AN INVESTIGATION OF MAINTENANCE AND THE CORRELATES OF DEPRESSION: RUMINATION, EMOTION REGULATION, COGNITIVE INHIBITION, SLEEP, AND DIET

By

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Abstract

Prior research has shown that depression is highly correlated with rumination, difficulties with emotion regulation and cognitive inhibition, and changes in sleep and appetite. Yet, little information exists regarding possible associations between these constructs themselves within depressed individuals. Given the possible link between the ongoing state of depression and these factors, it is important to determine how these factors are related to one another within the depressed state. Therefore, the present study extended previous research by investigating the relationship between rumination, emotion regulation, cognitive inhibition, sleep and diet in the context of depression. Questionnaires related to rumination, emotion regulation, sleep, and personal diet among controls, previously-depressed and currently depressed participants. Participants also completed a go/no-go computer task in which they identified the valance of emotional words. The results revealed that depressed and previously-depressed individuals responded more quickly to emotional words than healthy controls. The individuals with current depressive symptomology also exhibited impairments in the inhibition of negative stimuli.

The results also suggested that maladaptive emotional reactivity may be a remnant of previous functioning among those with a past history of depression, as the current sample demonstrated emotion regulation trouble despite their lower levels of depressive symptoms. A number of processes were also found to be related in the context of a depressive episode. These findings highlight the significance of identifying the nature of the relationships between symptoms thought to maintain one's depression, as this could help to develop more effective intervention strategies.

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Contents

An Investigation of Maintenance and the Correlates of Depression: Rumination, Emotion Regulation, Cognitive Inhibition, Sleep and Diet

The various mood disorders, including depression, are considered among the most prevalent and serious of all psychological illnesses. In fact, major depressive disorder is one of the leading causes of disability worldwide (World Health Organization, 2006). Some studies suggest that as many as 25% of women and 12% of men will experience depression sometime during their lifetimes (American Psychiatric Association, 2000). A current depressive episode is often associated with anhedonia, persistent sadness and disturbances of normal sleep, appetite, and cognitive functioning.

Depression is not only disabling, it is persistent. Although research suggests that most individuals recover from each depressive episode within the first year, approximately 10% will not achieve remission even after five years from the initial onset (Boland & Keller, 2010). Alternatively, over the course of a lifetime, as many as 20% of individuals with depression are expected to develop a chronic course of depression which has an average duration of 15 years (Kocsis et al., 2008; Spijker et al., 2002). Of those who do recover, more than half experience a relapse within two years (Keller et al., 1984). Furthermore, individuals remain at a high risk for recurrence throughout their lifetime, with recurrence risks increasing with each subsequent episode (Boland & Keller, 2010). Therefore, it is clear that this disorder is serious issue for both the medical and mental health fields, and one factor that makes the disorder serious is its persistence.

Causality and the Stages of Depression

Throughout the depression literature, researchers tend to differentiate between onset, maintenance, remission or recovery, and relapse or recurrence (Ingram, Miranda, & Segal,

1998). Although each of these depressive stages have received some attention, relatively little is known about the maintenance of depression. Although the value of studying depressive maintenance may appear less important, depression maintenance factors have a clear role in that they influence the duration of this disorder. Arguable in fact they play the most important role in depression because it is the persistence of depression rather than its initial onset that creates both misery and disability. More generally, the variables that maintain depression can thus be thought of as playing an important causal role in that they prolong distress and disability and interfere with the termination of the depressive episode.

Many argue that the importance of risk factors lies in the ability to identify vulnerable individuals after discovering those factors which are linked to the onset of the disorder (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Mazure, 1998). However, associations between risk factors and an extended period of time before recovery can also be useful in determining what perpetuates the depressive episode. Therefore, the investigation of dysfunctional processes that are theorized to underlie and maintain depression after its onset can increase our understanding of risk and causality, the disorder itself, and potentially help us develop more effective intervention strategies.

The Maintenance of Depression

There are a number of processes which often arise concurrently with depression; yet, these processes may in turn also contribute to the ongoing nature of the depressive episode. Therefore, an examination of how such variables are related to maintenance may be fruitful in determining which processes have the potential to act as maintenance variables. Research has shown that depression is correlated with rumination, difficulties with emotion regulation and cognitive inhibition, and changes in sleep and appetite. Therefore, the aim of the present study

was to investigate the relationship between maintenance and rumination, emotion regulation, cognitive inhibition, sleep and diet in the context of depression. Each of these possible maintenance variables is now examined.

Rumination

Several theorists have suggested that depressed persons' repeatedly engage in persistent negative self-thoughts (Beck, Rush, Shaw, & Emery, 1979; Teasdale, 1983). Research has confirmed that this pattern of thinking often does take place during depressive episodes and that for many it takes on the form of rumination (Pietromonaco & Markus, 1985; Nolen-Hoeksema et al., 1994), which is the tendency to repeatedly focus on depressive symptoms, feelings of sadness and the possible causes of one's emotional state (Nolen-Hoeksema, 1991). Research has also shown that rumination is the type of self-reflection that exhibits the strongest association with depression (Mor & Winquist, 2002).

In terms of its relationship with maintenance, Davis and Nolen-Hoeksema (2000) have argued that because rumination is related to an "increased recall of negative autobiographical memories, more negative interpretations of current situations, more negative predictions about one's future, and less effective interpersonal problem solving" (p. 700), this demonstrates that rumination acts to maintain the depressive state. Rumination has also been shown to be associated with longer and more severe periods of depression (Lyubomirsky & Nolen-Hoeksema, 1993). Other studies have demonstrated that ruminative styles of responding actually prolong mood disturbances (Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Thus, it has been suggested that rumination acts to maintain and possibly intensify depressive symptoms (Nolen-Hoeksema, 1991). A number of studies have supported this idea, demonstrating that rumination may play a role in the intensity and duration of depressed mood. This widespread finding has

been reported across experimental, cross-sectional and longitudinal designs (for a review see Thomsen, 2006).

Sleep

Problematic sleep is one of the most common complaints for individuals suffering from depression. Research has consistently demonstrated that depression and issues with sleep are highly correlated (Gregory et al., 2011; Breslau, Roth, Rosenthal, & Andreski, 1996). Indeed, early investigations found that an overall decline in quality of sleep is a problem reported by more than 90% of patients with depression (Mendelson, Gillin, & Wyatt, 1977), although more recent studies estimate that between 60 and 90% of depressed individuals report having difficulty sleeping (Kloss & Szuba, 2003; Tsuno, Besset, & Ritchie, 2005). Typical complaints include difficulty falling asleep, sleep disruptions, frequently or repeatedly waking up, shorter periods of sleep, and non-restorative sleep. Therefore, the relationship between depression and sleep problems has been thoroughly established, making it one of nine symptoms listed as diagnostic criteria for a Major Depressive Episode by the DSM-IV. For these reasons, sleep continues to be a highly researched correlate of depression.

In a longitudinal study examining depressive symptoms and sleep disturbances over a 17-week period with individuals meeting criteria for major depressive disorder, Sbarra and Allen (2009) found that the largest decreases in mood symptoms happened only after high levels of insomnia had first been reduced. The authors mention that it may be the case that these results uniquely apply to reductions in symptomatology. However, this finding may also relate to the issue of maintenance; if depressive symptoms can be decreased by reducing sleep disturbances, it seems to follow that a continuation of sleep problems may perpetuate depression. It has also been demonstrated that subjective perceptions of one’s sleep may significantly contribute to the

impact and severity of depression (Mayers & Baldwin, 2006). This may lead to negative cognitions, such as excessive worrying related to sleep. Thus, it has been proposed that these cognitive disturbances may contribute to the maintenance of insomnia (Harvey, 2000) and possibly depression as well.

Emotion Regulation

Some theories have suggested that depressed persons' do not respond differently to negative stimuli than do individuals who are not depressed. Instead, it may be that those who are depressed have a harder time recovering from the experience of negative emotions (Teasdale, 1988; Rottenberg, 2007). As such, this implies that depression is not a problem related to one's initial response to negative events, but is actually a problem with the subsequent regulation of emotions. This process of emotion regulation refers to the cognitive processes that individuals use in order to control the influence of emotionally arousing stimuli or information. Maladaptive emotion regulation strategies have been implicated as being a key mechanism in contributing to depression. Some go so far as to suggest that the disorder should in fact be conceptualized as a result of impaired emotion regulation (Campbell-Sills & Barlow, 2006).

Research has demonstrated that depression is associated with dysfunctional emotional reactivity, which supports the idea of deficits in emotion regulation among depressed individuals (Ehring, Fischer, Schnulle, Bösterling, & Tuschen-Caffier, 2008). Further investigations have suggested that certain emotion regulation strategies, such as rumination, suppression and catastrophizing, are particularly harmful when compared to strategies such as reappraisal and acceptance. These dysfunctional emotion regulation strategies are more frequently used by individuals with depression and are correlated with higher levels of depressive symptomatology (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Ehring, Tuschen-Caffier, Schnuelle,

Fischer, & Gross, 2010). Since these individuals have difficulties effectively regulating their emotions, many believe that this leaves them more susceptible to the development of depression (Ehring et al., 2008; Rude & McCarthy, 2003). Therefore, the use of certain emotion-regulation strategies may be central to studies of depression vulnerability and onset. In addition, these same dysfunctional strategies could exacerbate depressive symptoms or maintain the depressive state.

Cognitive Inhibition

It has previously been argued that cognitive inhibition plays a central role in the regulation of emotion (Joormann, Yoon, & Zetsche, 2007; Joormann, 2010), and thus it can be thought of as a specific emotion regulation strategy. Given that dysfunctional emotion regulation may play an important role in the development and maintenance of depression, deficits in this process are likely to have a considerable impact on mood disorders as well. In fact, Joormann and colleagues (2007) suggested that research on the topic of cognitive inhibition has the potential to increase our understanding of the processes through which negative mood states are maintained. This serves to highlight the importance of cognitive inhibition. Although this strategy is a complex mechanism of executive control involving numerous components, it can be described as the process of restricting or disregarding irrelevant information, preventing the activation of unnecessary information and updating the content of working memory (Hasher, Zacks, & May, 1999; Friedman, & Miyake, 2004).

In the context of depression, it has been suggested that depressed individuals may have a hard time ignoring or inhibiting the processing of negatively valenced information (Joormann, 2010). The same may be true for individuals who are vulnerable to the development of depression. Research has supported this hypothesis, demonstrating that depressed patient's exhibit impairments in the inhibition of negative stimuli both in a laboratory and hospital setting

(Joormann, 2004; Goeleven, Raedt, Baert, & Koster, 2006; Frings, Wentura, & Holtz, 2007). Since such findings indicate that these individuals cannot disinhibit from negative information, it has been suggested that deficits in this process may play a causal role in the negative cognitive biases long known to be associated with depression (Joormann et al., 2007). Considering that such cognitive biases have been shown to be associated with depressive episodes that are more severe and longer in duration (Alloy et al., 2000; Nolen- Hoeksema et al., 1994), it is reasonable to suggest that the dysfunctional inhibition may act to exacerbate symptoms and extend the depressive episode. Therefore, deficits in cognitive inhibition may play a central role in the maintenance of depressive psychopathology.

Diet

Significant dietary or appetite change are a DSM-IV symptom of depression. Numerous studies have documented associations between dietary patterns and depression (e.g., Nanri et al., 2010; Sanchez-Villegas et al., 2009). However, more recent investigations have begun to suggest that individual diet may be more than just a depressive symptom. In fact, some theorize that it may have a direct influence on depression through the modulation of physiological factors influenced by the intake of certain vitamins and minerals (Jacka & Berk, 2007). Christensen (1996) proposed that nutrition plays a likely role in both the development and maintenance of the depressive state. Therefore, research has begun to take a more detailed look at the relationship between these two constructs.

In a study using 10-year longitudinal data from women, a negative correlation was found between diet quality and major depression, as assessed by the Structured Clinical Interview for the DSM-IV-TR and a food frequency questionnaire (Jacka et al., 2010). In fact, healthier diets were associated with decreased levels of depression even after controlling for a number of

demographic characteristics. Akbaraly et al. (2009) investigated the relationship between dietary patterns and depression and found that habitual intake of a whole food diet (e.g., fruits, vegetables, fish) was associated with lower incidents of subsequent depression during a 5-year follow-up, whereas a diet containing processed foods was associated with high rates of subsequent depression. Interest has recently turned toward similar longitudinal studies as data continue to provide evidence that quality of diet may act as a risk factor for the onset of depression. For example, Sanchez-Villegas et al. (2011) demonstrated that intake of trans-unsaturated fats was correlated with an increased risk for depression over time, whereas an inverse dose-response relationship was found for the consumption of monounsaturated fatty acids and polyunsaturated fatty acids in relation to depression.

A different line of research has begun to indicate that a nutritional diet may be used as an intervention for depression. Early investigations demonstrated that a removal of caffeine and refined sugar from the diet produced significant improvements in a sample of individuals with major depressive disorder (Christensen & Burrows, 1990). The researchers suggested that symptoms might be managed by eliminating these dietary elements since they appear to contribute to the depression. A large body of research has also shown that omega-3 fatty acids may prove to be an effective treatment for depressive symptomatology (see Rocha Araujo, Vilarim, & Nardi, 2010). Likewise, another review found that nutritional supplements or diet alterations to target specific nutrients could be beneficial for depression, although further research is needed to confirm these findings (Harbottle & Schonfelder, 2008). Notably, it follows that if altering one's dietary quality can help to bring about the termination of the depressive state, it may be that the inverse is true as well – that an unhealthy diet could contribute to the perpetuation of depression. Considering this growing body of evidence, many researchers are

now proposing that the potential impact that diet can have on depression has long been underestimated (Christensen, 1996; Harbottle & Schonfelder, 2008). Furthermore, although diet and nutrition are often overlooked when examining factors related to depression, it has clear potential to be a significant factor in both the etiology and maintenance of the disorder.

Possible Depression Maintenance and the Relationship Between Variables

Although it has now been well established that correlations exist between these factors and the onset of depression, their relationship to the maintenance of depression is uncertain. Because the present study is not longitudinal, it cannot provide data on maintenance per say. However, given the possible associations between the ongoing state of depression and the previously discussed factors, it is important to determine how these factors are related to one another within the depressed state. Hence, determining whether and how these variables are linked may lead to a greater understanding of the nature of depression, and may provide clues to how depression is maintained.

Even though few studies have examined connections between sleep, rumination and depression, there is reason to believe that they may be linked. It has previously been found that people with insomnia often exhibit depressive symptoms and engage in ruminative processes (Kales, Caldwell, Soldatos, Bixler, & Kales, 1983), but Thomsen, Mehlsen, Christensen, and Zachariae (2003) were the first to investigate the relationship between rumination, subjective sleep quality and negative mood. Their results indicated significant correlations between these three factors, with sleep and rumination found to be associated even after controlling for negative affect. Carney, Edinger, Meyer, Lindman, and Istre (2006) attempted to replicate these findings using the Beck Depression Inventory (Beck, Steer, & Brown, 1996), the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991), and the Pittsburgh Sleep Quality Index

(Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) and discovered that, in response to depressive affect, poor quality sleepers ruminated more frequently than good sleepers. Therefore, despite this limited sample, sleep and rumination appear to be related within the context of ongoing depression.

There is some indication that rumination is related to emotion regulation as well. Research has demonstrated that rumination is correlated with the suppression and avoidance of negative thoughts and feelings, both of which are strategies that can be employed to regulate emotions (Moulds, Kandris, Starr, & Wong, 2007). Wenzlaff and Luxton (2003) also found that individuals who engage in high levels of emotional suppression experienced later increases in rumination, suggesting that suppression may contribute to rumination. Liverant, Kamholz, Sloan, and Brown (2011) again confirmed that rumination is associated with suppression in depression, but also found that emotional acceptance is negatively correlated with rumination. Other research has demonstrated that emotional processing is related to an increased pattern of rumination in males (Stanton, Kirk, Cameron, & Danoff-Burg, 2000). Taken together, these results seem to indicate that there is a link between emotion regulation strategies and rumination for individuals with depression.

Cognitive inhibition and rumination are also thought to be related constructs. A number of theorists have proposed that the inability to restrict or remove negative information from working memory sets the stage for rumination (Hertel, 1997; Joormann, 2010). In fact, Joormann (2006) found that depressed individuals with a tendency to ruminate exhibited a lack of inhibition for emotionally valent words despite being instructed to ignore them, whereas, individuals who scored lower on a rumination scale showed adequate inhibition. Zetsche, D'Avanzato, and Joormann (2012) also found that rumination was associated with deficits in the

ability to remove irrelevant negative information from working memory. Research investigating executive processes also found that the activation of rumination caused inhibitory deficits in dysphoric subjects only (Philippot & Brutoux, 2008). Thus, these studies support the idea that reduced inhibition is associated with increased rumination.

As noted, some theorists have proposed that cognitive inhibition may serve as a specific emotion regulation strategy relevant to mood disorders. Therefore, it seems reasonable to suggest that these two factors may be related. Although this relationship has gone mostly unexplored, one study has attempted to investigate a possible link. In particular, Joormann and Gotlib (2010) found that less effective inhibition for negative material was associated with increased use of emotional suppression, but decreased use of reappraisal strategies. This pattern of emotion regulation strategies was in turn correlated with more depressive symptoms. Therefore, the authors interpreted that these results indicate that deficits in cognitive inhibition, due to depression, are related to dysfunctional emotion regulation. Although this study provides support for an association, further research is clearly needed.

Summary of Literature

Research has thoroughly established that links exist between depression and each of the aforementioned individual constructs. However, even though evidence suggests the existence of strong correlations between rumination, emotion regulation, cognitive inhibition, sleep and diet with the state of depression, there is still substantial uncertainty as to whether these constructs might also play a causal role in the maintenance of depression. In terms of possible relationships between these constructs, research suggests a correlation exists between sleep and rumination, as well as between rumination and emotion regulation in the context of depression. Limited research also supports the idea that rumination is related to cognitive inhibition within

individuals with depression. Theorists have also proposed that a link exists between cognitive inhibition and emotion regulation, however, only a single study has attempted to explore the idea. Explorations of relationships between the remaining depressive constructs have been largely absent. Thus, there is a clear lack of information regarding possible associations between these constructs within depressed individuals.

The Present Study

As mentioned, there has been no research conducted to investigate a link between cognitive inhibition and sleep or between for emotional regulation and sleep. Studies have also failed to examine diet or appetite in relation to rumination, cognitive inhibition or emotion regulation. Therefore, the present study extended previous research by examining how these variables are related to one another in depression. Investigating whether these constructs are related can help us to gain a better understanding of the disorder itself. Furthermore, since these constructs may be possible maintenance variables, a greater understanding of these constructs may provide insights in the process of depression maintenance. In terms of clinical applications, understanding relationships between factors that may perpetuate the depressive episode could potentially also be helpful in allowing us develop more effective intervention strategies.

To examine these questions, participants completed questionnaires related to rumination, emotion regulation, sleep, and personal diet to identify the quality of these factors or whether there was a presence of such depressive correlates. Participants also answered depression questionnaires so that current and past levels of depressive symptomatology could be ascertained. In addition, participants completed a commonly used task to assess cognitive inhibition.

The present study assessed these constructs in individuals currently exhibiting depressive symptomatology, as well as in individuals with a history of depression who do not currently meet criteria for a major depressive episode (MDE). It has been suggested that in previously depressed individuals, the thinking patterns activated by negative mood will show similarities to patterns demonstrated during a current depressive episode. Teasdale (1988, 1997) has previously hypothesized that these reactivated patterns may act to maintain or even intensity the depressive mood state through ruminative cognitive processing. In fact, research investigating the cognitive patterns activated during a negative mood induction in individuals with and without previous depressive episodes have demonstrated the accuracy of such ideas (Ingram et al., 1998; Segal, Gemar, & Williams, 1999). Thus, investigating whether these depressive correlates are related in individuals with a past history of depression may be of interest. Furthermore, identifying whether group differences exist in the degree of these depressive correlates among individuals who are currently depressed and those who have been previously depressed may also be of interest.

Method

Participants

One hundred and thirty six participants were recruited from the pool of students enrolled in introductory psychology at the University of Kansas, each of whom received partial credit toward course requirements. This study recruited individuals who had no history of depression and were currently exhibiting an absence of depression, individuals who had a previous diagnosis of depression or who exhibited clinically significant levels of depressive symptoms in the past, and also individuals who were currently exhibiting depressive symptoms. These dimensions were assessed through questions from the mood module of the Structured Clinical Interview for the DSM-IV-TR and from the second edition of the Beck Depression Inventory

(BDI-II; Beck et al., 1996) which were included in the psychology study pool prescreen. Due to the nature of the computer task, only fluent English speakers were eligible for participation.

Of the 136 initially recruited, 122 of these participants meet criteria for one of the groups and participated in the study. Five participants endorsing current depressive symptoms at the time of the prescreen were excluded from analyses as they denied a prior history of depressive symptoms on the day of testing. An additional 20 participants were excluded since their symptoms could be accounted for by either bereavement or a general medical condition. Finally, data from seven participants were excluded since these participants endorsed symptoms consistent with bi-polar disorder. Therefore, of the 90 participants included in the data analysis, 30 were healthy controls, 30 were previously depressed, and 30 were exhibiting current depressive symptoms.

Inclusion Criteria

The Beck Depression Inventory possesses guidelines to be used for identifying individuals experiencing a current depressive episode. Thus, individuals had to score 14 or above in order to be included in the currently depressed group. In addition, self-report of the Structured Clinical Interview for the DSM-IV-TR allowed for the assessment of current and past major depressive episodes. Therefore, this questionnaire was also used to identify individuals for the currently depressed group based on DSM-IV-TR criteria. These individuals must have consistently experienced at least five of the depressive symptoms for at least a two week period. In addition, one of these symptoms must include depressed mood or a loss of interest in pleasurable activities.

Individuals in the previously depressed group were also selected through the BDI-II and self-report of the Structured Clinical Interview for the DSM-IV-TR. Previously depressed

participants had to meet criteria for a previous major depressive episode based on DSM-IV-TR criteria. In other words, these individuals had to endorse the past experience of five of the DSM-IV-TR depressive symptoms for at least a two week period. Again, one of these symptoms must have included depressed mood or a loss of interest in pleasurable activities. However, individuals in the previously depressed group must have scored 13 or below in order to meet inclusion criteria.

In order to meet criteria for the control group, participants had to score 7 or below on the BDI-II. Furthermore, these individuals must have endorsed the experience of only 3 or fewer of the DSM-IV-TR criteria depressive symptoms.

Measures

Demographic Questionnaire

Individuals answered questions pertaining to their demographic information. Specifically, these questions asked about age, gender, socioeconomic status, education level, and race or ethnicity. Of the 90 participants in the final sample, 57% (n=51) were female and 43% (n=39) were male. The average age of participants was 19.47 (SD=1.62) with a range of 18 through 30 years. Finally, the majority of participants described themselves as Caucasian (74%; n=67), while the remaining race representation was 7.8% African American (n=7), 7.8% Asian (n=7), 4.4% Hispanic (n=4), and 5.6% other (n=5). Participants did not significantly differ with respect to age, gender, ethnicity, education or income (see Table 1).

Beck Depression Inventory Second Edition

The second edition of the Beck Depressive Inventory (BDI-II; Beck et al., 1996) was used to select participants eligible for the study and to assess the severity of depressive symptoms. The BDI-II is a 21-item self-report measure and is one of the most common

questionnaires used to assess depressive symptoms. Individuals are asked to respond to each item based on a 0 to 3 scale in order to indicate the extent to which they have experienced a number of depressive symptoms within the past two weeks. Scores on the BDI-II can range from 0 to 63. Beck, Steer, and Brown (1996) suggested that scores should be interpreted based on the guidelines that scores below 14 indicate minimal depression, scores between 14 and 19 indicate mild depression, scores between 20 and 28 indicate moderate depression, and scores above 28 indicate clinically severe depression. The BDI-II has demonstrated good test-retest reliability, a great degree of convergent validity and adequate discriminant validity (Beck et al., 1996).

Structured Clinical Interview for the DSM-IV-TR

The Structured Clinical Interview for the DSM-IV-TR - Non-patient Edition (SCID-I/NP; First et al., 2002) is a commonly used semi-structured interview that assists clinicians in making DSM-IV-TR diagnoses. It consists of individual modules corresponding to both Axis I and Axis II disorders and each of the different classes of disorders. Questions related to each disorder help guide the interviewer through the process of determining whether the examinee meets criteria for a diagnosis. Thus, depression was also assessed through participants self-report of the unipolar depression section of the mood module. This self-report version contains 17 questions which ask participants to indicate whether they have experienced specific depressive symptoms. The instructions tell participants to answer the questions based on a time that they felt depressed, or during the period of time that they felt the worst if they have experienced more than one episode of depression. For each symptom, participants are also asked to indicate the duration of the symptom and when the symptom occurred. Therefore, self-report of this SCID module allows for the identification of either past or present depressive episodes. Questions are also included that ask about present medical conditions, use of medications, bereavement, and bi-polar symptoms.

Ruminative Responses Scale

The Response Styles Questionnaire (RRS; Nolen-Hoeksema & Morrow, 1991) is a measure designed to identify and assess four coping styles that individuals often use in response to depression. Thus, the questionnaire is comprised of four separate subscales which each assess one of the following coping styles: rumination, distraction, problem solving, and dangerous behaviors. The RRS is a 22-item subscale of the Response Styles Questionnaire (RSQ; Nolen-Hoeksema & Morrow, 1991) commonly used to assess aspects of depressive rumination. This scale assesses how often individuals engage in various responses during a negative mood state. Each item is scored on a four-point scale where responses indicate 1 = "almost never," 2 = "sometimes," 3 = "often," and 4 = "almost always." This scale has demonstrated good test–retest reliability (Nolen-Hoeksema, Parker, & Larson, 1994), and good internal consistency (Nolen-Hoeksema & Morrow, 1991). The RRS also possesses adequate convergent and predictive validity (Butler & Nolen-Hoeksema, 1994; Kuehner & Weber, 1999).

Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) is a self-report questionnaire used to assess sleep quality. The PSQI subjectively measures seven domains of sleep: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Individuals are asked to respond to 19 items based on the past month only. Scores are then used to create a global sleep score ranging from 0 to 21, with scores of 5 or above indicating poor sleepers. The PSQI has been shown to have good internal consistency and adequate test-retest reliability (Buysse et al., 1989). The PSQI has also demonstrated high degrees of convergent validity (Backhaus et al., 2002).

Emotion Regulation Questionnaire

The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is a 10-item questionnaire measuring the habitual use of emotion regulation strategies in response to both negative and positive affect. The measure consists of two sub-scales which assess individual use of cognitive reappraisal and expressive suppression. Item responses are based on a 7-point scale and range from “strongly disagree” to “strongly agree.” Previous research has shown good internal consistencies, test-retest reliabilities, and also demonstrated convergent and discriminate validity (Gross & John, 2003). Therefore, this questionnaire is considered to be a valid and reliable measure of these strategies.

The Emotion Amplification and Reduction Scales

The Emotion Amplification and Reduction Scales (TEARS; Hamilton et al., 2009) is an 18-item self-report questionnaire used to assess an individual's emotion regulation abilities, specifically through the amplification or reduction of emotional responses. Items are rated on a 4-point scale ranging from 1 = “not at all true of me” to 4 = “very true for me.” Scores on the TEARS can range between 18 to 72. TEARS has demonstrated high levels of criterion validity and internal consistency (Hamilton et al., 2009).

Diet Questionnaire

The Diet Questionnaire is an 8-item self-report measure developed for the purposes of the current study. It is intended to assess whether the individual has been adhering to a healthy or unhealthy diet over the past month. Individuals are presented with a list of types of foods and instructed to indicate how often they consume those foods. Food categories include fruits, vegetables, whole grains, healthy fats, fast food, desserts or sweets, fatty foods, and junk food. Examples of foods applicable to the category are provided for each grouping of food other than

fruits and vegetables. Item responses are based on a 5-point scale and range from “not at all true” to “extremely true.” This questionnaire provides both a Healthy Diet and Unhealthy Diet score.

It is important to note that there were many previously existing measures designed for the assessment of diet quality, which were considered for use within the current study. However, many of these instruments were designed for use in the context of eating disorders or obesity. Additionally, there are also numerous scales, such as the Food Frequency Questionnaire, that produced the desired diet quality ratings, yet, they were all too lengthy for the purposes of this study. It was only after an extensive search that the current measure was designed.

Profile of Mood States-Short Form

The Profile of Mood States-Short Form (POMS-SF; Shacham, 1983) is a self-report measure that was used to assess participant’s state positive and negative affect. This 37-item instrument consists of a list of descriptive words that convey a range of feelings. Participants were instructed to consider their feelings during the past 24 hours and mark each one-word adjective to indicate how accurately that item describes a mood-state they have experienced during this time period. The scale ranges from 0 to 4 for each item, with 0 indicating that the item was “not at all” accurate in describing how the participant felt, and 4 indicating that the item is “extremely” accurate. Results have indicated that the POMS-SF possess good psychometric properties similar to those of the original 65-item POMS. Furthermore, internal consistency estimates of the POMS-SF scales range from .80 to .91. Therefore, the POMS-SF is considered to be a useful alternative to the original POMS as there is little loss in validity and reliability (Shacham, 1983).

This measure also contains an 8-item depression subscale that specifically measures the individual’s depressed mood-state. Psychometric information has indicated that the validity of

the POMS-SF Depression subscale is not significantly lower than the Depression subscale from the original POMS questionnaire. Although the POMS-SF Depression subscale possess slightly lower internal consistency (POMS = .95, POMS-SF = .93), this scale has still been recommended for use.

Go/No-Go Task

Participants also completed a go/no-go computer task which allows for the effective study of cognitive inhibition within individuals exhibiting depressive symptoms (Murphy et al., 1999). In this design, participants were presented with one word at a time, either a target or a distracter. This go/no-go paradigm used emotionally valent stimuli as the targets and distracters. Participants were instructed to evaluate the valance of the target word as being either positive or negative for words that appeared in green letters. They were also instructed to not respond to distracter words that appeared in red letters. Thus, in each trial, the target words appeared in green letters and the distracter word appeared in red letters.

Response times and responses for each trial were collected through E-prime. These response times are considered to be a valid measure of the inhibitory processing of emotional information. The words used in this design were selected from the Affective Norms for English Words (ANEW) database which was developed to provide a set of normative words rated on emotional valence (Bradley & Lang, 1999). The negative and positive words are balanced to be similar on emotional valence, arousal and word length. The final set of 150 positive words had an average valence rating of M=7.725 (SD=0.43), an arousal rating of M=5.64 (SD=1.01), and the average word length was M=6.38 (SD=1.70). Whereas the final set of 150 negative words had an average valence rating of M=2.34 (SD=0.43), an average arousal rating of M=5.66 (SD=1.03) and the average word length was M=6.23 (SD=1.62).

Exactly 70% of the words appeared in green letters, with 35% being negatively valenced words and 35% being positively valenced words. The remaining 30% appeared in red letters, with 15% being negatively valenced words and 15% being positively valenced words. Each trial was designed so that a word was presented on the screen for 150 ms. Once the word was removed, a fixation cross appeared for 1500 ms. Participants were instructed to respond to each word as quickly as possible. Each participant completed 300 trials, for a total of 150 negative words and 150 positive words. This computer task took approximately 8.25 minutes.

Procedure

Participants were recruited through the psychology study pool prescreening which included questions from both the BDI-II and SCID-I/NP. After meeting the researcher, all individuals read and signed an informed consent which included the study description. Then, participants completed the BDI-II and the SCID-SR in order to determine eligibility. Participants who did not meet criteria were dismissed and awarded the appropriate number of credits. Participants who met criteria for the clinically depressed, formerly depressed, or the never-depressed control group completed the computer task next.

Participants were seated in front of a computer and instructions were displayed on the monitor. They were then be presented with two words, a target and a distractor, and asked to evaluate the valance of the target word as being either positive or negative. Participants first completed 3 practice trials, allowing the researcher to confirm that they understood the instructions. They then completed the remaining 300 trials. Upon completion of the computer task, participants completed the RSQ, the PSQI, the ERQ and the Appetite and Diet Scale in a randomized order. Finally, participants were debriefed and dismissed. The entire session lasted between 45 and 60 minutes for the majority of participants.

Results

Statistical Analyses

All of the administered questionnaires were used in the primary analyses, except for the SCID-SR which was used solely to determine research eligibility and to categorize participants into the appropriate group (depressed, previously-depressed, or never-depressed). Analysis of variance (ANOVA) and chi square tests were used in order to examine group differences in age, gender, ethnicity, education, and income. In addition, ANOVAs were conducted in order to determine whether group differences existed on any of the constructs of interest. All significant ANOVAs were followed up with post-hoc Tukey's tests.

It was decided *a priori* that reaction time analyses would exclude any trials in which the participant responded incorrectly to the normed valence of the word. In order to evaluate whether any of the groups demonstrated a bias in response time for negative versus positive words, a mixed 3 x 2 ANOVA was employed, consisting of a three-level between subjects factor for depression status (non-depressed, previously depressed, and currently depressed) and a two-level within-subjects factor for word valence (negative, positive). Analyses also examined errors of commission during no-go trials with red words. This element of whether the participants responded despite being instructed not to is what served as the measure of inhibition. Thus, analyses investigated whether any of the groups made higher rates of errors of commission.

In order to evaluate the possible relationships between the variables of interest, Pearson Product Moment correlations were calculated between each of the self-report measures and with the errors of commission. These calculations were conducted across the currently-depressed and control participants, as well as for both of these individual groups.

Descriptive information

Mood and Affect

A one-way between-subjects ANOVA was conducted to compare POMS-SF scores in control, previously-depressed, and currently-depressed conditions; results indicated that there were statistically significant differences among groups, $F(2, 87) = 17.85$, $p < .001$. Post hoc tests showed that the currently depressed group ($M = 59.07$) endorsed a significantly higher total mood disturbance score than either of the other groups (see table 1). In addition, the total mood disturbance of the previously depressed group ($M = 37.90$) was elevated above that of the control group ($M = 26.40$), although this was not a statistically significant difference. Analysis of the depression subscale of the POMS-SF indicated the presence of a statistically significant difference , $F(2, 87) = 19.07$, $p < .001$, such that currently depressed participants endorsed significantly higher state depression ($M = 10.33$) than previously depressed participants ($M = 3.93$) and control participants ($M = 1.43$). However, there was no statistically significant difference between the control and previously depressed groups.

Primary Analyses

Shapiro-Wilk tests were conducted on each of the variables of interest, for the control, previously-depressed, and currently depressed groups, in order to test whether the data fit a normal distribution. This data screening revealed that the control group possessed a slightly non-normal distribution of RRS scores. All other data followed a normal distribution. Therefore, normalization transformation techniques were not applied to data from the remaining variables of interest.

Further investigation of the control group RRS scores revealed a slight positive skew, skew = .49, which falls below the generally accepted absolute cutoff value of 2. In addition, the data possessed an absence of outliers and kurtosis indicated that the departure from normality

was not extreme (kurtosis = .98). Although, ANOVA's are relatively robust to violations of this assumption, the *F* test can be affected by small or unbalanced sample sizes. However, given that there were an equal number of participants across groups and the current study's sample size, it was unlikely that the ANOVA's power was significantly reduced. Therefore, it was determined that analysis through a one-way ANOVA was appropriate for these data without the use of transformation techniques.

Rumination

Not surprisingly, the ANOVA on the RRS scores revealed significant differences across groups, $F(2, 87) = 50.47$, $p < .001$. Specifically, the control group ($M = 33.60$) had statistically significantly lower rumination scores than the previously depressed group ($M = 50.07$) and the currently depressed group ($M = 60.10$). Furthermore, the previously depressed group also endorsed significantly lower rumination than the currently depressed group.

Sleep

Analyses of the PSQI demonstrated that there was a statistically significant difference between groups as determined by one-way ANOVA; $F(2, 87) = 11.88$, $p < .001$. The currently depressed group ($M = 8.27$) possessed significantly higher global sleep scores than the previously depressed group ($M = 6.03$) or the control group ($M = 4.67$). No significant difference existed between the control and previously depressed groups.

Emotion Regulation

ANOVAs were used to examine group differences on both the cognitive reappraisal and expressive suppression subscales of the ERQ. In terms of cognitive reappraisal, $F(2, 87) = 9.71$, $p < .001$, statistically significant differences were found using follow-up Tukey's tests between the both the currently depressed group ($M = 26.87$) and the control group ($M = 32.43$),

as well as between the currently depressed group and previously depressed group (M = 31.30). However, no statistically significant difference existed between the control and previously depressed group. In terms of expressive suppression, $F(2, 87) = 4.31$, $p = .016$, a statistically significant difference was found between the currently depressed group (M = 15.47) and control group (M = 12.10). No other significant differences were found.

The ANOVA on the TEARS emotional amplification scores revealed a significant effect of Group; $F(2, 87) = 3.19$, $p = .046$. Specifically, the currently depressed group (M = 2.43) endorsed significantly lower levels of emotional amplification on the TEARS than the control group (M = 2.87). The previously depressed group (M = 2.67) did not demonstrate statistically significant differences from either of the other groups. Furthermore, differences were revealed on TEARS emotional reduction scores as well; $F(2, 87) = 7.33$, $p = .001$. Both currently depressed (M = 2.27) and previously depressed participants (M = 2.49) also reported significantly lower levels of emotional reduction than control participants (M = 2.94). The currently depressed and previously depressed groups did not demonstrate a significant difference in use of emotional reduction.

Diet

Significant differences were not found between the control group, the previously depressed group, and the currently depressed group on the healthy subscale of the diet questionnaire. Likewise, there were no statistically significant differences found among the groups on the unhealthy subscale of this questionnaire.

Computer Task Analyses

A 3 (depression status: nondepressed, previously depressed, and currently depressed) x 2 (word valance: positive, negative) ANOVA revealed significant main effects for both Group,

$F(2, 14679) = 78.03$, $p < .001$, and Valance, $F(1, 14679) = 243.58$, $p < .001$, but no significant interaction. Means are listed in Table 2. In terms of valence, participants were quicker to identify positive words than negative words. Regarding depression status, both currently-depressed and previously-depressed individuals responded more quickly to emotional words than healthy controls. However, there was no statistically significant difference in response time between previously-depressed and currently-depressed participants.

Analyses also examined errors of commission during no-go trials with red words. A chi-square test revealed that a significant difference existed between groups in the number of responses to red words; χ^2 (2, N=740) = 12.76, $p = .002$. Follow-up tests revealed that control participants committed a greater number of errors of commission for positively valanced words than did previously depressed or currently depressed participants (see Figure 1). Whereas, for negatively valanced words, the currently depressed participants made greater rates of errors of commission (see Figure 2).

Relation Between Variables of Interest

In order to evaluate possible relationships between each of the variables of interest, Pearson Product Moment correlations were calculated between each of the self-report measures and the errors of commission across the sample of control and currently-depressed participants. Results indicated that rumination was significantly correlated with sleep, cognitive reappraisal, expressive suppression, emotional amplification, and emotional reduction. In addition, sleep was positively associated with expressive suppression and negatively associated with cognitive reappraisal and emotional amplification. Cognitive reappraisal was also related to the emotion regulation strategies of emotional amplification and emotional reduction. In addition, emotional amplification was positively correlated with emotional reduction. Not surprisingly, healthy diet

was negatively correlated with unhealthy diet, and inhibition of positive information was correlated with inhibition of negative information. All other correlations were non-significant (see Table 3).

As demonstrated, sleep was related to a number of the variables of interest. Thus, in order to fully examine the sleep component, the PSQI was divided into the seven comprising subscales which were then correlated with the other variables of interest (see Table 4). Subjective sleep quality was positively related to rumination, emotional suppression, and unhealthy diets, and also negatively related to cognitive reappraisal and emotional amplification. Sleep latency was significantly related to rumination and emotional suppression. Daytime dysfunction was positively related to rumination, and emotional suppression, and negatively related to cognitive reappraisal, emotional amplification and emotional reduction. Both sleep duration and sleep disturbances were positively correlated with rumination. In addition, habitual sleep efficiency was related to unhealthy diets.

Next, Pearson Product Moment correlations were calculated within the control and currently-depressed participant groups. For the control group, healthy diets were negatively correlated with unhealthy diets and emotional suppression (see table 5). The inhibition of positive information was positively associated with emotional reduction and the inhibition of negative information. Lastly, cognitive inhibition of negative information was associated with rumination. Again, the PSQI subscales were correlated with the other variables of interest. Although the overall PSQI score was unrelated to other depressive variables for the control group, a few subscales did exhibit associations (see Table 6). Subjective sleep quality was positively correlated with emotional suppression, and negatively correlated with emotional

amplification and healthy diets. Habitual sleep efficiency was negatively related to healthy diets. Lastly, daytime dysfunction was associated with emotional reduction.

In the currently depressed group, emotional amplification is positively correlated with cognitive reappraisal and healthy diets. Rumination is negatively correlated with both the inhibition of negative information and the inhibition of positive information. Like the other groups, the inhibition of positive information was associated with the inhibition of negative information. No other correlations were significant within groups (see table 7). In terms of the PSQI subscales, habitual sleep efficiency was positively associated with the inhibition of positive and negative stimuli and unhealthy diets (see Table 8). Sleep latency was also related to emotional suppression.

Regression Analyses

In order to further examine the relationship between these variables of interest, hierarchical linear regression analyses were conducted across the control and currently-depressed groups. For each regression model, group status was entered at Step 1. Eight sets of regressions were conducted that used: (a) rumination, (b) sleep, (c) cognitive reappraisal, (d) emotional suppression, (e) emotional amplification, (f) emotional reduction, (g) cognitive inhibition of positive stimuli, and (h) cognitive inhibition of negative stimuli as the dependent variables. The remaining variables of interest were used as predictors and were entered in Step 2 for each regression model. Diet was excluded from all regression analyses because there were no significant group differences for either the healthy or unhealthy subscale.

For the rumination regression, group status predicted rumination in step 1, $R = .81$, $F(1, 53) = 102.24$, $p < .01$ accounting for 65% of the variance in RRS total scores. In step 2, the sleep, emotional regulation, and cognitive inhibition variables were entered into the equation, $R = .87$,

$F(8, 46) = 17.81$, $p < .01$ accounting for 72% of the variance in RRS total scores. However, the only variable that has a significant unique contribution to the prediction of rumination was cognitive reappraisal (see Table 9).

For the second regression, group status predicted sleep in step 1, $R = .53$, $F(1, 53) = 20.69$, $p < .01$ accounting for 27% of the variance in PSQI total scores. In step 2, the rumination, emotional regulation, and cognitive inhibition variables were entered into the equation, $R = .62$, $F(8, 46) = 3.54$, $p < .01$ still accounting for only 27% of the variance in PSQI total scores. Additionally, none of the variables had a unique contribution for the prediction of sleep (see Table 10).

For the cognitive reappraisal regression, $R = .53$, $F(1, 53) = 20.43$, $p < .01$ group status accounted for 27% of the variance in step 1. In step 2, the rumination, sleep, cognitive inhibition and the remaining emotional regulation variables were entered into the equation, $R = .65$, $F(8, 46) = 4.18$, $p < .01$ and accounted for 32% of the variance in cognitive reappraisal scores (see Table 11).

For the emotional suppression regression, $R = .35$, $F(1, 53) = 7.60$, $p < .01$ group status accounted for 11% of the variance in step 1. In step 2, the rumination, sleep, cognitive inhibition and the remaining emotional regulation variables were entered into the equation, $R = .57$, $F(8, 46) = 2.70$, $p < .01$ and accounted for 20% of the variance in emotional suppression scores. Both emotional amplification and emotional reduction had a significant unique contribution to the prediction of emotional suppression (see Table 12).

For the emotional amplification regression, $R = .32$, $F(1, 53) = 5.99$, $p < .01$ group status accounted for 9% of the variance in step 1. In step 2, the rumination, sleep, cognitive inhibition and the remaining emotional regulation variables were entered into the equation, $R = .63$, $F(8,$

46) = 3.80, $p < .01$ and accounted for 29% of the variance in emotional amplification scores. Emotional suppression, emotional reduction, and the cognitive inhibition of positive information all had a significant unique contribution to the prediction of emotional amplification (see Table 13).

For the emotional reduction regression, $R = .42$, $F(1, 53) = 11.60$, $p < .01$ group status accounted for 16% of the variance in step 1. In step 2, the rumination, sleep, cognitive inhibition and the remaining emotional regulation variables were entered into the equation, $R = .66$, $F(8, 46) = 4.32$, $p < .01$ and accounted for 33% of the variance in emotional reduction scores. Emotional suppression, emotional amplification, and the cognitive inhibition of positive information all had a significant unique contribution to the prediction of emotional reduction (see Table 14).

For the cognitive inhibition of positive stimuli regression, $R = .06$, $F(1, 53) = .20$, *ns* group status accounted for 1% of the variance in step 1. In step 2, the rumination, sleep, emotional regulation and cognitive inhibition of negative stimuli variables were entered into the equation, $R = .91$, $F(8, 46) = 28.80$, $p < .01$ and accounted for 80% of the variance. Emotional reduction, emotional amplification, and the cognitive inhibition of negative information all had a significant unique contribution to the prediction of inhibiting positive stimuli (see Table 15).

For the cognitive inhibition of negative stimuli regression, $R = .01$, $F(1, 53) = .01$, *ns,* group status accounted for 1% of the variance in step 1. In step 2, the rumination, sleep, emotional regulation and cognitive inhibition of positive stimuli variables were entered into the equation, $R = .91$, $F(8, 46) = 25.90$, $p < .01$ and accounted for 79% of the variance. The only variable that has a significant unique contribution was the inhibition of positive stimuli (see Table 16).

Overall, these analyses revealed that rumination had a relationship with cognitive reappraisal, and cognitive reappraisal is also related to emotional amplification. Suppression is related to both emotional amplification and reduction. Emotional amplification is related to emotional reduction and the inhibition of positive information. Emotional reduction is also linked to the inhibition of positive information. Lastly, the inhibition of positive information and the inhibition of negative information are related.

Discussion

The purpose of the present study was to extend previous research by examining rumination, cognitive inhibition, emotion regulation, sleep and diet in order to determine whether these constructs are related to one another in the context of depression. As expected, results indicated that the currently depressed individuals reported higher levels of rumination, worse sleep quality, less frequent use of cognitive reappraisal, more frequent use of emotional suppression, and lower amplification and reduction of emotional responses as compared to the control group. These findings are consistent with previous literature indicating that individuals with depression are more likely to engage in rumination and have difficulties with sleep (Nolen-Hoeksema et al., 1994; Gregory et al., 2011). Seeing as how emotional suppression has been identified as a particularly harmful emotion regulation strategy, as compared to cognitive reappraisal, these results also support previous findings that depression is associated with dysfunctional emotion regulation (Ehring, Fischer, Schnulle, Bösterling, & Tuschen-Caffier, 2008). In addition, since depressed individuals reported a decreased ability to either intensify or reduce emotional responses, this further supports the idea that these individuals have difficulties effectively regulating their emotions.

Although there were no statistically significant differences among groups on the healthy or unhealthy subscales of the diet questionnaire, it is worth mentioning that the currently

depressed group endorsed the lowest consumption of healthy foods, as well as the highest consumption of unhealthy foods. Further, this lack of significant differences may in part be due to the nature of the diet questionnaire utilized in this study. It is possible that an extended likert-scale may have elicited increased accuracy and variability in participants' responses, which is turn, may have generated increased F values. Alternatively, use of a more comprehensive food-frequency questionnaire would likely have resulted in higher validity and reliability, which could have increased statistical power.

This research study also aimed to identify whether group differences exist in the degree of these depressive correlates among individuals who are currently depressed and those who have been previously depressed. Our results revealed that previously depressed individuals endorsed lower levels of rumination than currently depressed individuals; yet, these scores were also significantly higher than that of the controls. This supports prior suggestions that previously depressed individuals may exhibit thinking patterns which resemble the patterns demonstrated during a current depressive episode (e.g., Segal, Williams, Teasdale, & Gemar, 1996). Previously depressed individuals demonstrated better sleep quality and more frequent use of cognitive reappraisal than currently depressed individuals. Furthermore, we found no difference in the frequency of use of expressive suppression, or the ability to use emotional amplification or reduction, among currently and previously depressed individuals. Thus, a critical and unanticipated difference between the two groups is the finding that previously depressed participants sleep better and more often employ the beneficial strategy of cognitive reappraisal, as compared to their depressed counterparts. However, previously depressed participants displayed a trend for higher levels of expressive suppression and decreased ability to amplify or

reduce emotional responses, suggesting that maladaptive emotional reactivity may still characterize this population despite the use of cognitive reappraisal.

Our results revealed that both currently-depressed and previously-depressed individuals responded more quickly to emotional words than healthy controls. Interestingly, all three groups identified positive words more quickly that negative words. The finding indicates the presence of a processing bias for positive words in non-depressed individuals which is consistent with previous findings (Siegle et al., 2001). However, currently and previously depressed participants also responded faster to positively valanced words than negatively valanced words, which contrasts with previous research demonstrating a bias for negative information in depression (Siegle et al., 2002).

Regarding errors of commission, we found that control participants had difficulty inhibiting the processing of emotionally positive information, which again demonstrates the existence of a positive processing bias for non-depressed individuals. However, our results also demonstrate impairments in the inhibition of negative stimuli in individuals with current depressive symptomology, which is consistent with prior findings (Joormann & Gotlib, 2010). This result provides further support for Joormann's (2004) hypothesis of cognitive inhibition, which suggests that depression may be characterized by an inhibitory deficit for negative information, in the context of emotional information processing. In other words, it is likely that depressed individuals have a decreased ability to disinhibit from mood-congruent input.

Across the control and currently-depressed participants, rumination was significantly related to sleep which is consistent with previous findings (Thomsen, Mehlsen, Christensen, & Zachariae, 2003). It is likely that rumination activates emotional and cognitive arousal which in turn, makes it more difficult to fall asleep and increases sleep disturbances. Similarly, higher

levels of rumination were associated with lower levels of cognitive reappraisal, emotional amplification, and emotional reduction, as well as higher levels of emotional suppression. This result is again consistent with previous research suggesting that suppression is positively correlated with rumination (Wenzlaff & Luxton, 2003; Liverant, Kamholz, Sloan, & Brown, 2011). Taken as a whole, these findings indicate that emotional regulation is not only related to rumination, but also suggest that use of maladaptive strategies (e.g., suppression) may lead to increased rumination, whereas the use of functional strategies (e.g., cognitive reappraisal) is associated with lower levels of rumination.

Our results also showed sleep to be significantly correlated with cognitive reappraisal, emotional suppression, and emotional amplification. Higher levels of cognitive reappraisal were generally associated with better overall sleep quality. Similarly, increased ability to use emotional amplification was also related to better overall sleep quality. Whereas, higher levels of emotional suppression were associated with poorer overall sleep quality. These results could suggest that certain emotion regulation strategies mediate the relationship between depressed mood and sleep problems. Dysfunctional emotional regulation may generate increased emotional arousal and elevated cognitive distress, which consequently disrupts sleep patterns. Lastly, a positive association was found between the beneficial ability to amplify one's emotions and the ability to reduce one's emotions.

In terms of specific sleep symptoms, poor subjective sleep quality was associated with higher rumination, increased emotional suppression, and unhealthier diets. Whereas, when subjective sleep quality was described as better, this was related to increased use of cognitive reappraisal and the beneficial ability to amplify emotions. Similarly, daytime dysfunction is positively correlated with rumination and emotional suppression, and negatively correlated with

cognitive reappraisal, emotional amplification, and emotional reduction. Thus, it is likely that as maladaptive emotional regulation strategies are employed, daytime dysfunction increases. Whereas when beneficial emotional regulation strategies are employed, daytime dysfunction decreases. Habitual sleep efficiency was also related to unhealthy diets, such that diets become increasing unhealthy as habitual sleep becomes increasing inefficient. Rumination was also positively related to both sleep duration and sleep disturbances. That is, as rumination increases, sleep disturbances increase and sleep duration decreases.

The inhibition of positive information was positively associated with the inhibition of negative information within each of the individual groups. Interestingly, the currently depressed participants exhibited a lack of correlation between the consumption of healthy foods and the consumption of unhealthy foods. For control participants, there was a negative relationship between these two variables within this study. These findings suggest that reduced consumption of unhealthy foods indicates increased consumption of healthy foods, and vice versa, for these participants. Yet, this relationship does not exist among depressed individuals.

Within the control group, more frequent use of emotional suppression was associated with decreased consumption of healthy food items. The ability to inhibit negative information was also positively related to rumination. Since higher inhibition scores indicate decreased inhibition (more errors of commission), this likely suggests that an increased ability to inhibition negative input leads to reductions in rumination within never depressed individuals. Decreased inhibition of positive information was also related to increased use of beneficial emotional reduction. In the previously depressed group, emotional suppression was negatively associated with the advantageous use of emotional amplification. Again, this seems to suggest that

previously depressed individuals continue to struggle with effectively regulating their emotions, despite their recovery.

Subjective sleep quality was positively associated with emotional suppression and negatively associated with emotional amplification and healthy diets for this group. Again this indicates that poor subjective sleep quality relates to increased emotional suppression. Whereas, the use of emotional amplification and the increased consumption of healthy foods correlates with better sleep quality. It also appears that the increased consumption of healthy foods relates to the increased efficiency of habitual sleep. Lastly, daytime dysfunction was negatively correlated with emotional reduction. This indicates that as beneficial emotional reduction increases, daytime dysfunction decreases.

For currently depressed participants, emotional amplification was positively correlated with cognitive reappraisal and healthy diets. This finding likely reflects decreased use of both emotion regulation strategies and the infrequent intake of healthy foods among individual with depression. Furthermore, frequent rumination relates to a lack of cognitive inhibition for emotionally valent stimuli within the present group. This finding is consistent with theories proposing that an inability to restrict negative information from working memory sets the stage for rumination within depression (Hertel, 1997; Joormann, 2010). There is likely a strong link between these two factors such that cognitive inhibition deficits permit depressed individuals to attend to mood-congruent material which then activates the tendency to ruminate. For the sleep subscales, poor habitual sleep efficiency was related to unhealthy diets, such that as consumption of unhealthy foods increases, one's sleep becomes less efficient. Additionally, poor habitual sleep efficiency was positively correlated with a lack of inhibition of both positive and negative stimuli. This likely indicates that for those with depression, as the ratio of time spent in bed

versus time spent asleep worsens, these individuals subsequently have decreased abilities to inhibit emotional stimuli. Lastly, sleep latency was positively associated with emotional suppression. Thus, as emotional suppression increases, the length of time that one requires to fall asleep also increases.

In comparing correlations between groups, a number of patterns should be considered. For instance, there was a greater association between cognitive reappraisal and rumination for the currently depressed participants than for control participants. This likely indicates that as use of cognitive reappraisal decreases among those who are depressed, rumination subsequently increases. Therefore, this finding can be connected to appraisal theory which states that an individual's appraisal of a situation or event is what generates the affective response they experience (Lazarus, 1991). The use of cognitive reappraisal in response to negative material has been found to cause decreases in negative affect (e.g. Richards & Gross, 2000). If individuals with current depression are less likely to employ this strategy, then there is reason to believe that the resulting negative affect may trigger a ruminative response.

As mentioned, the ability to inhibit negative information was positively related to rumination among control participants. Yet, those exhibiting current depressive symptoms demonstrated a strong negative correlation. This indicates that for healthy individuals, the inhibition of negative information may be effective in reducing rumination. However, for those who are depressed, deficits in cognitive inhibition lead to increased rumination. Again, once negative information enters working memory this may lead to a process of rumination. Additionally, when we examine the relationship between emotional reduction and rumination across these groups, we see that the control group displays a very slight positive correlation and the currently-depressed group has some negative correlation. This seems to indicate that as

depressive symptoms arise, a relationship is formed between these variables. Specifically, for depressed individuals, increases in rumination translate to decreases in the beneficial use of emotional reduction. Likewise, this pattern is seen for cognitive reappraisal and unhealthy diets. The control group shows no correlation, while the currently-depressed group shows a negative correlation. Within the presence of depressive symptoms, it is likely that as cognitive reappraisal decreases, consumption of unhealthy foods increases.

For control participants we also see a correlation between overall sleep quality and healthy diets, yet this correlation does not exist for the depressed group. This suggests that increased sleep disturbances may lead to a lower intake of healthy foods. This is consistent with previous research demonstrating that individuals who do not receive enough sleep are less likely to eat adequate amounts of healthy foods (Garaulet et al., 2011). This may suggest that for those who have never been depressed, increased sleep disturbances or decreased sleep quality may lead to unhealthy food cravings or increased impulsiveness related to food choices.

Lastly, a correlation was seen between the inhibition of negative information and emotional reduction for the control participants only. This may indicate a link between these two factors, such that the inhibition of negative input precludes the need to effectively reduce one's emotions. Thus, it is likely that healthy individuals who are able to remove negative stimuli from working memory have less need to employ the strategy of emotional reduction. Whereas, there was no significant relationship observed between these two factors for the currently-depressed group. Therefore, the presence of deficits in the ability to inhibit this information, in those who are depressed, does not seem to indicate the increased use of this emotion regulation strategy.

Additionally, rumination was shown to be related to the inhibition of both positive and negative information for those who are currently depressed, but only related to the inhibition of

negative information among healthy individuals. Thus, individuals with depression show a unique association between rumination and the inhibition of positive information. While this finding may be surprising, it indicates that rumination, by nature, may be characterized by the processing of negative information. Whereas, depression may instead be characterized by the inability to inhibit any emotionally valanced cues. Therefore, the disinhibition of negative information may lead to rumination among those who are depressed, as well as those who have never been depressed.

Overall, the present study also found that previously-depressed individuals still seem to be plagued by difficulties effectively regulating their emotions. These participants endorsed significantly higher levels of rumination than healthy participants. In addition, those who were previously-depressed showed similar levels of expressive suppression, emotional amplification, and emotional reduction to those who are currently-depressed. This indicates that these coping strategies continue to resemble those of depressed individuals despite the absence of depressive symptomology. It may be that such difficulties put these individuals at an increased risk for depressive relapse. If so, improving emotion regulation strategies within this population may help to maximize recovery and prevent future relapses.

A number of regression analyses were also conducted in order to further examine any relationships between the variables of interest. Overall, these analyses revealed that rumination was related to cognitive reappraisal, which was consistent with the correlational analyses. The link between these two emotion regulation strategies suggests that as rumination increases, the use of cognitive reappraisal decreases regardless of depression status. Cognitive reappraisal was again confirmed to be positively related to the beneficial use of emotional amplification. Additionally, emotional amplification was again found to be linked to emotional reduction, such

that increases in one strategy leads to increases in the other. Lastly, the inhibition of positive information and the inhibition of negative information were again positively related. This suggests that as one's ability to inhibit negative stimuli increases, the ability to inhibit positive stimuli also increases, and vice versa.

These regressions also revealed suppression to be negatively related to emotional amplification and positively related to emotional reduction. Not surprisingly, this suggests that individuals who use emotional suppression are less likely to use emotional amplification, but more likely to use emotional reduction. Emotional amplification was found to be negatively related to the inhibition of positive information. Therefore, individuals who are able to beneficially amplify their emotions, show more of an ability to inhibit positive information. Furthermore, emotional reduction was also positively linked to the inhibition of positive information. This suggests that individuals who are able to beneficially reduce their emotions, may show more of an ability to inhibit positive information. In sum, a number of depressive processes, theorized to underlie and maintain depression, were found to be associated in the present study. Although we cannot ascertain causal relationships based on the present results, it is clear that many of these dysfunctional variables are related to higher levels of maladaptive factors and lower levels of adaptive factors (e.g., disrupted sleep is related to poor dietary quality, frequent rumination correlated with infrequent cognitive reappraisal). However, regardless of how these variables interact, it is likely that they reinforce each other within the context of depression.

As discussed, each of these depressive symptoms may act as potential maintenance variables. The interaction of such variables would undoubtedly influence the duration of the depressive disorder. In addition, if these dysfunctional processes do underlie and maintain the

depressive state is may be important to address them in treating the disorder. For example, targeting symptoms which interact with other problem areas may assist in achieving a larger reduction in overall symptoms than do strategies that target distal or extraneous symptoms. These findings can be viewed as an important initial step since identifying the relationship between symptoms and those that prolong the depression, in particular, would help us to develop more effective intervention strategies.

Limitations and Future Directions

This study possessed several limitations which should be acknowledged. Although depression symptoms were assessed through two separate measures, a self-report version of the Structured Clinical Interview for the DSM-IV-TR was used in the interest of time. Therefore, the diagnoses of currently depressed and previously depressed are not definitive in terms of a Major Depressive Episode. It is thus possible that the present findings might differ from studies in which depressive status is confirmed by a structured diagnostic interview.

Although the results of the current study yield insights concerning which depressive variables are linked, we cannot discern the nature of the relationship between constructs. These variables almost certainly interact, but we cannot determine if certain symptoms develop first and instigate the formation of subsequent symptoms, or if these constructs are concurrently generated and then build upon or intensify each other. Future studies should attempt to use a longitudinal design to further clarify how these variables are related both within and out of a depressive episode.

It is also worth mentioning that although individuals formally diagnosed with bi-polar disorder were excluded from enrollment in this study, others were subsequently excluded from analyses if they endorsed symptoms consistent with bi-polar disorder. However, such decisions

were made subjectively rather than by standardized cut-offs, such as through the use of the bi-polar module of the SCID-I/NP. Furthermore, in order to reduce the likelihood of including confounding variables, the present study assessed for current medical conditions, use of psychiatric medications, bereavement, and bi-polar symptoms. Yet, in future analyses, careful consideration should be made to determine whether additional variables would be appropriate to use as exclusion criteria.

Conclusions

The present study examined the relationships between rumination, sleep, diet, emotion regulation, and cognitive inhibition in the context of current and previous depressive symptomology, which revealed several potentially important findings. First, deficits in the inhibition of negative stimuli were observed within individuals with current depressive symptomology which confirms previous theories. The results also suggested that maladaptive emotional reactivity may be a remnant of previous functioning among those with a past history of depression, as the current sample demonstrated emotion regulation trouble despite their lower levels of depressive symptoms. Finally, interesting correlations were revealed within the control and currently-depressed groups as well. Notably, for the currently depressed group, frequent rumination was related to a lack of cognitive inhibition. This suggests that cognitive inhibition deficits may permit this population to attend to mood-congruent material which then subsequently activates the tendency to ruminate. Correlational patterns also indicated that as use of cognitive reappraisal decreases among those who are depressed, rumination subsequently increases. In turn, such increases in rumination translate to decreases in the beneficial use of emotional reduction. Furthermore, within the presence of depressive symptoms, it is likely that as cognitive reappraisal decreases, consumption of unhealthy foods increases. Although

replication is needed, the results from this study demonstrate the existence of relationships between depressive constructs. Future studies should further explore these relationships in order to gain a more clear understanding. Identifying the relationship between factors that prolong the depressive state would be an important step in increasing our knowledge of risk and causality, the disorder itself, and potentially help us develop more effective intervention strategies. Such research would also shed light on the process of depressive maintenance – an area largely overlooked within the literature.

References

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., Kim, R. S., & Lapkin, J. B. (2000). The Temple–Wisconsin Cognitive Vulnerability to Depression (CVD) project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive vulnerability to depression. Journal of Abnormal Psychology, 109, 403–418. doi:I0.1037//0021-S43X.109.3.403

Akbaraly, T. N., Brunner, E. J., Ferrie, J. E., Marmot, M. G., Kivimaki, M., & Singh-Manoux, A. (2009). Dietary pattern and depressive symptoms in middle age. *British Journal of Psychiatry, 195*, 408–413. doi:10.1192/bjp.bp.108.058925

American Psychiatric Association. (2000). Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision. Washington, DC: American Psychiatric Association.

Backhaus, J., Junghanns, K., Broocks, A., Riemann, D., & Hohagen, F. (2002). Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *Journal of Psychosomatic Research, 53*, 737– 740. doi:10.1016/S0022-3999(02)00330-6

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Harper & Row.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck Depression Inventory, 2nd ed*. San Antonio, TX: The Psychological Corporation.

Boland, R. J., & Keller, M. B. (2010). Course and Outcome of Depression. In I. H. Gotlib, & C. L. Hammen (Eds.), *Handbook of depression* (2nd ed.) (pp. 23-43). New York: Guilford.

Bradley, M. M., & Lang, P. J. (1999). *Affective norms for English words (ANEW): Stimuli, instruction manual and affective ratings. Technical Report C-1*. Gainesville, FL: The Center for Research in Psychophysiology, University of Florida.

Breslau, N., Roth, T., Rosenthal, L., & Andreski, P. (1996). Sleep disturbance and psychiatric disorders: A longitudinal epidemiological study of young adults. *Biological Psychiatry, 39,* 411-418. doi: 10.1016/0006-3223(95)00188-3

Butler, L. D., & Nolen-Hoeksema, S. (1994). Gender differences in responses to a depressed mood in a college sample. *Sex Roles*, *30,* 331–346. doi:10.1007/BF01420597

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburg Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*, 193–213. *doi:*10.1016/0165-1781(89)90047-4

Campbell-Sills, L., & Barlow, D. H. (2006). Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 542–559). New York: Guilford.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion, 6*(4), 587–595. doi: 10.1037/1528-3542.6.4.587

Carney, C.E., Edinger, J.D., Meyer, B., Lindman, L, Istre, T. (2006). Symptom-focused rumination and sleep disturbance. *Behavioral Sleep Medicine,* 4, 228-241. doi:10.1207/s15402010bsm0404_3

Christensen, L. (1996). Nutrition and depression. In L. Christensen (Ed.), *Diet–behavior relationships: Focus on depression* (pp. 89-108). Washington, DC: American Psychological Association.

Christensen, L., & Burrows, R. (1990). Dietary treatment of depression. *Behavior Therapy, 21,* 183-193. doi:http://dx.doi.org.10.1016/S0005-7894(05)80276-X

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

Ehring, T., Fischer, S., Schnulle, J., Bösterling, A., & Tuschen-Caffier, B. (2008). Characteristics of emotion regulation in recovered depressed versus never depressed individuals. *Personality and Individual Differences, 44,* 1574–1584. doi:10.1016/j.paid.2008.01.013

Ehring, T., Tuschen-Caffier, B., Schnuelle, J., Fischer, S., & Gross, J. J. (2010). *Emotion regulation and vulnerability to depression: Spontaneous versus instructed use of emotion suppression and reappraisal. Emotion,* 10, 563-572. doi:10.1037/a0019010

Friedman, N.P., & Miyake, A. (2004). The relations among inhibition and interference control functions: A latent-variable analysis. *Journal of Experimental Psychology: General,* 133, 101–135. doi:10.1037/0096-3445.133.1.101

Frings, C., Wentura, D., & Holtz, M. (2007). Dysphorics cannot ignore unpleasant information. *Cognition and Emotion*, 21, 1525-1534. doi:10.1080/02699930601054042

Garaulet, M., Ortega, F. B., Ruiz, J. R., Rey-López, J. P., Béghin, L., Manios, Y., Cuenca-García, M., Plada, M., Diethelm, K., Kafatos, A., Molnár, D., Al-Tahan, J., & Moreno, L. A. (2011). Short sleep duration is associated with increased obesity markers in European adolescents: Effect of physical activity and dietary habits. The HELENA study. *International Journal of Obesity*, 35, 1308-1317. doi:10.1038/ijo.2011.149

Goeleven, E., DeRaedt, R., Baert, S., & Koster, E. H. (2006). Deficient inhibition of emotional information in depression. *Journal of Affective Disorders, 93,* 149–157. doi:10.1016/j.jad.2006.03.007

Gregory, A. M., Buysse, D. J., Willis, T. A., Rijsdijk, F. V., Maughan, B., Rowe, R., Cartwright, S., Barclay, N. L., & Eley, T. C. (2011). Associations between sleep quality and anxiety and depression symptoms in a sample of young adult twins and siblings. *Journal of Psychosomatic Research*, 71, 250-255. doi:10.1016/j.jpsychores.2011.03.011

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85*, 348-362. doi:10.1037/0022-3514.85.2.348

Hamilton, N. A., Karoly, P., Gallagher, M., Stevens, N., Karlson, C., & McCurdy, D. (2009). The assessment of emotion regulation in cognitive context: The emotion amplification and reduction scales. *Cognitive Therapy and Research, 33*, 255-263. doi:10.1007/s10608-007-9163-9

Harvey, A. G. (2000). Pre-sleep cognitive activity: A comparison of sleep-onset insomniacs and good sleepers. *British Journal of Clinical Psychology*, 39, 275-286. doi:10.1348/014466500163284

Harbottle, L., & Schonfelder, N. (2008). Nutrition and depression: A review of the evidence. *Journal of Mental Health*, *17*, 576–587. doi:10.1080/09638230701677746

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.

Hathaway, S. R., & McKinley, J. C. (1943). *The Minnesota Muhiphasic Personality Inventory*. New York: The Psychological Corporation.

Hertel, P. T. (1997). On the contribution of deficient cognitive control to memory impairments in depression. *Cognition and Emotion*, 11, 569-583. doi:10.1080/026999397379890a

Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). *Cognitive vulnerability to depression*. New York: Guilford Press.

Jacka, F. N., & Berk, M. (2007). Food for thought. *Acta Neuropsychiatrica, 19*, 321–323. doi:10.1111/j.1601-5215.2007.00246.x

Jacka, F. N., Pasco, J. A., Mykletun, A., Williams, L. J., Hodge, A. M., O'Reilly, S. L., Nicholson, G. C., Kotowicz, M. A., & Berk, M. (2010). Association between western and traditional diets and depression and anxiety in women. *American Journal of Psychiatry, 167,* 305-311. doi:10.1176/ajp.2009.09060881.

Joormann, J. (2004). Attentional bias in dysphoria: The role of inhibitory processes. *Cognition and Emotion*, 18, 125-147. doi:10.1080/02699930244000480

Joormann, J. (2006). Differential Effects of Rumination and Dysphoria on the Inhibition of Irrelevant Emotional Material: Evidence from a negative priming task. *Cognitive Therapy and Research*, 30, 149–160. doi:10.1007/s10608-006-9035-8

Joormann, J. (2010). Cognitive Inhibition and Emotion Regulation in Depression. *Current Directions in Psychological Science*, 19, 161-166. doi:10.1177/0963721410370293

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion, 24*, 281-298. doi:10.1080/02699930903407948

Joormann, J., Yoon, K.L., & Zetsche, U. (2007). Cognitive inhibition in depression. *Applied and Preventive Psychology*, 12, 128-139. doi:10.1016/j.appsy.2007.09.002

Kales, A., Caldwell, A. B., Soldatos, C. R., Bixler, E. O., & Kales, J. D. (1983). Biopsychobehavioural correlates of insomnia. II. Pattern specificity and consistency with the Minnesota Multiphasic Personality Inventory. *Psychosomatic Medicine, 45*, 341–356.

Keller, M. B., Klerman, G. L., Lavori, P. W., Coryell, W., Endicott, J., & Taylor, J. (1984). Long-term outcome of episodes of major depression: Clinical and public health significance. *Journal of American Medical Association, 252*, 788-792. doi:10.1001/jama.1984.03350060032024

Kendall, P. C., Hollon, S. D., Beck, A. T., Hammen, C. L., & Ingram, R. E. (1987). Issues and recommendations regarding use of the Beck Depression Inventory, *Cognitive Therapy and Research, 11*, 289-299. doi:http://dx.doi.org.2048/10.1007/BF01186280

Kloss, J. D., & Szuba, M. P. (2003). Insomnia in psychiatric disorders. In M. P. Szuba, J. D. Kloss, & D. F. Dinges (Eds), *Insomnia: Principles and Management* (pp. 43–70). New York: Cambridge University Press.

Kocsis, J. H., Gelenberg, A. J., Rothbaum, B., Klein, D. N., Trivedi, M. H., Manber, R., Keller, M. B., Howland, R. H., & Thase, M. E. (2008). Chronic forms of major depression are still undertreated in the 21st century: systematic assessment of 801 patients presenting for treatment. *Journal of Affective Disorders, 110*, 55–61. doi:10.1007/s11414-009-9194-y

Kraemer, H. C., Stice, E., Kazdin, A., Offord, D., & Kupfer, D. (2001). How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. *American Journal of Psychiatry, 158*, 848-856.

Kuehner, C., & Weber, I. (1999). Responses to depression in unipolar depressed patients: An investigation of Nolen-Hoeksema's response styles theory. *Psychological Medicine, 29*, 1323-1333. doi:10.1017/S0033291799001282

Lazarus, R. S. (1991). Emotion and adaptation. New York: Oxford University Press.

Liverant, G. I., Kamholz, B. W., Sloan, D. M., & Brown, T. A. (2011). Rumination in clinical depression: A type of emotional suppression? *Cognitive Therapy and Research, 35,* 253–265. doi:10.1007/s10608-010-9304-4

Lyubomirksy, S., & Nolen-Hoeksema, S. (1993). Self-perpetuating properties of dysphoric rumination. *Journal of Personality and Social Psychology, 65,* 339–349. doi:10.1037/0022-3514.65.2.339

Matthews, G., & Southall, A. (1991). Depression and the processing of emotional stimuli: A study of semantic priming. *Cognitive Therapy and Research, 15,* 238-302. doi:10.1007/BF01205174

Mayers, A. G., & Baldwin, D. S. (2006). The relationship between sleep disturbance and depression. *International Journal of Psychiatry in Clinical Practice,* 10, 2-16. doi:10.1080/13651500500328087

Mazure, C. M. (1998). Life Stressors as Risk Factors in Depression. *Clinical Psychology: Science and Practice, 5,* 291–313. doi: 10.1111/j.1468-2850.1998.tb00151.x

Mendelson, W. B., Gillin, J. C., & Wyatt, R. D. (1977). *Human sleep* and *its disorders. New York: Plenum Press.*

Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: A meta-analysis. *Psychological Bulletin,* 128, 638–662. doi:10.1037/0033-2909.128.4.638

Moulds, M. L., Kandris, E., Starr, S., & Wong, A. C. M. (2007). The relationship between rumination, avoidance and depression in a non-clinical sample. *Behaviour Research and Therapy, 45,* 251–261.

Murphy, F. C., Sahakian, B. J., Rubinsztein, J. S., Michael, A., Rogers, R. D., Robbins, T. W., & Paykel, A. S. (1999). Emotional bias and inhibitory control processes in mania and depression. *Psychological Medicine,* 29, 1307–1321. doi:10.1017/S0033291799001233

Nanri, A., Kimura, Y., Matsushita, Y., Ohta, M., Sato, M., Mishima, N., Sasaki, S., & Mizone, T. (2010). Dietary patterns and depressive symptoms among Japanese men and women. *European Journal of Clinical Nutrition, 64,* 832–839. doi:10.1038/ejcn.2010.86

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., & 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., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology, 102*, 20–28. doi:10.1037/0021-843X.102.1.20

Nolen-Hoeksema, S., Parker, L., & 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

Philippot, P., & Brutoux, F. (2008). Induced rumination dampens executive processes in dysphoric young adults. *Journal of Behavior Therapy and Experimental Psychiatry, 39,* 219-27. doi:10.1016/j.jbtep.2007.07.001

Pietromonaco, P. R., & Markus, H. (1985). The nature of negative thoughts in depression. *Journal of Personality and Social Psychology, 48*, 799-807. doi:10.1037/0022-3514.48.3.799

Richards, J. M., & Gross, J. J. (2000). Emotion regulation and memory: The cognitive costs of keeping one's cool. Journal of Personality & Social Psychology, 79, 410-424. doi:10.1037/0022-3514.79.3.410

Rocha Araujo, D. M., Vilarim, M. M., & Nardi, A. E. (2010). What is the *effectiveness of the use of polyunsaturated fatty acid omega-3 in the treatment of depression? Expert Review of Neurotherapeutics, 10*, 1117–1129. doi:10.1586/ern.10.77

Rottenberg, J. (2007). Major depressive disorder: Emerging evidence for emotion context insensitivity. In J. Rottenberg & S.L. Johnson (Eds.), *Emotion and psychopathology: Bridging affective and clinical science* (pp. 151–165). Washington, DC: American Psychological Association.

Rude, S. S., & McCarthy, C. T. (2003). Emotional functioning in depressed and depression-vulnerable college students. *Cognition & Emotion, 17*, 799–806. doi:10.1080/02699930302283

Sanchez-Villegas, A., Delgado-Rodriguez, M., Alonso, A., Schlatter, J., Lahortiga, F., Serra Majem, L., & Martínez-González, M. A. (2009). Association of the Mediterranean dietary pattern with the incidence of depression: The Seguimiento Universidad de Navarra/University of Navarra follow-up (SUN) cohort. *Archives of General Psychiatry, 66*, 1090–1098. doi:10.1001/archgenpsychiatry.2009.129

Sánchez-Villegas, A., Verberne, L., De Irala, J., Ruíz-Canela, M., Toledo E., *Serra*-Majem, L., & Martínez-González, M. A. (2011). *Dietary fat intake and the risk of depression: The SUN Project. PLoS ONE, 6,* 1-7. doi:10.1371/journal.pone.0016268

Sbarra, D. A., & Allen, J. J. (2009). Decomposing depression: On the prospective and reciprocal dynamics of mood and sleep disturbances. Journal of Abnormal Psychology, 118, 171-82. doi:10.1037/a0014375

Segal, Z. V., Gemar, M., & Williams, S. (1999). Differential cognitive response to a mood challenge following successful cognitive therapy or pharmacotherapy for unipolar depression. *Journal of Abnormal Psychology*, *108,* 3-10. doi:10.1037/0021-843X.108.1.3

Segal, Z. V., Williams, J. M. G., Teasdale, J. D., & Gemar, M. (1996). A cognitive science perspective on kindling and episode sensitization in recurrent affective disorder. *Psychological Medicine, 26*, 371–380. doi:10.1017/S0033291700034760

Shacham, S. (1983). A shortened version of the Profile of Mood States. Journal of Personality Assessment, 47, 305-306. doi: 10.1207/s15327752jpa4703_14

Siegle, G. J., Granholm, E., Ingram, R. E., & Matt, G. E. (2001). Pupillary and reaction time measures of sustained processing of negative information in depression. *Biological Psychiatry, 49*, 7, 624-636. doi:10.1016/S0006-3223(00)01024-6

Siegle, G. J., Ingram, R. E., & Matt, G. E. (2002). Affective interference: An explanation for negative attention biases in dysphoria? *Cognitive Therapy and Research, 26*, 73-87. doi:10.1023/A:1013893705009

Spijker, J., de Graaf, R., Bijl, R. V., Beekman, A. T., Ormel, J., & Nolen, W. A. (2002). Duration of major depressive episodes in the general population: results from The Netherlands

Mental Health Survey and Incidence Study (NEMESIS). *British Journal of Psychiatry 181*, 208–213. doi:10.1192/bjp.181.3.208

Stanton, A. L., Kirk, S. B., Cameron, C. L., & Danoff-Burg, S. (2000). Coping through emotional approach: Scale construction and validation. Journal of Personality and Social Psychology, 78, 1150–1169. doi:10.1037/0022-3514.78.6.1150

Teasdale, J. D. (1983). Negative thinking in depression: Cause, effect, or reciprocal relationship? *Advances in Behavior Research and Therapy, 5,* 3–26. doi:10.1016/0146-6402(83)90013-9

Teasdale, J. D. (1988). Cognitive vulnerability to persistent depression. *Cognition and Emotion*, 2, 247-274. doi:10.1080/02699938808410927

Teasdale, J. D. (1997). The relationship between cognition and emotion: The mind-in-place in mood disorders. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behaviour therapy* (pp. 67-93). Oxford, England: Oxford University Press.

Thomsen, D. K. (2006). The association between rumination and negative affect: A review. *Cognition and Emotion, 20,* 1216–1235. doi:10.1080/02699930500473533

Thomsen, D. K., Mehlsen, M. Y., Christensen, S., & Zachariae, R. (2003). Rumination – relationship with negative mood and sleep quality. *Personality and Individual Differences*, 34, 1293-1301. doi:10.1016/S0191-8869(02)00120-4

Tsuno, N., Besset, A., & Ritchie, K. (2005). Sleep and Depression. *Journal of Clinical Psychiatry, 66,* 1254-1269. doi:10.4088/JCP.v66n1008

Wentura, D. (1999). Activation and inhibition of affective information: Evidence for negative priming in the evaluation task. *Cognition & Emotion, 13,* 65-91. doi:10.1080/026999399379375

Wenzlaff, R. M., & Luxton, D. D. (2003). The role of thought suppression in depressive rumination. Cognitive Therapy and Research, 22, 293–308.

World Health Organization. (2006). Mental Health; Depression Fact Sheet [Electronic Version] from http://www.who.int/mental_health/management/depression/definition/en/.

Yao, S., Liu, M., Liu, J., Hu, Z., Yi, J., & Huang, R. (2010). Inhibition dysfunction in depression: Event-related potentials during negative affective priming. *Psychiatry Research: Neuroimaging, 182,* 172-179. doi:10.1016/j.pscychresns.2010.01.010

Zetsche, U., D'Avanzato, C., & Joormann, J. (2012). Depression and rumination: Relation to components of inhibition. *Cognition & Emotion, 26,* 758-767. doi:10.1080/02699931.2011.613919

Table 1

Table 1: Summary of Participants' Descriptive Characteristics

	Controls *M* (SD)	Previously Depressed *M* (SD)	Currently Depressed *M* (SD)	All Participants *M* (SD)
N	30	30	30	90
Age	19.48 (1.33)	18.98 (0.66)	19.37 (3.89)	19.47 (1.62)
Gender (M/F)	14/16	20/10	17/13	51/39
Caucasian/White	76.7%	60%	86.7%	74.4%
African American	13.3 %	6.7%	3.3%	7.8%
Asian	6.7%	10.0%	6.7%	7.8%
Hispanic	3.3%	6.7%	3.3%	4.4%
Other Ethnicity	-	16.7%	-	5.6%
BDI-II Score	1.87 (2.03)	6.30 (3.68)	18.6 (4.77)	8.92 (7.99)
Number of DSM depressive symptoms	0.60 (0.96)	6.27 (1.17)	7.03 (1.29)	4.63 (3.10)
POMS-SF	26.40	37.90	59.07	41.12
POMS-SF: Depression subscale	1.43	3.93	10.62	5.23

BDI-II: Beck Depression Inventory – II
DSM: Diagnostic and Statistical Manual of Mental Disorders
POMS-SF: Profile of Mood States – Short Form

Table 2

Table 2:
Mean Reaction Times for Group and Valance on the Computer Task in milliseconds

Valance:

	Positive *M* (SD)	Negative *M* (SD)	Total *M* (SD)
All participants	521.7 (218.4)	578.7 (221.5)	548.2 (221.6)

Group:

	Control Participants *M* (SD)	Previously-Depressed Participants *M* (SD)	Currently-Depressed Participants *M* (SD)
All words	579.7 (219.9)	529.8 (211.7)	533.5 (230.1)

Table 3

Table 3: Summary of Pearson's correlations between variables of interest across the sample of control and currently-depressed participants.

	RRS	PSQI	ERQ-CR	ERQ-ES	TEARS-EA	TEARS-ER	Diet-H	Diet-U	CI-Positive
PSQI	0.52**								
ERQ-CR	-0.56**	-0.31*							
ERQ-ES	0.29*	0.34*	-0.09						
TEARS-EA	-0.32*	-0.27*	0.40**	-0.23					
TEARS-ER	-0.46**	-0.24	0.31*	0.04	0.36**				
Diet-H	-0.17	-0.13	0.15	-0.21	0.23	0.10			
Diet-U	0.19	0.25	-0.19	-0.03	-0.04	-0.03	-0.33**		
CI-Positive	0.00	0.12	-0.19	-0.14	-0.12	0.16	-0.01	0.01	
CI-Negative	0.09	0.10	-0.10	-0.04	-0.26	0.03	-0.08	-0.18	0.89**

Note: RRS: Response Styles Questionnaire; PSQI: Pittsburgh Sleep Quality Index; ERQ-CR: Emotion Regulation Questionnaire – Cognitive Reappraisal; ERQ-ES: Emotion Regulation Questionnaire – Expressive Suppression; TEARS-EA: The Emotion Amplification and Reduction Scales – Emotion Amplification; TEARS-ER: The Emotion Amplification and Reduction Scales – Emotion Reduction; Diet-H: Diet Questionnaire – Healthy Subscale; Diet-U: Diet Questionnaire – Unhealthy Subscale; CI-Positive – Cognitive Inhibition of Positive Information; CI-Negative – Cognitive Inhibition of Negative Information
*indicates $p < 0.05$; ** indicates $p < 0.01$.

Table 4

Table 4: Summary of Pearson's correlations between variables of interest and PSQI subscales across the sample of control and currently-depressed participants.

	Subjective Sleep Quality	Sleep Latency	Sleep Duration	Habitual Sleep Efficiency	Sleep Disturbances	Use of Sleeping Medication	Daytime Dysfunction
RRS	0.39**	0.30*	0.35**	0.18	0.26*	0.09	0.59**
ERQ-CR	-0.36**	-0.09	-0.25	-0.06	-0.12	-0.01	-0.41**
ERQ-ES	0.27*	0.41**	0.25	0.03	0.12	0.05	0.26*
TEARS-EA	-0.33*	-0.21	-0.05	0.03	-0.16	-0.07	-0.39**
TEARS-ER	-0.16	-0.13	0.07	-0.06	-0.10	-0.15	-0.51**
Diet-H	-0.16	-0.09	0.00	-0.11	-0.11	-0.04	-0.03
Diet-U	0.27*	0.07	0.20	0.37**	0.07	-0.06	0.05
CI-Positive	0.05	-0.08	0.14	0.22	0.03	0.19	-0.06
CI-Negative	0.19	0.14	0.16	-0.10	0.12	-0.07	-0.02

Note: RRS: Response Styles Questionnaire; PSQI: Pittsburgh Sleep Quality Index; ERQ-CR: Emotion Regulation Questionnaire – Cognitive Reappraisal; ERQ-ES: Emotion Regulation Questionnaire – Expressive Suppression; TEARS-EA: The Emotion Amplification and Reduction Scales – Emotion Amplification; TEARS-ER: The Emotion Amplification and Reduction Scales – Emotion Reduction; Diet-H: Diet Questionnaire – Healthy Subscale; Diet-U: Diet Questionnaire – Unhealthy Subscale; CI-Positive – Cognitive Inhibition of Positive Information; CI-Negative – Cognitive Inhibition of Negative Information

*indicates $p < 0.05$; ** indicates $p < 0.01$.

Table 5

Table 5: Summary of Pearson's correlations between variables of interest for control participants.

	RRS	PSQI	ERQ-CR	ERQ-ES	TEARS-EA	TEARS-ER	Diet-H	Diet-U	CI-Positive
PSQI	0.23								
ERQ-CR	-0.14	0.06							
ERQ-ES	-0.11	0.36	0.05						
TEARS-EA	-0.09	-0.08	0.24	-0.13					
TEARS-ER	0.08	-0.24	0.18	0.06	0.36				
Diet-H	-0.10	-0.34	0.13	-0.49**	-0.03	-0.16			
Diet-U	-0.13	0.16	-0.02	0.33	-0.17	0.33	-0.56**		
CI-Positive	0.33	-0.07	-0.30	-0.20	-0.11	0.39*	0.09	-0.07	
CI-Negative	0.38*	-0.04	-0.31	-0.23	-0.07	0.35	0.10	-0.11	0.99**

Note: RRS: Response Styles Questionnaire; PSQI: Pittsburgh Sleep Quality Index; ERQ-CR: Emotion Regulation Questionnaire – Cognitive Reappraisal; ERQ-ES: Emotion Regulation Questionnaire – Expressive Suppression; TEARS-EA: The Emotion Amplification and Reduction Scales – Emotion Amplification; TEARS-ER: The Emotion Amplification and Reduction Scales – Emotion Reduction; Diet-H: Diet Questionnaire – Healthy Subscale; Diet-U: Diet Questionnaire – Unhealthy Subscale; CI-Positive – Cognitive Inhibition of Positive Information; CI-Negative – Cognitive Inhibition of Negative Information

*indicates $p < 0.05$; ** indicates $p < 0.01$.

Table 6

Table 6: Summary of Pearson's correlations between variables of interest and PSQI subscales for control participants.

	Subjective Sleep Quality	Sleep Latency	Sleep Duration	Habitual Sleep Efficiency	Sleep Disturbances	Use of Sleeping Medication	Daytime Dysfunction
RRS	-0.11	0.01	0.28	0.16	0.20	0.12	0.17
ERQ-CR	-0.14	0.13	0.00	0.15	0.19	0.06	-0.20
ERQ-ES	0.36*	0.16	0.29	0.29	-0.13	0.05	0.13
TEARS-EA	-0.42*	-0.09	0.19	0.32	-0.00	-0.20	-0.33
TEARS-ER	-0.21	-0.24	0.33	0.02	-0.16	-0.14	-0.53**
Diet-H	-0.40*	-0.06	-0.36	-0.51**	0.26	-0.11	0.05
Diet-U	0.27	0.04	0.18	0.15	-0.23	-0.08	0.11
CI-Positive	0.08	0.02	0.14	-0.24	-0.07	-0.03	-0.15
CI-Negative	0.07	0.04	0.17	-0.22	-0.07	-0.06	-0.13

Note: RRS: Response Styles Questionnaire; PSQI: Pittsburgh Sleep Quality Index; ERQ-CR: Emotion Regulation Questionnaire – Cognitive Reappraisal; ERQ-ES: Emotion Regulation Questionnaire – Expressive Suppression; TEARS-EA: The Emotion Amplification and Reduction Scales – Emotion Amplification; TEARS-ER: The Emotion Amplification and Reduction Scales – Emotion Reduction; Diet-H: Diet Questionnaire – Healthy Subscale; Diet-U: Diet Questionnaire – Unhealthy Subscale; CI-Positive – Cognitive Inhibition of Positive Information; CI-Negative – Cognitive Inhibition of Negative Information
*indicates $p < 0.05$; ** indicates $p < 0.01$.

Table 7

Table 7: Summary of Pearson's correlations between variables of interest for currently-depressed participants.

	RRS	PSQI	ERQ-CR	ERQ-ES	TEARS-EA	TEARS-ER	Diet-H	Diet-U	CI-Positive
PSQI	-0.04								
ERQ-CR	-0.30	-0.13							
ERQ-ES	-0.01	0.12	0.15						
TEARS-EA	0.00	-0.14	0.37*	-0.14					
TEARS-ER	-0.26	0.16	0.03	0.35	0.16				
Diet-H	-0.10	0.07	0.06	-0.02	0.38*	0.22			
Diet-U	0.35*	0.21	-0.23	-0.23	0.17	0.06	-0.15		
CI-Positive	-0.44*	0.20	-0.09	-0.15	-0.11	0.05	-0.05	0.20	
CI-Negative	-0.43*	0.19	-0.08	-0.13	-0.10	0.03	-0.06	0.19	0.99**

Note: RRS: Response Styles Questionnaire; PSQI: Pittsburgh Sleep Quality Index; ERQ-CR: Emotion Regulation Questionnaire – Cognitive Reappraisal; ERQ-ES: Emotion Regulation Questionnaire – Expressive Suppression; TEARS-EA: The Emotion Amplification and Reduction Scales – Emotion Amplification; TEARS-ER: The Emotion Amplification and Reduction Scales – Emotion Reduction; Diet-H: Diet Questionnaire – Healthy Subscale; Diet-U: Diet Questionnaire – Unhealthy Subscale; CI-Positive – Cognitive Inhibition of Positive Information; CI-Negative – Cognitive Inhibition of Negative Information

*indicates $p < 0.05$; ** indicates $p < 0.01$.

Table 8

Table 8: Summary of Pearson's correlations between variables of interest and PSQI subscales for currently-depressed participants.

	Subjective Sleep Quality	Sleep Latency	Sleep Duration	Habitual Sleep Efficiency	Sleep Disturbances	Use of Sleeping Medication	Daytime Dysfunction
RRS	0.07	0.11	0.04	-0.16	0.00	-0.31	0.19
ERQ-CR	-0.23	-0.04	-0.22	0.01	-0.18	0.20	-0.12
ERQ-ES	0.05	0.47**	0.10	-0.19	0.12	-0.06	0.05
TEARS-EA	-0.13	-0.17	-0.04	0.00	-0.16	0.05	-0.20
TEARS-ER	0.19	0.20	0.19	0.08	0.16	-0.02	-0.18
Diet-H	0.01	-0.06	0.25	0.12	-0.30	0.01	0.07
Diet-U	0.20	0.02	0.14	0.45*	0.21	-0.12	-0.19
CI-Positive	0.01	-0.17	0.13	0.44*	0.08	0.25	-0.09
CI-Negative	-0.02	-0.16	0.15	0.45*	0.05	0.25	-0.13

Note: RRS: Response Styles Questionnaire; PSQI: Pittsburgh Sleep Quality Index; ERQ-CR: Emotion Regulation Questionnaire – Cognitive Reappraisal; ERQ-ES: Emotion Regulation Questionnaire – Expressive Suppression; TEARS-EA: The Emotion Amplification and Reduction Scales – Emotion Amplification; TEARS-ER: The Emotion Amplification and Reduction Scales – Emotion Reduction; Diet-H: Diet Questionnaire – Healthy Subscale; Diet-U: Diet Questionnaire – Unhealthy Subscale; CI-Positive – Cognitive Inhibition of Positive Information; CI-Negative – Cognitive Inhibition of Negative Information
*indicates $p < 0.05$; ** indicates $p < 0.01$.

Table 9

Table 9
Summary of Hierarchical Regression Analyses for the Prediction of Rumination controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	11344.756	1	11344.756	102.246	.000[b]
	Residual	5880.626	53	110.955		
	Total	17225.382	54			
2	Regression	13020.796	8	1627.599	17.807	.000[c]
	Residual	4204.586	46	91.404		
	Total	17225.382	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	13.25	1.41	0.78	0.00
Step 2:				
PSQI Global	0.83	0.49	0.15	0.10
ERQ Emotional Suppression	0.07	0.32	0.02	0.82
ERQ Cognitive Reappraisal	-0.61	0.28	-0.20	0.04
TEARS Amplification	-0.45	2.52	-0.02	0.86
TEARS Reduction	-3.14	2.32	-0.13	0.18
Cognitive Inhibition Positive	-1.39	0.93	-0.26	0.14
Cognitive Inhibition Negative	1.88	1.04	0.30	0.08

Note: $R^2 = 0.597$ for Step 1; $\Delta R2 = 0.036$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 10

Table 10
Summary of Hierarchical Regression Analyses for the Prediction of Sleep controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	161.953	1	161.953	20.694	.000[b]
	Residual	414.774	53	7.826		
	Total	576.727	54			
2	Regression	219.686	8	27.461	3.538	.003[c]
	Residual	357.041	46	7.762		
	Total	576.727	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	1.72	0.38	0.53	0.00
Step 2:				
RRS Total Score	0.07	0.04	0.38	0.10
ERQ Emotional Suppression	0.15	0.09	0.22	0.11
ERQ Cognitive Reappraisal	0.00	0.09	0.00	0.99
TEARS Amplification	0.16	0.73	0.03	0.83
TEARS Reduction	0.26	0.69	0.06	0.71
Cognitive Inhibition Positive	0.05	0.28	0.05	0.87
Cognitive Inhibition Negative	0.04	0.31	0.04	0.89

Note: $R^2 = 0.267$ for Step 1; $\Delta R2 = 0.006$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 11

Table 11
Summary of Hierarchical Regression Analyses for the Prediction of Cognitive Reappraisal controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	489.180	1	489.180	20.434	.000[b]
	Residual	1268.820	53	23.940		
	Total	1758.000	54			
2	Regression	740.175	8	92.522	4.181	.001[c]
	Residual	1017.825	46	22.127		
	Total	1758.000	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	-2.98	0.66	-0.53	0.00
Step 2:				
RRS Total Score	-0.15	0.07	-0.46	0.04
PSQI Score	0.00	0.25	0.00	0.99
ERQ Emotional Suppression	0.15	0.15	0.13	0.33
TEARS Amplification	2.19	1.20	0.26	0.07
TEARS Reduction	-0.05	1.16	-0.01	0.97
Cognitive Inhibition Positive	-0.10	0.47	-0.06	0.84
Cognitive Inhibition Negative	0.31	0.53	0.16	0.56

Note: $R^2 = 0.109$ for Step 1; $\Delta R2 = 0.092$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 12

Table 12
Summary of Hierarchical Regression Analyses for the Prediction of Emotional Suppression controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	167.492	1	167.492	7.597	.008[b]
	Residual	1168.435	53	22.046		
	Total	1335.927	54			
2	Regression	426.729	8	53.341	2.699	.016[c]
	Residual	909.198	46	19.765		
	Total	1335.927	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	1.75	0.63	0.35	0.01
Step 2:				
RRS Total Score	0.02	0.07	0.06	0.82
PSQI Score	0.37	0.23	0.24	0.11
ERQ Cognitive Reappraisal	0.14	0.14	0.16	0.33
TEARS Amplification	-2.62	1.11	-0.35	0.02
TEARS Reduction	2.37	1.04	0.35	0.03
Cognitive Inhibition Positive	-0.45	0.44	-0.30	0.31
Cognitive Inhibition Negative	0.17	0.50	0.10	0.74

Note: $R^2 = 0.109$ for Step 1; $\Delta R2 = 0.092$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 13

Table 13
Summary of Hierarchical Regression Analyses for the Prediction of Emotional Amplification controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	2.432	1	2.432	5.991	.018[b]
	Residual	21.517	53	.406		
	Total	23.949	54			
2	Regression	9.534	8	1.192	3.803	.002[c]
	Residual	14.415	46	.313		
	Total	23.949	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	-0.21	0.09	-0.32	0.02
Step 2:				
RRS Total Score	-0.00	0.01	-0.04	0.86
PSQI Score	0.01	0.03	0.03	0.83
ERQ Cognitive Reappraisal	0.03	0.02	0.27	0.07
ERQ Emotional Suppression	-0.04	0.02	-0.31	0.02
TEARS Reduction	0.33	0.13	0.36	0.02
Cognitive Inhibition Positive	-0.11	0.05	-0.57	0.04
Cognitive Inhibition Negative	0.05	0.06	0.22	0.41

Note: $R^2 = 0.085$ for Step 1; $\Delta R2 = 0.208$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 14

Table 14
Summary of Hierarchical Regression Analyses for the Prediction of Emotional Reduction controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	5.149	1	5.149	11.601	.001[b]
	Residual	23.523	53	.444		
	Total	28.671	54			
2	Regression	12.305	8	1.538	4.323	.001[c]
	Residual	16.367	46	.356		
	Total	28.671	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	-0.31	0.09	-0.42	0.00
Step 2:				
RRS Total Score	-0.01	0.01	-0.30	0.18
PSQI Score	0.01	0.03	0.05	0.71
ERQ Cognitive Reappraisal	0.00	0.02	-0.01	0.97
ERQ Emotional Suppression	0.04	0.02	0.29	0.03
TEARS Amplification	0.37	0.15	0.34	0.02
Cognitive Inhibition Positive	0.13	0.06	0.60	0.03
Cognitive Inhibition Negative	-0.10	0.07	-0.37	0.15

Note: $R^2 = 0.164$ for Step 1; $\Delta R2 = 0.166$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 15

Table 15
Summary of Hierarchical Regression Analyses for the Prediction of Cognitive Inhibition of Positive Stimuli controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	2.281	1	2.281	.199	.657[b]
	Residual	606.519	53	11.444		
	Total	608.800	54			
2	Regression	507.469	8	63.434	28.796	.000[c]
	Residual	101.331	46	2.203		
	Total	608.800	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	-0.20	0.46	-0.06	0.66
Step 2:				
RRS Total Score	-0.03	0.02	-0.18	0.14
PSQI Score	0.01	0.08	0.01	0.87
ERQ Cognitive Reappraisal	-0.01	0.05	-0.02	0.84
ERQ Emotional Suppression	-0.05	0.05	-0.07	0.31
TEARS Amplification	-0.79	0.37	-0.16	0.04
TEARS Reduction	0.80	0.35	0.17	0.03
Cognitive Inhibition Negative	1.00	0.08	0.85	0.00

Note: $R^2 = 0.015$ for Step 1; $\Delta R2 = 0.790$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Table 16

Table 16
Summary of Hierarchical Regression Analyses for the Prediction of Cognitive Inhibition of Negative Stimuli controlling for Group Status (N = 60).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.055	1	.055	.007	.935[b]
	Residual	436.381	53	8.234		
	Total	436.436	54			
2	Regression	357.137	8	44.642	25.896	.000[c]
	Residual	79.299	46	1.724		
	Total	436.436	54			

Predictor	B	SE B	β	*p*
Step 1: Group Status	0.03	0.39	0.01	0.94
Step 2:				
RRS Total Score	0.04	0.02	0.22	0.08
PSQI Score	0.01	0.07	0.01	0.89
ERQ Cognitive Reappraisal	0.02	0.04	0.05	0.56
ERQ Emotional Suppression	0.01	0.04	0.03	0.74
TEARS Amplification	0.28	0.34	0.07	0.41
TEARS Reduction	-0.46	0.32	-0.12	0.15
Cognitive Inhibition Positive	0.79	0.06	0.93	0.00

Note: $R^2 = 0.003$ for Step 1; $\Delta R2 = 0.784$ for Step 2. B = unstandardized coefficient. SE B = standard error. β = standardized coefficient.

Figure 1

Figure 1: Errors of commission during no-go trials for positively valanced words. Higher values indicate a greater number of errors of commission.

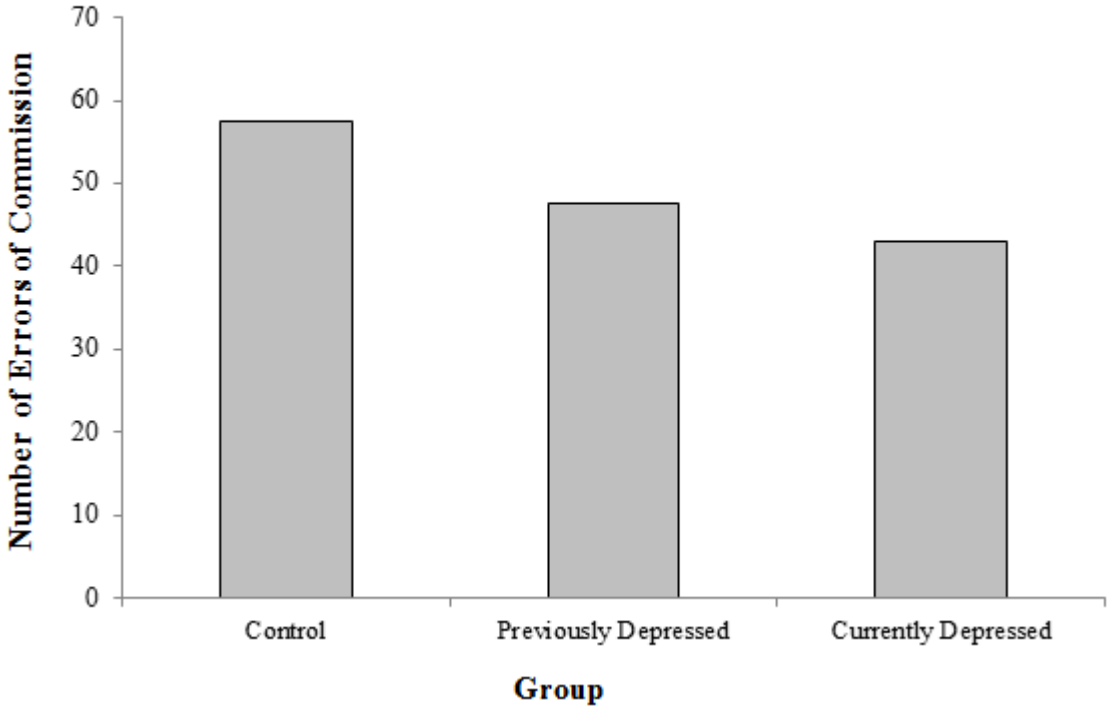

Figure 2

Figure 2: Errors of commission during no-go trials for negatively valanced words. Higher values indicate a greater number of errors of commission.