect. Rumination passively rehearses negative, personally-relevant, emotional content. Without a direct mood measure, it is not possible to determine if participants' moods were impacted enough to result in a thinking pattern that truly reflects rumination. It is possible that participants were successfully verbally rehearsing their narratives, but did not feel lasting

negative repercussions. As described above, analyzing negative and positive words from participants' narratives can serve as an indirect measure of affect, partially mitigating this problem.

The restricted variability in BDI-II scores may also have been a limiting factor. The restricted range of BDI-II scores and positively skewed distribution may have limited the ability to detect significant differences due to depressive symptoms. It should be noted, however, that there were still robust correlations between BDI-II scores and rumination measures, suggesting that BDI-II scores were variable enough to detect strong relationships.

Another potential limitation is that participants were all between the ages of 18 and 23 years old. Past research has found conflicting evidence of cognitive impairments in depression, with some studies yielding no significant findings while others find cognitive deficits. A review focusing on adolescence and early adulthood reported mixed results for working memory and visual memory deficits in depression, but no effects of depression on attention or verbal memory. Furthermore, young adults at high- and low-risk for depression have been shown to not perform significantly differently on a working memory task (Mannie et al., 2010). These findings, in combination with the current results, suggest that cognitive deficits in depression may be more prevalent in middle-aged or older- adults than in young adults. The cognitive effects of both depression and rumination may have been more robust if this study included middle- and older-adults in the sample.

A fourth possible limitation is the statistically significant difference between AMI and VMI scores. Participants performed above average on visual memory tasks, but not verbal memory tasks. This difference cannot be explicitly explained by the current data. One possible explanation is that the mood induction altered performance on either the visual or

verbal memory tasks. Additionally, it is possible that there are unique characteristics to this sample, resulting in improved visual memory.

### **Conclusions and Future Directions**

Subsequent studies should further investigate the relationship between rumination and worrying. The current study found that habitual rumination, as measured by the RRS, is associated with improved verbal memory, which challenges previous theories that rumination may have cognitive effects similar to worrying. No measure of habitual worrying was included in this study, thus the relationship between worrying and verbal memory cannot be analyzed. Future research could include both measures of worrying and rumination when measuring cognition to determine if these processes are as linked as past research has theorized.

Future studies aiming to experimentally manipulate rumination should consider possible changes to increase the strength of the induction. It is possible that the induced rumination was not long-lasting enough to significantly affect all memory tests. To address this problem, the memory battery could be shortened. Additionally, the variability in the severity of the narratives may have resulted in the rumination induction not being uniformly strong. Future studies could assign participants a specific topic to ruminate about to reduce this variability. For example, the experimenter could provide false positive, neutral, or negative feedback to participants on a personality or intelligence measure and ask participants to reflect on the feedback. This would ensure that all participants are ruminating about a similar, but still self-relevant, topic.

Another finding to further investigate is the relationship between memory and the subcomponents of rumination. Present correlational findings suggest that reflective

rumination is positively correlated to verbal memory, but the other two subcomponents (brooding and depression) are not. Future studies should seek to disentangle the cognitive effects of these three subcomponents.

Ultimately, this study provides valuable insight to the relationship between cognition and rumination. The present results suggest that rumination may not be as cognitively harmful as previously thought. The positive relationship between rumination and verbal memory does not easily fit into the literature which has found correlations between 1) increased rumination and increased depressive symptoms and 2) increased depressive symptoms and decreased cognitive functioning. Though rumination is associated with decreased mood and increased mental illness, it is possible that rumination may be cognitively beneficial.



### References

- Airaksinen, E., Wahlin, A., Forsell, Y., & Larsson, M. (2007). Low episodic memory performance as a premorbid marker of depression: Evidence from a 3-year follow-up. *European Psychiatry, 22*, S221. doi: 10.1016/j.eurpsy.2007.01.739
- Aldao, A. & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology, 121*, 276-281. doi: 10.1037/a0023598
- Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217-237. doi: 10.1016/j.cpr.2009.11.004
- American Psychiatric Association. (2013). Depressive Disorders. *In Diagnostic and Statistical manual of mental disorders* (5th ed.). doi: 10.1176/appi.books.9780890425596
- Andrews, P. W., & Thomson, J. A. Jr. (2009). The bright side of being blue: Depression as an adaptation for analyzing complex problems. *Psychological Review, 116*, 620-654. doi:10.1037/a0016242
- Baumeister, R. F., Bratslavsky, E., Muraven, M., & Tice, D. M. (1998). Ego depletion: Is the active self a limited resource? *Journal of Personality and Social Psychology, 74*, 1252-1265. doi: 10.1037/0022-3514.74.5.1252
- Baune, B. T., Czira, M. E., Smith, A. L., Mitchell, D., & Sinnamon, G. (2012). Neuropsychological performance in a sample of 13-25 year olds with a history of non-psychotic major depressive disorder. *Journal of Affective Disorders, 141*, 441-448. doi: 0.1016/j.jad.2012.02.041

- Baune, B. T., Fuhr, M., Air, T., & Hering, C. (2014). Neuropsychological functioning in adolescents and young adults with major depressive disorder – A review. *Psychiatry Research, 218*, 261-271. doi: 10.1016/j.psychres.2014.04.052
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893–897.
- Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford.
- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561-571.
- Berking, M., & Wupperman, P. (2012). Emotion regulation and mental health: Recent finding, current challenges, and future directions. *Current Opinion in Psychiatry, 25*, 128-134. Doi: 10.1097/YCO.0b013e3283503669
- Burwell, R. A., & Shirk, S. R. (2007). Subtypes of rumination in adolescence: Associations between brooding, reflection, depressive symptoms and coping. *Journal of Clinical Child and Adolescent Psychology, 36*, 56-65. doi: 10.1080/15374410709336568
- Carver, C. S. (1997). You want to measure coping but your protocol's too long: Consider the Brief COPE. *International Journal of Behavioral Medicine, 4*, 92-100.
- Carver, C. S., Scheier, M. F., & Weintraub, J. K. (1989). Assessing coping strategies: A theoretically based approach. *Journal of Personality and Social Psychology, 56*, 267-283.
- Centers for Disease Control and Prevention. (2013). Mental Health Surveillance Among Children – United States, 2005-2011. *Morbidity and Mortality Weekly Report, 62*.

- Curci, A., Lanciano, T., Soleti, E., & Rimé, B. (2013). Negative emotional experiences arouse rumination and affect working memory capacity. *Emotion, 13*, 867-880. doi: 10.1037/a0032492
- Davis, R. N. & Nolen-Hoeksema, S. (2000). Cognitive inflexibility among ruminators and nonruminators. *Cognitive Therapy and Research, 24*, 699-711. doi:10.1023/A:1005591412406
- Engle, R. W., Tuholski, S. W., Laughlin, J. E., & Conway, A. R. (1999). Working memory, short-term memory, and general fluid intelligence: A latent-variable approach. *Journal of Experimental Psychology: General, 128*, 309-331.
- Gross, J. J. (1998a). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2*, 271-299. doi:10.1037/1089-2680.2.3.271
- Gross, J. J. (1998b). Antecedent- and response-focused emotion regulation: Divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*, 224-237. doi: 10.1037/0022-3514.74.1.224
- Gross, J. J. (2014). Emotion regulation: Conceptual and empirical foundations. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 3-20). New York, NY: Guilford.
- Hasher, L., Zacks, R. T., & May, C.P. (1999). Inhibitory control, circadian arousal, and age. In D. Gopher & A. Koriat (Eds.) *Attention and performance XVII: Cognitive regulation of performance: Interaction of theory and application* (pp. 653–675). Cambridge, MA: MIT Press.
- Herrera-Gurzmán, I., Gudayoi-Ferré, E., Herrera-Abarca, J. E., Herrera-Guzmán, D., Montelongo-Pedraza, P., Blázquez, F. P., ... Guárdia-Olmos, J. (2010). Major depressive disorder in recovery and neuropsychological functioning: Effects of

- selective serotonin reuptake inhibitor and dual inhibitor depression treatments on residual cognitive deficits in patients with major depressive disorder in recovery. *Journal of Affective Disorders*, *123*, 341-350. doi: 10.1016/j.jad.2009.10.009
- Hill, P. F., & Emery, L. (2013). Episodic future thought: Contributions from working memory. *Consciousness & Cognition*, *22*, 677-683. doi: 10.1016/j.concog.2013.04.002
- Izard, C. E. (2010). The many meanings/aspects of emotion: Definitions, functions, activation, and regulation. *Emotion Review*, *2*, 363-370. doi: 10.1037/a0021843
- Joormann, J. (2010). Cognitive inhibition and emotion regulation in depression. *Current Directions in Psychological Science*, *19*, 161-166. doi: 10.1177/0963721410370293
- Joormann, J. & D'Avanzato, C. (2010). Emotion regulation in depression: Examining the role of cognitive processes. *Cognition and Emotion*, *24*, 913-939. doi: 10.1080/02699931003784939
- Joormann, J., Dkane, M., & Gotlib, I. H. (2006). Adaptive and maladaptive components of rumination? Diagnostic specificity and relation to depressive biases. *Behavior Therapy*, *37*, 269-280.
- Joormann, J. & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion*, *24*, 281-298. doi: 10.1080/02699930903407948
- Kleinginna, P. R. & Kleinginna, A. M. (1981). A categorized list of emotion definitions with suggestions for a consensual definition. *Motivation and Emotion*, *5*, 345-379. doi: 10.1007/BF00993889

Klimkeit, E. I., Tonge, B., Bradshaw, J. L., Melvin, G. A., & Gould, K., (2011).

Neuropsychological deficits in adolescent unipolar depression. *Archives of Clinical Neuropsychology*, *26*, 662-676. doi: 10.1093/arclin/acr051

Korhonen, V., Laukkanen, E., Antikainen, R., Peiponen, S., Lehtonen, J., & Viinamaki, H.

(2002). Effect of major depression on cognitive performance among treatment seeking adolescents. *Nordic Journal of Psychiatry*, *56*, 187-193. doi: 10.1080/080394802317607165

Kyte, Z. A., Goodyear, I. M., & Sahakian, B. J. (2005). Selected executive skills in

adolescents with recent first episode major depression. *Journal of Child Psychology and Psychiatry*, *46*, 995-1005. doi: 10.1111/j.1469-7610.2004.00400.x

Lee, R. S., Hermens, D. F., Porter, M. A., & Redoblado-Hodge, M. A. (2012). A meta-

analysis of cognitive deficits in first-episode major depressive disorder. *Journal of Affective Disorders*, *140*, 113-124. doi: 10.1016/j.jad.2011.10.023

Mannie, Z. N., Harmer, C. J., Cowen, P. J., & Norbury, R. (2010). A functional magnetic

resonance imaging study of verbal working memory in young people at increased familial risk of depression. *Biological Psychology*, *67*, 471-477. doi:10.1016/j.biopsycho.2009.10.006

Martin, L. L., & Tesser, A. (1996). Some ruminative thoughts. In R. S. Wyer Jr. (Ed.),

*Advances in social cognition* (pp. 1-47). Hillsdale, NJ: Erlbaum.

Matthews, K., Coghill, D., & Rhodes, S. (2008). Neuropsychological functioning in

depressed adolescent girls. *Journal of Affective Disorders*, *111*, 113-118. doi: 10.1016/j.jad.2008.02.003

- McClintock, S. M., Husain, M. M., Greer, T. L., & Cullum, C. M. (2010). Association between depression severity and neurocognitive function in major depressive disorder: A review and synthesis. *Neuropsychology, 24*, 9-34. doi: 10.1037/a0017336
- McClure, E., Rogeness, G. A., & Thompson, N. M. (1997). Characteristics of adolescent girls with depressive symptoms in a so-called 'normal' sample. *Journal of Affective Disorders 42*, 187-197.
- Moreno, A. L., Ánvila-Souza, J., Gomes, W. B., & Gauer, G. (2015). Effects of worry on verbal and visual working memory. *Psychology & Neuroscience, 8*, 341-349. doi: 10.1037/h0101277
- Morrow, J. & Nolen-Hoeksema, S. (1990). Effects of responses to depression on the remediation of depressive affect. *Journal of Personality and Social Psychology, 58*, 519-52
- National Institute of Mental Health. (n.d.). *Major depression among adults*. Retrieved July 13, 2016 from: <http://www.nimh.nih.gov/health/statistics/prevalence/major-depression-among-adults.shtml>
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*, 569-582. doi: 10.1037/0021-843X.100.4.569
- Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109*, 504-511. doi: 10.1037//0021-843X.109.3.504

- Nolen-Hoeksema, S. & Davis, C. G. (1999). "Thanks for sharing that": Ruminators and their social support networks. *Journal of Personality and Social Psychology*, 77, 801-14. doi: 10.1037/0022-3514.77.4.801
- Nolen-Hoeksema, S., Larson, J., & Grayson, C. (1999). Explaining the gender difference in depressive symptoms. *Journal of Personality and Social Psychology*, 77, 1061-1072. doi: 10.1037/0022-3514.77.5.1061
- Nolen-Hoeksema, S. & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta earthquake. *Journal of Personality and Social Psychology*, 61, 115-121.
- Nolen-Hoeksema, S., Parker, L. E., & Larson, J. (1994). Ruminative coping with depressed mood following loss. *Journal of Personality and Social Psychology*, 67, 92-104. doi:10.1037/0022-3514.67.1.92
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubovitsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, 3, 400-424. doi:10.1111/j.1745-6924.2008.00088.x
- Oswald, F. O., McAbee, S. T., Redick, T. S., & Hambrick, D. Z. (2015). The development of a short domain-general measure of working memory capacity. *Behavior Research Methods*. doi: 10.3758/s13428-014-0543-2
- Peters, M., Giesbrecht, T., Jelicic, M., & Merckelbach, H. (2007). The random number generation task: Psychometric properties and normative data of an executive function task in a mixed sample. *Journal of the International Neuropsychological Society*, 13, 626-634. doi: 10.1017/S1355617707070786

- Reppermund, S., Ising, M., Lucae, S., & Zihl, J. (2008). Cognitive impairment in unipolar depression is persistent and non-specific: Further evidence for the final common pathway disorder hypothesis. *Psychological Medicine, 39*, 603-614. Doi: 10.1017/S003329170800411X
- Richards, J. M. & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology, 79*, 410-424. doi:10.1037/70022-3514.79.3.410
- Rock, P. L., Roiser, J. P., Riedel, W. J., & Blackwell, A. D. (2013). Cognitive impairment in depression: A systematic review and meta-analysis. *Psychological Medicine, 44*, 2029-2040. doi: 10.1017/S0033291713002535
- Shoofs, H., Hermans, D., & Raes, F. (2010). Brooding and reflection as subtypes of rumination: Evidence from confirmatory factor analysis in nonclinical samples using the Dutch Ruminative Response Scale. *Journal of Psychopathology and Behavioral Assessment, 32*, 609-617. doi: 10.1007/s10862-010-9182-9
- Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research, 27*, 247-259. doi: 0147-5916/03/0600-0247/0
- Trivedi, M. H. & Greer, T. L. (2014). Cognitive dysfunction in unipolar depression: Implications for treatment. *Journal of Affective Disorders, 152-154*, 19-27. doi: 10.1016/j.jad.2013.09.012
- Wagner, S., Doering, B., Helmreich, I., Lieb, K., & Tadic, A. (2012). A meta-analysis of executive dysfunctions in unipolar major depressive disorder without psychotic



- symptoms and their changes during antidepressant treatment. *Acta Psychiatrica Scandinavica*, *125*, 281–292. doi: 10.1111/j.1600-0447.2011.01762.x
- Watkins, E. & Brown, R. G. (2002). Rumination and executive function in depression: An experimental study. *Journal of Neurology, Neurosurgery & Psychiatry*, *72*, 400-402. doi: 10.1136/jnnp.72.3.400
- Wechsler, D. (2009). Wechsler Memory Scale-Fourth Edition (WMS-IV): Technical and interpretive manual. San Antonio, TX: Pearson.
- Williams, J. M. G., Barnhofer, T., Crane, C., Hermans, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin*, *133*, 122-148. doi: 10.1037/0033-2909.133.1.122
- World Health Organization. (2016a). *Depression*. Retrieved on July 13, 2017 from <http://www.who.int/mediacentre/factsheets/fs369/en/>
- World Health Organization. (2016b). *Suicide*. Retrieved on July 13, 2017 from <http://www.who.int/mediacentre/factsheets/fs398/en/>

Table 1

*BAI, BDI-II, RRS, and RRS Subsections Descriptive Statistics*

Measure	<i>M</i>	<i>SD</i>
BAI	10.34	8.95
BDI-II	8.39	6.51
RRS: Total	41.43	11.84
RRS: Depression	22.17	6.62
RRS: Brooding	9.83	3.29
RRS: Reflection	9.49	3.38

*Note.* BAI = Beck Anxiety Inventory. BDI = Beck Depression Inventory. RRS = Ruminative Response Scale.

Table 2

*Mean Beck Depression Inventory-II Scores by Diagnostic and Medication Status*

Diagnostic Status	Current Antidepressant Medication?	
	No	Yes
Current Depression Diagnosis	$M = 31.0, SD = 0.0$	$M = 18.8, SD = 7.0$
Remitted Depression Diagnosis	$M = 8.1, SD = 6.4$	$M = 19.7, SD = 9.7$
No History of Depression	$M = 6.8, SD = 4.6$	$M = 12.0, SD = 0.0$

*Note.* Where  $SD = 0, N = 1$ .

Table 3

*Correlations Between BDI-II, RRS, and Memory Scores*

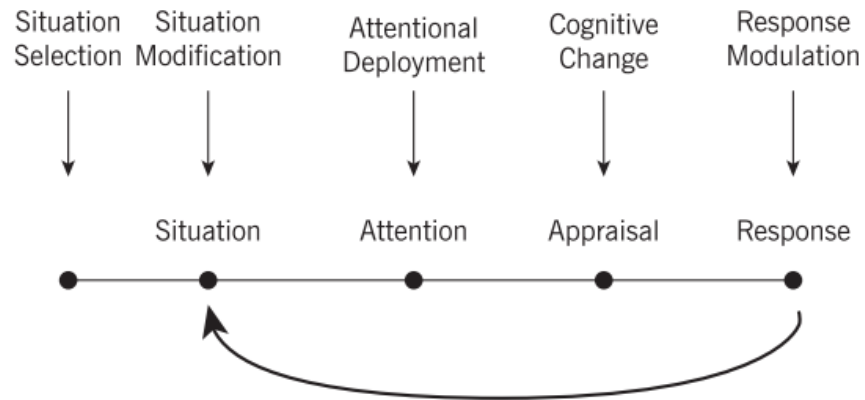
Variables	1	2	3	4	5	6	7
1. BDI-II	--						
2. RRS Total	.60**	--					
3. RRS Depression	.65**	.95**	--				
4. RRS Brooding	.50**	.83**	.70**	--			
5. RRS Reflection	.35**	.84**	.70**	.58**	--		
6. AMI Score	.04	.18	.16	.04	.23*	--	
7. VMI Score	-.04	.15	.15	.13	.10	.33**	--

*Note.* \*\* $p < .001$ , \*  $p < .05$ . BDI = Beck Depression Inventory; RRS = Ruminative Response Scale; AMI = WMS-IV Auditory Memory Index; VMI = WMS-IV Visual Memory Index.

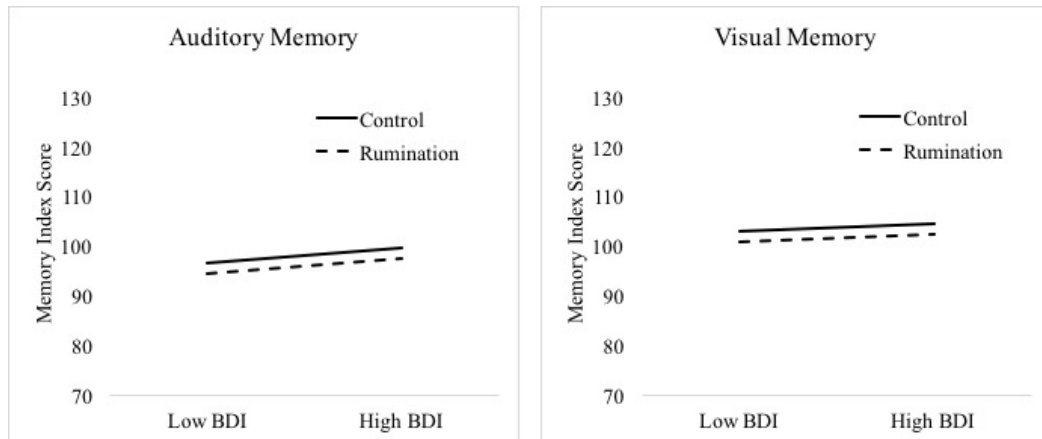
Table 4

*Auditory and Visual Memory Index Descriptive Statistics*

Diagnostic Status	Memory Index	
	AMI	VMI
Current Depression	$M = 96.57, SD = 13.54$	$M = 107.57, SD = 9.31$
Prior Depression	$M = 104.83, SD = 7.94$	$M = 109.75, SD = 11.93$
No Depression	$M = 97.24, SD = 11.46$	$M = 103.46, SD = 11.73$



*Figure 1.* Process Model of Emotion Regulation. Reprinted from *Handbook in Emotion Regulation* (p. 7) by J. J. Gross, 2014, New York, NY: Guilford.



*Figure 2.* Impact of Experimental Condition and BDI-II scores on Auditory Memory (Left) and Visual Memory (Right). Plots were made using parameter estimates, with BDI-II Scores at +/- 1 SD below the mean.

## Appendix A



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 Email: [irb@appstate.edu](mailto:irb@appstate.edu)  
 Federalwide Assurance (FWA) #00001076

**To:** Kathryn Hardin  
 Psychology  
 CAMPUS EMAIL

**From:** Lisa Curtin, PhD, IRB Chairperson  
**Date:** 10/27/2016  
**RE:** Notice of IRB Approval by Expedited Review (under 45 CFR 46.110)  
**Agrants #:**  
**Grant Title:**

**STUDY #:** 17-0009  
**STUDY TITLE:** Memory Recollection and Cognition  
**Submission Type:** Initial  
**Expedited Category:** (6) Collection of Data from Recordings made for Research Purposes,(7) Research on Group Characteristics or Behavior, or Surveys, Interviews, etc.  
**Approval Date:** 10/27/2016  
**Expiration Date of Approval:** 10/26/2017

The Institutional Review Board (IRB) approved this study for the period indicated above. The IRB found that the research procedures meet the expedited category cited above. IRB approval is limited to the activities described in the IRB approved materials, and extends to the performance of the described activities in the sites identified in the IRB application. In accordance with this approval, IRB findings and approval conditions for the conduct of this research are listed below.

All approved documents for this study, including consent forms, can be accessed by logging into IRBIS. Use the following directions to access approved study documents.

1. Log into IRBIS
2. Click "Home" on the top toolbar
3. Click "My Studies" under the heading "All My Studies"
4. Click on the IRB number for the study you wish to access
5. Click on the reference ID for your submission
6. Click "Attachments" on the left-hand side toolbar
7. Click on the appropriate documents you wish to download

### Approval Conditions:

**Appalachian State University Policies:** All individuals engaged in research with human participants are responsible for compliance with the University policies and procedures, and IRB determinations.

**Principal Investigator Responsibilities:** The PI should review the IRB's list of PI responsibilities. The Principal Investigator (PI), or Faculty Advisor if the PI is a student, is ultimately responsible for ensuring the



protection of research participants; conducting sound ethical research that complies with federal regulations, University policy and procedures; and maintaining study records.

**Modifications and Addendums:** IRB approval must be sought and obtained for any proposed modification or addendum (e.g., a change in procedure, personnel, study location, study instruments) to the IRB approved protocol, and informed consent form before changes may be implemented, unless changes are necessary to eliminate apparent immediate hazards to participants. Changes to eliminate apparent immediate hazards must be reported promptly to the IRB.

**Approval Expiration and Continuing Review:** The PI is responsible for requesting continuing review in a timely manner and receiving continuing approval for the duration of the research with human participants. Lapses in approval should be avoided to protect the welfare of enrolled participants. If approval expires, all research activities with human participants must cease.

**Prompt Reporting of Events:** Unanticipated Problems involving risks to participants or others; serious or continuing noncompliance with IRB requirements and determinations; and suspension or termination of IRB approval by an external entity, must be promptly reported to the IRB.

**Closing a study:** When research procedures with human subjects are completed, please log into our system at [https://appstate.myresearchonline.org/irb/index\\_auth.cfm](https://appstate.myresearchonline.org/irb/index_auth.cfm) and complete the Request for Closure of IRB review form.

**Websites:**

1. PI responsibilities:

<http://researchprotections.appstate.edu/sites/researchprotections.appstate.edu/files/PI%20Responsibilities.pdf>

2. IRB forms: <http://researchprotections.appstate.edu/human-subjects/irb-forms>

CC:

Lisa Emery, Psychology

**Consent to Participate in Research**  
*Information to Consider About this Research*

***Memory Recognition and Cognition***

Principal Investigator: Kathryn Hardin

Department: Psychology

Contact Information:

Kathryn Hardin – PI  
[hardinkl@appstate.edu](mailto:hardinkl@appstate.edu)  
(940) 393 – 5137

Dr. Emery – Faculty Advisor  
[emerylj@appstate.edu](mailto:emerylj@appstate.edu)  
828-262-2272, ext. 416

You are being invited to take part in a research study about memory recollection and cognition. If you take part in this study, you will be one of about 75 people to do so. By doing this study we hope to learn about cognitive performance and memory.

The research procedures will be conducted on the second floor of Smith-Wright on the campus of Appalachian State University.

You will be asked to describe in detail a recent memory, which will be audio recorded. Additionally, you complete a set of standardized cognitive tests with the experimenter and fill out a number of questionnaires on your own. You cannot volunteer for this study if you are under 18 years of age.

**What are possible harms or discomforts that I might experience during the research?**

To the best of our knowledge, the risk of harm for participating in this research study is no more than you would experience in everyday life.

**What are the possible benefits of this research?**

There may be no personal benefit from your participation but the information gained by doing this research may help others in the future by expanding the scientific community's understanding of the relationship between memory recollection and cognition.

**Will I be paid for taking part in the research?**

We will compensate you for the time you volunteer while being in this study. Participants participating for class credit will receive 3 ELC credits.

*ELC Credit:* You will not be paid for your participation in this study. However, you can earn 3 ELC credits for your participation. There are other research options and non-research options for obtaining extra credit or ELC's. One non-research option to receive 1 ELC is to read an article and write a 1-2 page paper summarizing the article and your reaction to the article. More information about this option can be found at: [psych.appstate.edu/research](http://psych.appstate.edu/research). You may also wish to consult your professor to see if other non-research options are available.

*Non ELC Credit:* Participants who are not eligible for ELC credits will receive \$20. Payment will be paid in full in the event that you chose to end the study early.

**How will you keep my private information confidential?**

We will make every effort to prevent anyone who is not on the research team from knowing that you gave us information or what that information is. Your data will only be associated with a subject number, which will not be linked with your name. Data will be kept indefinitely but will be stripped of any personal identifiers.

**Who can I contact if I have questions?**

The people conducting this study will be available to answer any questions concerning this research, now or in the future. You may contact the Principal Investigator at [hardinkl@appstate.edu](mailto:hardinkl@appstate.edu). If you have questions about your rights as someone taking part in research, contact the Appalachian Institutional Review Board Administrator at 828-262-2692 (days), through email at [irb@appstate.edu](mailto:irb@appstate.edu) or at Appalachian State University, Office of Research and Sponsored Programs, IRB Administrator, Boone, NC 28608.

**Do I have to participate? What else should I know?**

Your participation in this research is completely voluntary. If you choose not to volunteer, there will be no penalty and you will not lose any benefits or rights you would normally have. If you decide to take part in the study you still have the right to decide at any time that you no longer want to continue. There will be no penalty and no loss of benefits or rights if you decide at any time to stop participating in the study. If you decide to participate in this study, let the research personnel know. A copy of this consent form is yours to keep.

This research project has been approved by the Institutional Review Board (IRB) at Appalachian State University.

This study was approved on: September 26, 2016

This approval will expire on September 25, 2017 unless the IRB renews the approval of this research.

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Participant's Name (PRINT)

Signature

Date

**Appendix B**  
**Demographics Information**

1. Age: \_\_\_\_\_

2. Gender: \_\_\_\_\_

3. **Ethnic Background:**

- a. American Indian or Alaskan Native
- b. Asian or Pacific Islander
- c. African American, Black (Not Hispanic origin)
- d. Hispanic
- e. Caucasian, White (Not Hispanic origin)
- f. Other

4. **Have you been diagnosed with a depressive disorder?**

- a. Yes, and am currently feeling depressed
- b. Yes, but am not currently feeling depressed
- c. No history of depression
- d. Prefer not to answer

5. **Are you currently taken any anti-depressant medication?**

Yes \_\_\_\_\_ No \_\_\_\_\_

### **Vita**

Kathryn L. Hardin was born in Omaha, Nebraska, on June 22, 1992, to Rick and Laurie Hardin. She has one younger sibling, Anna Hardin. She graduated valedictorian from the Uruguayan American School in Montevideo, Uruguay, in June of 2010. In September of 2010, Kathryn began her undergraduate studies at Northeastern University in Boston, Massachusetts, and graduated magna cum laude in May of 2015 with a Bachelors of Science in Psychology. To further her education, Kathryn moved to Boone, NC, in August of 2015 to pursue a Masters of Arts in the Experimental Psychology Program at Appalachian State University. After graduating in May of 2017, Kathryn will be attending Indiana University-Purdue University Indianapolis to pursue a Ph.D. in Clinical Psychology.