INTERPLAY BETWEEN STRESS, RUMINATION, AND MEMORY IN PREDICTING DEPRESSION: AN ECOLOGICAL MOMENTARY ASSESSMENT STUDY

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ABSTRACT

Rumination is a well-established vulnerability factor for major depressive disorder (MDD) that may exert deleterious effects both independently and in interaction with life stress, and may contribute to the negative memory biases associated with MDD. Chapter 1 examines the role of both momentary ruminative self-focus (MRS) and stress-reactive rumination (SRR) as predictors of increases in depressive symptoms utilizing a smartphone ecological momentary assessment (EMA) design. SRR, but not MRS, independently predicted increases in depressive symptoms. Interactions emerged between negative life events (NLEs) and both MRS and SRR, such that experiencing higher levels of NLEs and rumination at an observation predicted greater increases in depressive symptoms. The results suggest that rumination levels in response to stress vary within individuals and can have an important effect on depressed mood. Chapter 2 tests the hypotheses that 1) engaging in greater SRR relative to an individual's mean would lead to deeper encoding and improved retrieval of stressors, and 2) this biased memory for negative autobiographical information would predict increases in depressive symptoms over time. NLEs followed by increased SRR relative to individuals' means were significantly more likely to be recalled two weeks later. In addition, a significant interaction emerged between the number of NLEs experienced and proportional recall of those events, such that individuals who endorsed and recalled greater numbers of stressors during the EMA week displayed increased depressive symptoms at follow-up. These findings support the role of rumination and memory biases as vulnerability factors for depression, and suggest potential clinical benefits of modifying ruminative response styles to daily stressors.

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CHAPTER 1

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Abstract

Rumination is a well-established vulnerability factor for depression that may exert deleterious effects both independently and in interaction with life stress. The current study examined the role of both momentary ruminative self-focus (MRS) and stress-reactive rumination (SRR) as predictors of increases in depressive symptoms utilizing a smartphone ecological momentary assessment (EMA) design. A sample of 121 undergraduates responded to four text message alerts per day for one week in which they indicated the occurrence of negative life events (NLEs), rumination, and depressed mood. SRR, but not MRS, independently predicted increases in depressive symptoms, suggesting that engaging in introspective thought is not inherently maladaptive, but that it becomes detrimental when coupled with the experience of stress. Interactions emerged between NLEs and both MRS and SRR, such that experiencing higher levels of NLEs and rumination at an observation predicted greater increases in depressive symptoms. To our knowledge, the current study is the first to demonstrate that state rumination moderates, versus mediates, the effect of stress in prospectively predicting change in depressive symptoms using EMA methodology. The results suggest that rumination levels in response to stress vary within individuals and can have an important effect on depressed mood. These findings may have important clinical implications, as lessening individuals' tendency to engage in rumination directly following NLE occurrence may help to alleviate depressive symptoms over time.

Keywords: rumination, ecological momentary assessment, stress, depression

Rumination Interacts with Life Stress to Predict Depressive Symptoms: An Ecological Momentary Assessment Study

Major depressive disorder (MDD) is the most common psychiatric disorder, associated with major personal, societal, and economic costs (Birnbaum et al., 2010; Kessler, Merikangas, & Wang, 2007). Rumination, characterized by repetitive, passive thoughts about the symptoms, causes, and future repercussions of one's depression, is thought to perpetuate depressed mood, as outlined in the response styles theory of depression (Nolen-Hoeksema, 1991). Indeed, a wealth of research has established trait rumination's role as a major risk factor for MDD that is predictive of the onset, length, and number of depressive episodes experienced (Wisco & Nolen-Hoeksema, 2008). The current study utilized ecological momentary assessment (EMA) methodology to better understand the role of state-level rumination as a predictor of depression over time.

Rumination may contribute to depression in interaction with the experience of negative life events (NLEs), in line with the well-supported vulnerability-stress model of depression (Abramson et al., 2002; Hammen, 2005). Indeed, in an influential early study, individuals scoring higher in initial levels of trait rumination demonstrated greater increases in depressive symptoms following the experience of a major stressor, the 1989 Lomo Prieta earthquake (Nolen-Hoeksema & Morrow, 1991). This combination of NLE occurrence and rumination in predicting depressive symptoms also has been supported in experimental paradigms, in which individuals who either scored higher on self-report measures of rumination (Feldner, Leen-Feldner, Zvolensky, & Lejuez, 2006) or underwent a rumination induction (Watkins, Moberly, & Moulds, 2008) demonstrated greater increases in depressive symptoms following an in-lab stressor.

An extension of this literature has examined rumination occurring after the experience of NLEs, referred to as stress-reactive rumination (SRR; Robinson & Alloy, 2003). Individuals engaging in SRR make negative inferences about the events that they have experienced and ruminate about these beliefs, such as that the event's occurrence was entirely their fault. Those displaying higher trait levels of SRR, as measured by the Stress-Reactive Rumination Scale (SRRS; Robinson & Alloy, 2003), are more likely to experience major depressive disorder over time, suggesting that SRR serves as an important vulnerability for MDD (Alloy et al., 2000; Robinson & Alloy, 2003).

Given that SRR occurs in response to life stress, it is vital to study its frequency following the experience of actual stressors. A daily diary study found that although SRR did not independently predict depressive symptoms, there was a significant positive interaction between SRR and number of NLEs per day in predicting same-day depressive symptoms, consistent with a vulnerability-stress framework (Genet & Siemer, 2012). Ecological momentary assessment (EMA) designs, in which the occurrence of rumination and stress are measured multiple times per day, have found that momentary rumination mediated the role of life stress in prospectively predicting depressive symptoms (Moberly & Watkins, 2008a; Ruscio et al., 2015). Although neither study found *momentary*, state-level rumination to moderate the role of NLEs in predicting depression, such a relationship was reported between *trait* rumination and daily stress, providing support for response styles theory (Moberly & Watkins, 2008a).

In addition to examining the effect of SRR on depressive symptoms, the degree to which individuals engage in ruminative, self-focused thought irrespective of proximal stressors is also worthy of study. A novel line of research has investigated the relationship

between momentary ruminative self-focus (MRS) during periods of rest, in which individuals are not engaged in a specific task and their minds are able to wander, and their subsequent mood symptoms. It was found that individuals who scored higher on a baseline scale of cognitive reactivity and engaged in more ruminative, internally-focused thought during rest, as measured by the Momentary Ruminative Self-focus Inventory (MRSI; Mor, Marchetti, & Koster, 2013) showed increased depressive symptoms (Marchetti, Koster, & De Raedt, 2013). Furthermore, EMA studies have found that engaging in MRS predicts depressive symptoms both concurrently (Takano & Tanno, 2010) and prospectively (Moberly & Watkins, 2008b) irrespective of the occurrence of proximal stressors. Thus, these findings indicate that momentary rumination is also an important vulnerability factor for depressive symptoms independent of the experience of life stress.

Together, this research emphasizes the need to examine rumination, both in response to life stress and as an independent predictor of depression, as it occurs in real-time using EMA techniques. The use of this methodology is important for several reasons. First, EMA assessment is less likely to fall victim to recall biases in which individuals provide inaccurate retrospective reports of rumination levels or NLEs. Second, these designs capture stress and ruminative processes in a natural setting, versus in structured laboratory paradigms, which may not generalize to real-world experiences. Third, the use of EMA results in many data points, allowing for examination of temporal relationships as well as providing increased confidence in the reliability of the data (Armey, Schatten, Haradhvala, & Miller, 2015; Shiffman, Stone, & Hufford, 2008; Stone et al., 1998; Wenze & Miller, 2010). Furthermore, it has been demonstrated that rumination levels fluctuate over time and are modestly stable at best, suggesting that trait measures may not

accurately capture the phenomenology of this construct (Moberly & Watkins, 2008a; Takano & Tanno, 2011).

Current Study

The current study sought to build on previous findings by utilizing an EMA design to assess the role of both 1) momentary ruminative self-focus (e.g., *Right now, I am thinking about the possible meaning of the way I feel*) and 2) stress-reactive rumination in response to NLE occurrence (e.g., *I'm thinking about how the stressful event is all my fault*) in predicting depressive symptoms over time. Participants responded to text message alerts on their smartphones four times a day for one week, in which they indicated the occurrence of NLEs, their degree of engagement in SRR directly after event occurrence, and their current mood state and level of momentary ruminative self-focus regardless of whether a NLE was reported. The SRR and MRS EMA measures were derived from the validated SRRS and MRSI questionnaires, respectively. Trait measures of rumination and depression also were collected at a baseline session.

It was hypothesized that 1) observation-level increases in both MRS and SRR relative to individuals' mean levels would independently predict increases in depressive symptoms, and 2) significant interactions would emerge between NLE occurrence and both MRS and SRR in predicting increases in depressive symptoms over time. It also was predicted that individuals with higher mean levels of MRS and SRR across the EMA week would display greater increases in depressive symptoms. Finally, in accordance with response styles theory, it was hypothesized that significant interactions would emerge between trait measures of rumination collected at baseline and the experience of NLEs during the EMA week in predicting prospective increases in depressive symptoms.

The present EMA study is the first to examine both momentary ruminative self-focus and stress-reactive rumination within the same design, and is the first to derive its momentary rumination items directly from the validated SRRS and MRSI questionnaires in order to best assess these constructs. Furthermore, previous depressive symptoms serve as controls in all analyses, allowing for truly prospective tests of increases in depressive symptoms. It is important to note that previously conducted EMA and daily diary studies either did not control for depressive symptoms at the previous observation (Genet & Siemer, 2012; Moberly & Watkins, 2008a; Takano & Tanno, 2010) or their effects were no longer significant when doing so (Ruscio et al., 2015), precluding interpretations that momentary rumination predicted relative increases in depressive symptoms over time, and thus, highlighting a necessary area of further study.

The current design allows for a thorough examination of rumination's contribution to temporal changes in depressive symptoms, both independently and in interaction with NLEs, during participants' daily functioning outside of the laboratory. Findings from this research not only will serve to better characterize the nature of ruminative thought patterns, but also will help to identify the ways in which rumination contributes to depressed mood, allowing for more targeted interventions with the ultimate goal of alleviating depressive symptoms.

Method

Participants

One hundred twenty-two students were recruited from the online study pool of the Temple University psychology department. For inclusion, participants were required to be age 18 or older, fluent in English, and cognitively able to complete all study

components. Participants were offered the choice of cash or course credit as compensation for study participation. This study was approved by the Temple University Institutional Review Board and all participants provided informed consent prior to participation. One participant withdrew due to time constraints, resulting in a final sample of 121 participants (M_{age} =21.74 years ± 5.21, range: 18 – 50). The sample was 69.4% female and racially and ethnically diverse (54.5% White, 18.2% Black, 14% Asian, 9.1% Hispanic, and 4.2% Biracial).

Measures

Semi-structured diagnostic interview.

Schedule of Affective Disorders and Schizophrenia, Lifetime (SADS-L, Endicott & Spitzer, 1978). The current study utilized the depression module of the expanded version of the SADS-L semi-structured diagnostic interview (exp-SADS-L; see Alloy et al., 2012). This modified version included additional probes to allow for diagnosis of DSM–IV–TR (American Psychiatric Association, 2000) depressive disorders. The depression module of the exp-SADS-L was conducted by student investigators who received extensive training in interview administration, including assigned readings, training on case vignettes, role-playing, and conducting supervised live interviews. Exp-SADS-L interviews administered by researchers trained using this protocol display high interrater reliability with kappas > .80 (Alloy et al., 2000).

Trait-level questionnaires.

Stress-Reactive Rumination Scale (SRRS; Robinson & Alloy, 2003). The SRRS is a 25-item questionnaire assessing the extent to which individuals direct their focus on negative attributions and inferences, hopeless cognitions, and coping and problem-

solving strategies following the experience of life stress. Participants rate their degree of focus from 0% (*Not focus on this at all*) to 100% (*Focus on this to a great extent*). The current study utilized the 9-item negative attributions and inferences subscale, which has demonstrated good test-retest reliability, internal consistency and validity in predicting major depressive episodes (Robinson & Alloy, 2003). For ease of interpretation, total scores were divided by ten in the current analyses, creating a response range of 0 to 90. The negative attributions and inferences subscale of the SRRS (referred to hereafter as the SRRS) had an $\alpha = .82$ in this sample.

Ruminative Response Scale of the Response Styles Questionnaire (RRS; Nolen-Hoeksema & Morrow, 1991). The RRS contains 22 items assessing a person's tendency to think about the symptoms, causes, and consequences of their depressed mood. A subsequent psychometric analysis of the RRS, intended to better isolate the construct of ruminative thought from overall depressive symptomatology, resulted in the removal of 12 items and the creation of reflective and brooding subscales (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). The current study utilized the 5-item brooding subscale (RRS-B), which measures more passive, repetitive cognitions regarding one's depressed mood and is thought to represent maladaptive rumination. The RRS is internally consistent and moderately correlated with alternative measures of rumination (Nolen-Hoeksema & Morrow, 1991; Siegle, Moore, & Thase, 2004). The RRS-B had an $\alpha = .74$ in this sample.

Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). On a scale of 0 to 3, participants rate the degree to which they experienced symptoms of depression during the past two weeks. The BDI-II is the most widely used self-report measure of

depressive symptoms and has demonstrated excellent internal consistency and validity in undergraduates (Dozois, Dobson, & Ahnberg, 1998; Storch, Roberti, & Roth, 2004). The BDI-II had an $\alpha = .85$ in this sample.

EMA questionnaires.

Stress-Reactive Rumination Scale-State (SRRS-S). The SRRS-S is a modified 5-item version of the SRRS (Robinson & Alloy, 2003) created to measure participants' responses directly after the experience of stress during the EMA week. It includes one item per component of the negative inferences and attributions domain: Negative Attributions/Stable (*Think about how things like this always happen to you*), Negative Attributions/Global (*Think that the cause of the event will lead to additional stressful events in your life*), Negative Attributions/Internal (*Think about how the stressful event is all your fault*), Negative Inferences/Self (*Think about what the occurrence of the event means about you*), and Negative Inferences/Future (*Think about how the negative event will negatively affect your future*). The SRRS rating scale of 0 to 100% was presented as a slider bar on participants' smartphones. For ease of interpretation, total scores were divided by ten in the current analyses, creating a response range of 0 to 50. The SRRS-S had an $\alpha = .81$ and was moderately correlated with baseline SRRS ($r = .49$) and RRS-B ($r = .36$) scores in this sample.

Momentary Ruminative Self-Focus Inventory-Abbreviated (MRSI-A). The MRSI is a 6-item questionnaire measuring state-level fluctuations in ruminative self-focus (Mor et al., 2013). Three items were chosen for use during the EMA week, referred to here as the MRSI-A: 1) *Right now, I am thinking about how happy or sad I feel*, 2) *Right now, I wonder why I react the way I do*, and 3) *Right now, I am thinking about the*

possible meaning of the way I feel. Participants indicate their degree of rumination at the time of the alert using a fixed 7-point Likert scale, ranging from 1 (*Strongly disagree*) to 7 (*Strongly agree*). The MRSI has displayed excellent internal consistency and is moderately correlated with alternative measures of rumination (Mor et al., 2013). The MRSI-A had an $\alpha = .85$ and was moderately correlated with baseline RRS-B ($r = .25$) and SRRS ($r = .31$) scores in this sample.

State Depression Scale (SDS). The SDS is a 3-item scale in which participants rated their level of depressed mood, anhedonia, and irritability on 7-point Likert scales ranging from 1 (*Not at all*) to 7 (*Very much*) at the time of the alert. The SDS is in part modeled after a scale used in a previous EMA study of rumination and depression (Moberly & Watkins, 2008a). The scale was presented as a slider bar on participants' smartphones. The SDS had an $\alpha = .77$ and was strongly correlated with baseline BDI scores ($r = .50$) in this sample.

Procedure

At the Time 1 in-laboratory assessment (T1), participants completed the consenting process, the SADS-L, all trait-level self-report questionnaires (SRRS, RRS, BDI-II), and a briefing on the EMA procedure. Participants completed a sample text message alert sent to their smartphones and reviewed their responses with the researcher to ensure proper EMA functioning and procedure comprehension. Beginning one day following the in-lab assessment, participants received four smartphone alerts per day for seven days during a 12-hour period that they chose based on their anticipated schedule during the EMA week (e.g., 11 AM to 11 PM). Alerts were programmed at random times occurring a minimum of 90 minutes apart within four three-hour periods. Alerts were sent

using the online text message reminder service OhDontForget.com and contained a link to a questionnaire on the customizable survey website FluidSurveys.com. Participants were instructed to respond to each alert within 30 minutes of receiving the text message and were informed that they would receive a cash or credit bonus if they completed at least 80% of EMA questionnaires within this window.

At each EMA alert, participants reported if any NLEs had occurred since the previous alert within the following categories: school, work, social relationships, money, health, other. Participants were able to indicate up to two events per category. For each NLE, participants wrote a brief description of the event and completed the SRRS-S. To prevent participants from neglecting to report NLEs experienced in order to complete alerts faster, participants reporting no NLEs indicated a neutral or positive event that occurred during the time period and completed an alternate version of the SRRS-S modified for neutral/positive events. This modified version was intended primarily to encourage accurate reporting and scores were not examined in the current study. Participants also completed the MRSI-A and SDS at each alert. In sum, at each alert participants reported event occurrence since the last alert and SRR at the time of the *event* (T_E) and also rated their current level of MRS and depressive symptoms at the time of the *alert* (T_A). One week following EMA completion, participants returned to the laboratory for a follow-up session to complete additional questionnaires and receive compensation[1].

[1] This procedure is further described in Chapter 2.

Statistical Analyses

Hierarchical linear modeling analyses were performed using HLM 7 Hierarchical Linear and Nonlinear Modeling (Raudenbush, Bryk, & Congdon, 2010). EMA observations (Level 1) were nested within individuals (Level 2), in order to differentiate between within-person and between-person variance in predicting depressive symptoms over time.

Momentary ruminative self-focus analyses.

The first set of analyses tested whether the occurrence of a NLE interacted with MRS at the time of the alert (T_A MRSI-A) to predict depressive symptoms at the *subsequent* alert (T_{A+1} SDS), controlling for depressive symptoms at the *previous* alert (T_{A-1} SDS), in order to capture change in depressive symptoms over time.[2] Although participants had the option to report multiple NLEs per alert, participants reported only one event at 85% of the alerts (skewness = 1.21). To correct for the non-normality of this distribution, NLE occurrence was dummy coded to indicate either the absence or presence of any NLEs at an alert. The Level 1 model can be described as:

$$SDS_{ti} = \pi_{0i} + \pi_{1i}(NLE_{ti}) + \pi_{2i}(MRSI\text{-}A_{ti}) + \pi_{3i}(NLE \text{ X } MRSI\text{-}A_{ti}) + \pi_{4i}(\text{lagged } SDS_{ti}) + e_{ti}$$

SDS_{ti} refers to depressive symptoms at observation *t* (T_{A+1}) for individual *i*, being predicted by NLE, rumination, and the interaction between these two variables at observation *t* (T_A) for individual *i*, controlling for previous alert depressive symptoms (T_{A-1}). MRSI-A scores were person-mean centered to best capture the effect of

[2] These analyses were not also run predicting depressive symptoms at the concurrent alert (T_A SDS), given that MRSI-A and SDS scores measured rumination and depressive symptoms occurring at the same time, precluding tests of causality.

fluctuations in rumination relative to individuals' mean levels during the EMA week; the interaction term was created by multiplying this mean centered variable by the dummy coded NLE variable. Time between alerts was not a significant predictor of depressive symptoms, and therefore, was not controlled for in the model. The Level 2 model can be described as:

$\pi_{0i} = \beta_{00} + \beta_{01}(\text{Aggregated NLE}_i) + \beta_{02}(\text{Aggregated MRSI-A}_i) + \beta_{03}(\text{T1 BDI}_i) + \beta_{04}(\text{T1 SRRS}_i) + r_{0i}$

Aggregated NLE and MRSI-A variables were included to account for participants' average experience of NLEs and rumination throughout the EMA week. These scores were controlled for at Level 2 to partition within-person variance, referring to fluctuations in NLE and MRSI-A scores from alert to alert within individuals at Level 1, from between-person variance, referring to differences in participants' average number of NLEs and rumination over the EMA week. T1 BDI-II score and SRRS score were controlled for, as they were significant (BDI-II; $B = .13$, $p < .05$) and marginally significant (SRRS; $B = .03$, $p < .10$) predictors of T_{A+1} SDS, controlling for T_{A-1} levels. RRS-B score, age, sex, and history of MDD were not significant predictors and were not controlled for in the model. All Level 2 variables were grand-mean centered. Allowing the slopes of all Level 1 predictors to randomly vary resulted in a significantly improved model fit ($\chi^2(9) = 25.04$, $p < .01$), indicating that the effect of these variables differed between people; therefore these random effects were retained in analyses.

Cross-level analyses were conducted by removing the Level 1 interaction of NLE and MRSI-A, and instead including the interaction term derived from the Level 2 variable of interest (SRRS or RRS-B) and Level 1 NLE score. Each interaction term was tested

separately. The independent effects of all Level 2 variables also were controlled for. Analyses were run both predicting depressive symptoms at the subsequent timepoint (T_{A+1} SDS) and the concurrent timepoint (T_A SDS), controlling for previous symptoms (T_{A-1} SDS).

Stress-reactive rumination analyses.

These analyses tested an interaction between NLEs and SRRS-S score in predicting depressive symptoms over time. The SRRS-S was only completed if a participant indicated the occurrence of an NLE; given that there were no SRRS-S data at alerts for which no NLE was reported, this interaction could not be tested utilizing the above alert-level procedure with a dummy-coded NLE variable. Instead, composite per-day variables were created from all Level 1 measures to allow for variability in number of NLEs experienced, as these scores take into account a maximum of four alert responses per day. As SRRS-S refers to rumination at the time of the event (T_E) and is reported retrospectively, and SDS score refers to depressive symptoms at the time of alert completion (T_A), it was deemed appropriate to predict to SDS on the same day as well as the subsequent day, given temporal difference in these variables. The Level 1 model can be described as:

$$SDS_{ti} = \pi_{0i} + \pi_{1i}(NLE_{ti}) + \pi_{2i}(SRRS\text{-}S_{ti}) + \pi_{3i}(NLE \text{ X } SRRS\text{-}S_{ti}) + \pi_{4i}(\text{lagged } SDS_{ti}) + e_{ti}$$

SDS_{ti} refers to depressive symptoms at observation *t* (T_D when predicting to the average of alerts on the same day, T_{D+1} when predicting to next day), averaged per day for individual *i*, being predicted by total NLEs per day, average SRRS-S per day, and the interaction between these two variables at observation *t* (T_D) for individual *i*, controlling for previous day depressive symptoms (T_{D-1}). NLE and SRRS-S scores were person-

mean centered, and the interaction term was derived from these centered values. Time between measurements was not a significant predictor of depressive symptoms and therefore was not controlled for in the model. The Level 2 model can be described as: $\pi_{0i} = \beta_{00} + \beta_{01}(\text{Aggregated NLE}_i) + \beta_{02}(\text{Aggregated SRRS-S}_i) + \beta_{03}(\text{T1 BDI}_i) + \beta_{04}(\text{T1 SRRS}_i) + r_{0i}$

T1 BDI-II score and SRRS score were controlled for, as they were significant or marginally significant predictors of T_D and T_{D+1} SDS (*Bs* > .03, *p*s < .06), controlling for T_{D-1} levels. RRS-B score, age, sex, and history of MDD were not significant predictors and were not controlled for in the model. All Level 2 variables were grand-mean centered. Allowing the slopes of all Level 1 predictors to randomly vary did not result in a significantly improved model fit ($\chi^2(9) = 12.22$, $p > .05$); therefore, these random effects were not retained in analyses. The conditional effects (simple slopes) of significant interactions were probed using the protocol set forth by Preacher, Curran, and Bauer (2003).

Results

Preliminary Analyses

Participants completed a total of 2,933 EMA alerts[3]. Five percent of alerts were completed more than 30 minutes after receipt and were removed from analyses, resulting in a total of 2,791 alerts. Participants responded to 82% of alerts on time (23.07 alerts ± 3.78), which is comparable to response rates reported in similar EMA studies (Moberly & Watkins, 2008a; Ruscio et al., 2015). Number of alerts completed did not significantly

[3] Sample characteristics and descriptive statistics are also reported in Chapter 2.

differ based on age, sex, history of MDD, BDI, SRRS, or RRS-B score. Participants reported experiencing at least one negative event at 46% of completed alerts, with a maximum of five NLEs being reported at any one alert. When alerts were summed within days, participants reported experiencing at least one negative event on 78% of the days, with a maximum of seven NLEs being reported on any one day. Descriptive statistics for all remaining EMA measures are presented in Table 1.1. Intraclass correlations (ICCs) indicate that about half of the variance in Level 1 measures (range: 43- 52%) existed between individuals, with the remaining variance being within individuals; these values are consistent with previous designs (Genet & Siemer, 2012) and indicate that a hierarchical linear modeling approach is appropriate.

Table 1.1 Descriptive Statistics of EMA Measures

Predictor	*Mean*	*SD*	*ICC*	*Cronbach's alpha*
MRSI-A	9.77	4.38	.52	.85
SRRS-S	21.34	12.45	.45	.81
SDS	6.01	3.96	.43	.77

Note. Analyses include 2,791 alerts. MRSI-A= Momentary Ruminative Self-Focus Inventory-Abbreviated; SRRS-S= Stress-Reactive Rumination Scale-State; SDS = State Depression Scale

There was a significant range in depressive symptoms within the sample as measured by the BDI-II (M = 11.88 ± 8.16). At T1, 18% (n = 22) indicated mild depression (BDI scores: 14-19), 14% (n = 17) displayed moderate depression (BDI scores: 20-28), and 2% (n = 3) reported severe depression (BDI scores: 29-63). Forty-one percent of the sample met criteria for having experienced at least one major depressive episode (MDE) in their life (n = 50) and 17% (n =21) had a history of multiple MDEs.

Five participants were experiencing major depression at the time of study initiation. Rumination scores also varied within the sample (RRS-B $M = 10.48 \pm 3.21$; SRRS $M = 39.83 \pm 15.56$).

Momentary Ruminative Self-Focus Analyses

Experiencing a NLE at a given alert significantly predicted increases in next alert depressive symptoms, controlling for previous alert depressive symptoms ($B = .47$, $p < .01$; Table 1.2); experiencing a greater overall proportion of negative events during the EMA week (aggregated NLE) also significantly predicted increases in depressive symptoms over time ($B = 3.24$, $p < .001$). The effects of both alert-level fluctuations in ruminative self-focus (MRSI-A) relative to an individual's mean, as well as average levels of MRSI-A during the EMA week (aggregated MRSI-A) were not significant in predicting increases in depressive symptoms (Bs < .09, ps > .10).

There was a significant positive interaction between NLEs and MRSI-A in predicting increases in depressive symptoms at the next alert, controlling for previous alert symptoms ($B = .10$, $p < .05$; Table 1.2), such that engaging in high levels of ruminative self-focus relative to one's mean at alerts in which an NLE was reported led to significantly greater increases in depressive symptoms at subsequent alerts compared to alerts at which rumination was high but no NLE was endorsed (conditional effect = .77, $p < .01$; Figure 1.1). When MRSI-A levels were low relative to individuals' means, the presence or absence of a NLE did not significantly influence the prediction of subsequent depressive symptoms (conditional effect = .18, $p > .10$). There also was a significant effect such that, the experience of a NLE coupled with higher than average MRSI-A levels predicted significantly greater increases in depressive symptoms at

subsequent alerts compared to alerts at which a NLE was reported but MRSI-A was low (conditional effect = .08, p < .05). Conversely, differences in relative level of MRSI-A did not significantly affect subsequent depressive symptoms when no NLE was reported (conditional effect = -.02, $p > .10$). This NLE by MRSI-A interaction was significant despite controlling for the independent effect of NLEs at the alert level and at the aggregated individual level, as well as T1 BDI-II and SRRS scores, all of which also significantly predicted change in depressive symptoms over time.

There were no significant cross-level interactions between Level 1 NLEs and either SRRS or RRS-B in predicting concurrent or subsequent depressive symptoms, controlling for previous alert depressive symptoms. As previous EMA research has found rumination to mediate the role of NLEs in predicting depressive symptoms (Moberly & Watkins, 2008a; Ruscio et al., 2015), the direct and indirect paths were tested for significance to determine if a mediational relationship also existed. The direct effect of NLEs predicting next alert depressive symptoms, controlling for previous alert symptoms, was significant ($B = .50$, $p < .01$), as was the effect of NLEs in predicting rumination ($B = .42$, p < .01); however, MRSI-A did not significantly predict depressive symptoms ($B = .02$, $p > .10$), precluding further tests of mediation.

Stress-Reactive Rumination Analyses

Experiencing greater relative levels of NLEs per day predicted significant increases in same day depressive symptoms, controlling for previous day symptoms ($B = .69$, $p < .001$; Table 1.3); experiencing a greater overall number of negative events during the EMA week (aggregated NLEs) also significantly predicted increases in symptoms

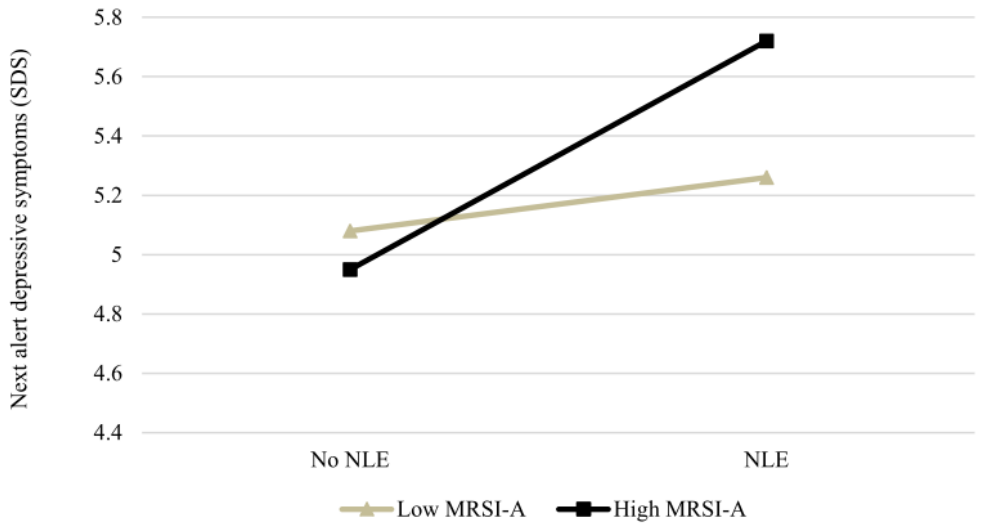
Table 1.2 MRSI-A by NLE Interaction Predicting Next Alert Depressive Symptoms

Predictor	*B*	*SE*
Alert-Level		
Lagged depressive symptoms (T_{A-1} SDS)	.14***	.02
Negative life events (NLE)	.47**	.16
Ruminative self-focus (MRSI-A)[a]	-.02	.03
NLE X MRSI-A	.10*	.05
Individual-Level		
Intercept	5.02***	.21
Aggregated NLE[b]	3.24***	.82
Aggregated MRSI-A[b]	.09	.06
T1 depressive symptoms (BDI-II)[b]	.07**	.02
T1 rumination (SRRS)[b]	.02*	.01

Note. Analyses include 2,530 alerts. SDS= State Depression Scale, NLE = negative life events, MRSI-A = Momentary Ruminative Self-Focus Inventory-Abbreviated, BDI-II = Beck Depression Inventory-II, SRRS = Stress-Reactive Rumination Scale.

* $p < .05$, ** $p < .01$, ***$p < .001$
[a] = person-mean centered [b] = grand-mean centered

Figure 1.1 MRSI-A by NLE Interaction Predicting Next Alert Depressive Symptoms



Note. Analyses include 2,530 alerts. NLE= negative life event; MRSI-A= Momentary Ruminative Self-Focus Inventory-Abbreviated; SDS= State Depression Scale. High and low MRSI values ± 1 SD from individual-level mean. Analyses controlled for previous alert depressive symptoms.

over time ($B = .74$, $p < .01$). The effects of both daily fluctuations in stress-reactive rumination relative to an individual's mean (SRRS-S; $B = .07$, $p < .001$), as well as average levels of SRRS-S during the EMA week (aggregated SRRS-S; $B = .10$, $p < .001$) were significant in predicting increases in daily depressive symptoms.

There was a significant interaction predicting depressive symptoms on the same day controlling for previous day depressive symptoms ($B = .04$, $p < .05$; Table 1.3), such that engaging in high levels of SRR relative to one's mean led to significantly greater increases in depressive symptoms on days when more NLEs were reported (conditional effect = .97, $p < .001$; Figure 1.2). Increases in daily NLEs also predicted greater depressive symptoms even when relative SRR levels were low (conditional effect = .40, $p < .05$). Furthermore, there was a significant effect such that higher daily NLEs coupled with higher than average SRR levels predicted significantly greater increases in depressive symptoms compared to high stress days when SRR levels were low (conditional effect = .10, $p < .001$). Conversely, differences in relative level of SRR did not significantly affect depressive symptoms on low stress days (conditional effect = -.03, $p > .10$). This interaction was significant when controlling for the independent effects of NLEs and SRRS-S at the day level and at the aggregated individual level, as well as T1 BDI-II score, all of which significantly predicted increases in daily depressive symptoms. Direct and indirect paths were tested for significance to determine if a mediational relationship also existed. The direct effect of NLEs predicting same day depressive symptoms, controlling for previous day symptoms, was significant ($B = .82$, $p < .001$), as

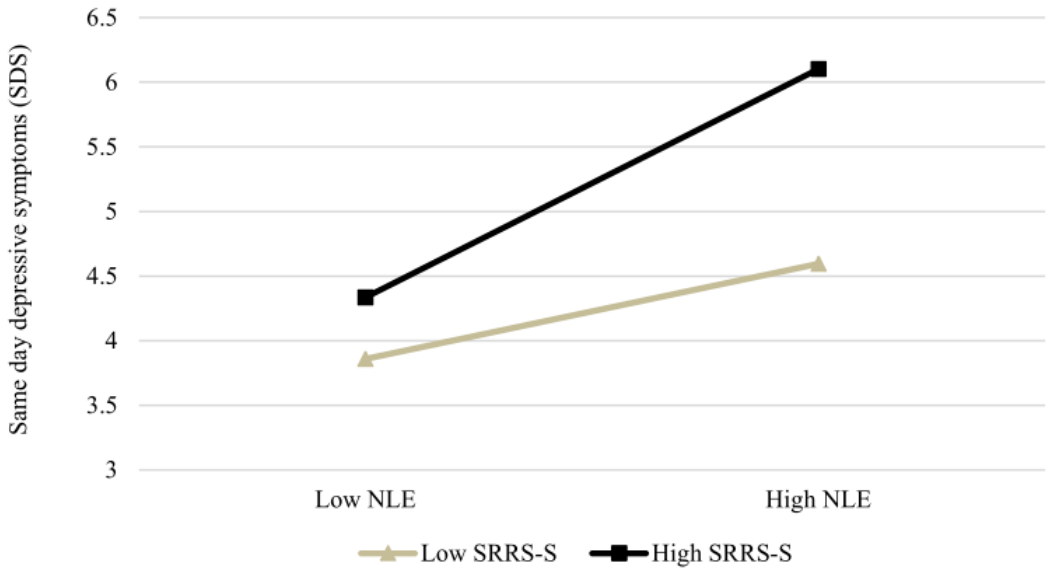
Table 1.3 SRRS-S by NLE Interaction Predicting Same Day Depressive Symptoms

Predictor	*B*	*SE*
Day-Level		
Lagged depressive symptoms (T_{D-1} SDS)	.25***	.04
Negative life events (NLE)[a]	.69***	.12
Rumination (SRRS-S)[a]	.07***	.01
NLE X SRRS-S	.04*	.02
Individual-Level		
Intercept	4.72***	.31
Aggregated NLE[b]	.74**	.24
Aggregated SRRS-S[b]	.10***	.02
T1 depressive symptoms (BDI-II)[b]	.08**	.02
T1 rumination (SRRS)[b]	.00	.01

Note. Analyses included 505 days. SDS= State Depression Scale, NLE = negative life events, SRRS-S = Stress-Reactive Rumination Scale-State, BDI-II = Beck Depression Inventory-II, SRRS = Stress-Reactive Rumination Scale.

* $p < .05$, ** $p < .01$, ***$p < .001$
[a] = person-mean centered [b] = grand-mean centered

Figure 1.2 SRRS-S by NLE Interaction Predicting Same Day Depressive Symptoms



Note. Analyses include 505 days. NLE= negative life events; SRRS-S= Stress-Reactive Rumination Scale-State; SDS= State Depression Scale. High and low values ± 1 SD from individual-level means. Analyses controlled for previous day depressive symptoms.

was the effect of SRR in predicting symptoms (B = .06, p < .001). However, the effect of NLEs in predicting SRR was not significant (B = -.38, p > .10), precluding further tests of mediation. The interaction between NLEs and SRRS-S was not significant in predicting depressive symptoms on the subsequent day controlling for previous day depressive symptoms (B = .01, p > .10).

Discussion

The current study sought to investigate the role of rumination, both independently and in interaction with stress, in predicting relative increases in depressive symptoms utilizing EMA methodology. It was hypothesized that increases in momentary ruminative self-focus (MRS) irrespective of the experience of negative life events (NLEs), as well as stress-reactive rumination (SRR), would exert independent effects in predicting heightened depressive symptoms over time. It was further hypothesized that both MRS and SRR would moderate the effect of NLEs in predicting increases in depressive symptoms. Finally, in line with response styles theory, baseline trait measures of rumination also were expected to interact with the experience of NLEs to increase depressive symptoms over time.

The present findings emphasize an important interplay between rumination and stress in contributing to depression. Contrary to hypotheses, MRS did not independently predict increases in depressive symptoms at the next alert, controlling for previous alert symptoms; however, as expected, a positive effect of MRS did emerge in interaction with the experience of NLEs. In other words, engaging in greater MRS only led to higher subsequent depression scores when it occurred at an EMA alert in which a NLE was reported. Furthermore, when individuals engaged in higher levels of MRS relative to their

means in response to a NLE, they experienced greater prospective increases in depressive symptoms compared to alerts at which they engaged in lower relative levels of MRS in response to stress. These findings suggest that engaging in introspective thought about one's feelings is not inherently detrimental, but that MRS becomes maladaptive when occurring following the recent experience of a NLE.

Current findings with regards to stress-reactive rumination were in line with hypotheses. Engaging in greater SRR, whether relative to one's own mean at the alert level or relative to the grand mean at the individual level, exerted a significant independent effect in predicting increases in daily depressive symptoms, controlling for previous day symptoms. SRR also significantly moderated the effect of NLEs in predicting depressive symptoms. The nature of this interaction was similar to that described with regards to MRS, in that 1) higher levels of SRR relative to an individual's mean predicted greater depressive symptoms on days when participants reported higher than average numbers of NLEs, compared to low stress days, and 2) depressive symptoms were greater on high stress days when participants engaged in more SRR, compared to high stress days during which they did not ruminate as much.

No significant interactions were observed between baseline trait measures of rumination and NLE occurrence during the EMA week in predicting depressive symptoms, in contrast to predictions based on response styles theory (Nolen-Hoeksema, 1991) and previous EMA findings (Moberly & Watkins, 2008a). However, the abovementioned significant interactions between MRSI-A and SRRS-S scores and NLEs certainly could be viewed as consistent with response styles theory. Although these interactions involved state measures of rumination, these variables were moderately

correlated with baseline trait rumination measures (SRRS and RRS-B), suggesting that they may be measuring a separate but closely related facet of ruminative thought. Indeed, such state variables may serve as more accurate measures of individuals' average cognitive responses to stress compared to trait questionnaires.

To our knowledge, the current study is the first to demonstrate significant positive interactions between rumination and life stress in prospectively predicting depressive symptoms utilizing an EMA design. Whereas previous EMA studies have found rumination to mediate the role of NLEs in predicting depression (Moberly & Watkins, 2008a; Ruscio et al., 2015), the current work suggests that instead of serving to *explain* the detrimental effects of NLE occurrence on mood, variability in rumination can indeed *change* the magnitude of stressor effects, by increasing the potency of life stress as a vulnerability factor for depression. Our results complement findings from a daily diary study of SRR, life stress, and depressive symptoms (Genet & Siemer, 2012), and extend this work by utilizing an EMA design in which variables of interest were measured multiple times per day to increase validity.

Furthermore, the current design assessed both momentary ruminative self-focus and stress-reactive rumination, utilizing multiple items from validated measures of these constructs, the MRSI and SRRS, neither of which have yet been examined using EMA methodology. By assessing these subtypes of rumination, the differential effects of MRS and SRR were able to be parsed apart; namely, that whereas SRR independently predicted increases in depressive symptoms, engaging in MRS only increased symptoms when in response to a stressor. Although previous research has found that engaging in MRS while at rest is predictive of increases in depressive symptoms (Marchetti et al.,

2013), it should be noted that this effect only held among individuals scoring high on a trait measure of cognitive reactivity[4]. Instead, the current findings demonstrate that engaging in more MRS, whether relative to one's own mean or relative to the mean of the sample, does not independently lead to increases in depressive symptomatology. Interestingly, although not a significant effect, the lowest levels of depressive symptoms were seen following alerts at which individuals experienced no NLE but engaged in higher than average MRS (Figure 1.1), suggesting that focusing on one's feelings in the absence of a proximal stressor may have beneficial effects on mood. Although such conclusions cannot be drawn from the current findings, the results highlight a potential avenue of future research.

The current findings are strengthened by the study's analytic strategy. Alert-level rumination variables were centered on individual means in order to assess the effect of relative fluctuations in rumination during the EMA week on depressive symptoms. Analyses also controlled for aggregated alert-level variables at the individual level; this control strategy accounted for overall differences in rumination and NLEs during the EMA week, to better examine the role of alert-level variation in the prediction of depressive symptoms within each participant. Furthermore, all analyses controlled for previous depressive symptoms, whether at the previous alert or day, in order to truly assess whether depressive symptoms changed over time due to the occurrence of rumination or NLEs. Past studies either did not control for previous symptoms (Genet &

[4] Follow-up analyses tested whether significant interactions existed between trait-level rumination (SRRS, RRS-B) at baseline and MRSI scores during the EMA week in predicting depressive symptoms; no significant relationships emerged.

Siemer, 2012; Moberly & Watkins, 2008a; Takano & Tanno, 2010) or their effects were no longer significant when doing so (Ruscio et al., 2015), leading the current findings to serve as the first prospective evidence that rumination, in combination with life stress, increases depressive symptoms over time.

The use of EMA methodology lends multiple strengths to the current findings. Collecting data at several timepoints per day helped to lessen recall biases and increase reliability of the variables of interest. Furthermore, the findings were specific to participants' actual experience of daily life stressors and rumination occurring outside of a controlled laboratory setting, and captured the effects of relative fluctuations in these levels over time. By utilizing online questionnaires sent to participants' smartphones that were time stamped upon completion, there was increased confidence that participants were completing EMA alerts within assigned time windows. Furthermore, allowing participants to choose the 12-hour window during which they received alerts maximized the percentage of waking hours assessed and likely benefited overall response rates.

However, limitations also should be noted. Although the current EMA design helped decrease the incidence of recall biases, there still exists the possibility of such occurrence, given that participants were asked to retrospectively report on life stress and their degree of SRR since the last alert. However, because the time interval between alerts was only hours rather than days, weeks, or months, recall biases would be expected to be minor compared to non-EMA studies of rumination, stress, and depression. As explained earlier, SRR analyses were conducted by calculating daily averages to allow for variation in life event occurrence, which decreased the number of available data points. In addition, the interaction of SRR and NLEs was only significant predicting same

day, and not next day, depressive symptoms. Although SRR ratings referred to rumination at the time of event occurrence and depressive symptoms referred to mood at time of alert completion, indicating a temporal difference in the variables of interest, our results would have been further strengthened if the interaction term predicted next day symptoms. Finally, despite the wide range of depression scores reported among the current participants, the sample was nonclinical in nature. Assessing such relationships within a clinically depressed sample is an important and much-needed extension of the current work.

The results from this study may have important clinical implications. Specifically, findings of significant interactions between rumination and life stress suggest that ruminative thought is not merely a by-product of experiencing NLEs, but is instead a response style that exhibits variation both within and between individuals, such that engaging in relatively higher levels leads to greater increases in depression. Therefore, it would be important to address individuals' tendency to engage in rumination directly after the experience of stress, such that decreasing relative levels could potentially reduce the deleterious effects of NLEs on mood. Indeed, trainings intended to decrease levels of abstract, repetitive thought regarding negative information have demonstrated promising preliminary results (Joormann, Hertel, LeMoult, & Gotlib, 2009; Watkins, Baeyens, & Read, 2009). Such designs could benefit from the integration of EMA methodology; for instance, depressed individuals could engage in trainings intended to reduce rumination directly after the experience of stress, after which EMA techniques could monitor relative changes in rumination and depressive symptoms following NLE occurrence during daily functioning. Indeed, EMA is increasingly being employed to track longer-term outcomes

following active interventions (Hoorelbeke, Koster, Demeyer, Loeys, & Vanderhasselt, in press).

The current research sheds light on the role of rumination in response to daily life stressors as a contributor to depressed mood over time. The findings of significant interactions between MRS, SRR and NLEs suggest that the degree of rumination engaged in following the experience of stress is variable and can lead to greater increases in depressive symptoms when occurring at higher levels relative to individual means. These results have significant clinical implications in that they emphasize the importance of targeting individuals' momentary response styles to stress in order to help decrease depressive symptoms over time. Future research should continue to capitalize on the many benefits of EMA methodology in order to better understand this relationship and lessen the substantial burden of major depressive disorder.

CHAPTER 2

MANUSCRIPT 2 IN JOURNAL ARTICLE FORM

Negative Memory Bias as a Vulnerability for Depression: Relationship between Momentary Stress-Reactive Rumination and Event Recall

Abstract

The current research utilized EMA methodology to test the hypotheses that 1) engaging in greater rumination following the experience of stress (stress-reactive rumination; SRR) relative to an individual's mean would lead to deeper encoding and improved retrieval of the stressor, and 2) this biased memory for negative autobiographical information would predict increases in depressive symptoms over time. A nonclinical sample of 121 participants received four smartphone alerts per day for one week in which they reported on their experience of negative life events (NLEs) as well as their degree of SRR and depressed mood after event occurrence. Participants' memory for NLEs was tested at a two-week follow-up session and depressive symptoms were measured again two weeks later. NLEs followed by increased SRR relative to individuals' means were significantly more likely to be recalled two weeks later. Neither SRR nor proportional recall of NLEs was a significant independent predictor of increased depressive symptoms over time. However, a significant interaction emerged between the number of NLEs experienced and proportional recall of those events, such that individuals who endorsed and recalled greater numbers of stressors during the EMA week displayed increased depressive symptoms at follow-up. These findings support the role of rumination and memory biases as vulnerability factors for depression, and suggest potential clinical benefits of modifying ruminative response styles to daily stressors.

Keywords: rumination, memory, depression, ecological momentary assessment, cognitive biases

Negative Memory Bias as a Vulnerability for Depression: Relationship between Momentary Stress-Reactive Rumination and Event Recall

A wealth of research has sought to identify vulnerability factors for major depressive disorder (MDD), given the substantial burdens imposed by this common and debilitating condition whose causes are not yet well understood (Kessler, Merikangas, & Wang, 2007). Rumination, the act of perseverative, passive thought regarding one's down mood, has long been demonstrated to contribute to the development and maintenance of depression (Nolen-Hoeksema, 1991; see Wisco & Nolen-Hoeksema, 2008 for a review). Indeed, rumination has been found to predict the onset, duration, and number of depressive episodes experienced (Just & Alloy, 1997; Roberts, Gilboa, & Gotlib, 1998; Spasojević & Alloy, 2002).

Despite the substantial body of research identifying rumination as a vulnerability for depression, considerably less work has aimed to understand the specific mechanisms through which rumination exerts its deleterious effects on mood. It has been hypothesized that rumination may contribute to depressed affect by increasing the availability of negative thoughts and memories (Wisco & Nolen-Hoeksema, 2008). Negative memory biases are a well-established correlate of major depression (see Matt, Vázquez, & Campbell, 1992, for a review) that also have been associated with depressive symptoms in nonclinical samples (Connolly, Abramson, & Alloy, 2016), including individuals at risk for MDD who had not yet developed the disorder (Alloy, Abramson, Murray, Whitehouse, & Hogan, 1997). Such findings support negative memory biases' potential role as an important vulnerability factor for depression.

Joormann and colleagues have proposed an integrated theory of the relations among rumination, memory, and depression (Joormann, Yoon, & Zetsche, 2007; Joormann, 2010). They posit that poor inhibitory control is associated with increased negative information entering and remaining in working memory, leading to heightened levels of elaboration and rehearsal. This repetitive, negative processing can be conceptualized as rumination, a cognitive style that Joormann hypothesizes will lead to deeper encoding of negative stimuli. Maintaining increased amounts of negative information in long-term memory and ruminating about this information would therefore fuel negative memory biases and perpetuate depressed mood over time.

Several studies have investigated whether rumination may lead to negative information being more strongly encoded and better retrieved. Work by Moulds, Kandris, and Williams (2007) and Hertel and El-Messidi (2006) demonstrated that dysphoric participants randomized to a rumination induction prior to a memorization task subsequently recalled more personally-interpreted words compared to those randomized to a distraction condition. The findings suggest that undergoing rumination induction may cause participants to dwell on negative self-referent stimuli, leading to deeper encoding and improved retrieval compared to participants encouraged to distract. Individuals scoring high in rumination also have been found to remember more emotional words they had been instructed to forget compared to low ruminators, controlling for depression (Hertle & Gerstle, 2003; Joormann & Tran, 2009). These results support the hypothesis that rumination is linked to difficulties inhibiting the processing of emotional information, thereby improving memory for these stimuli.

Rumination also has been associated with heightened recall of negative autobiographical information. An early set of studies provided evidence that individuals engaging in more self-focused, ruminative thought, whether measured at the trait level or increased through an in-lab induction, were more likely to generate negative events when asked to freely recall autobiographical memories (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; McFarland and Buehler, 1998; Pyszczynski, Hamilton, & Herring, 1989). This relationship was demonstrated both in dysphoric samples (Lyubomirsky et al., 1998; Pyszczynski et al., 1989), as well as after inducing negative mood in a community sample (McFarland & Buehler, 1998).

Together, this research provides important preliminary support for the relationship between rumination and memory biases as factors influencing depressed mood. These findings invite further inquiry in order to address methodological weaknesses in the abovementioned studies. Importantly, longitudinal approaches were not used to test whether these risk factors were predictive of future depressive symptoms or diagnoses. Indeed, several of the studies only found rumination to foster memory biases among already depressed or dysphoric samples. Therefore, it is yet unclear whether these observed effects are present only when individuals are already in a dysphoric state, or whether the link between rumination and memory biases may serve as a risk factor for increases in depressive symptoms and episodes over time. In addition, none of the reviewed studies explicitly assessed the cognitive strategies used in the encoding of negative information, which is necessary in order to best understand rumination's contribution to improved recall of those stimuli. Furthermore, in several studies, memory was assessed by measuring recall of preset lists of negative words. Although such designs

allow for the standardization of experimental stimuli, the personal relevance of these words likely varied between participants, and the memorization of a word list is not directly comparable to the encoding of negative experiences encountered during an individual's daily life. In contrast, the reviewed studies of autobiographical memory retrieval sacrificed standardization by allowing participants to freely recall any memories from their pasts. Although this may represent a more ecologically valid measure of recall bias, these studies are restricted by their inability to analyze processes occurring at the encoding stage, as the recalled memories reference content experienced perhaps many years prior outside of the laboratory.

In order to better understand this purported relationship between rumination and memory for negative self-referent information, it is crucial to examine the degree of rumination individuals engage in directly following the experience of stress, referred to as stress-reactive rumination (SRR; Robinson & Alloy, 2003). Individuals engaging in SRR make negative inferences about experienced stressors and dwell on these beliefs, such as thinking that an event was entirely their fault. Individuals scoring higher in trait SRR were more likely to experience depressive episodes over time (Robinson & Alloy, 2003), and SRR interacted with the experience of stress to prospectively predict depressive symptoms in a daily diary study (Genet & Siemer, 2012), highlighting its role as a significant vulnerability factor for MDD.

No study to our knowledge has yet examined the relationship between SRR and subsequent memory for negative information. However, research within the social anxiety literature on post-event processing, a behavior with striking parallels to the

construct of depressive rumination with regards to its repetitive, self-focused, and maladaptive qualities (Brozovich & Heimberg, 2008; McEvoy, Mahoney, & Moulds, 2010), provides important preliminary support. Increased post-event processing following a stressful in-lab task has been linked to improved recall of negative details of the event at follow-up (Cody & Teachman, 2010; Mellings & Alden, 2000). These findings suggest that engaging in repetitive, negative thought directly following the experience of stress improves memory for that information. Such designs represent a compromise between standardizing the experience of a negative life event while also providing a realistic stressor that may better illustrate individuals' general coping styles and subsequent memory biases. However, it must be noted that these situations (e.g., receiving negative feedback from a panel of judges after giving a speech) are still constructed within a laboratory setting and likely demonstrate large variation in their degree of relevance and stressfulness between participants, lessening their ecological validity.

This review highlights the need for prospective research examining the role of rumination in the encoding and subsequent recall of negative experiences occurring in individuals' daily lives, in order to better understand the potential link between these vulnerability factors in predicting increases in depressive symptoms. Exploring this relationship via ecological momentary assessment (EMA), in which participants provide multiple daily measurements of stress outside of a controlled laboratory setting, provides several advantages. Its frequent assessment schedule helps to eliminate retrospective recall biases in reporting, and its repeated measurement of study variables improves data reliability. As such, the rich and nuanced data collected through EMA designs allows for refined statistical analyses assessing the effect of fluctuations in target variables within

individuals (Shiffman, Stone, & Hufford, 2008; Stone et al., 1998). Furthermore, EMA assessment allows for the analysis of participants' experience of, and subsequent cognitive response to, actual negative life events occurring during their daily functioning outside of the laboratory, substantially increasing the validity of the data.

Current Study

The current study employed an EMA design to measure rumination in response to stressors reported in real time during participants' everyday lives. After completing baseline measures of depression, rumination, and neutral memory ability, participants received four smartphone alerts per day for one week in which they reported on their experience of negative life events (NLEs) as well as their degree of SRR and depressed mood following the occurrence of these stressors. Participants' memory for neutral words and NLEs was then tested at a two-week follow-up session, and depressive symptoms were measured again two weeks later.

It was hypothesized that events followed by greater amounts of SRR relative to an individual's mean would be more likely to be recalled at follow-up, as these events would be encoded more strongly after their occurrence and therefore better retrieved. This effect was hypothesized to persist when controlling for individuals' long-term memory for neutral stimuli. It also was predicted that SRR would lead to increases in depressive symptoms over time, and that this relationship would be mediated by recall of NLEs. Finally, it was hypothesized that an interaction would emerge between number of NLEs reported during the EMA week and recall of those events in predicting depression at follow-up, such that the effects of negative recall biases would be amplified by the

experience of stress. This hypothesis was in accordance with vulnerability-stress models of depression, positing that cognitive vulnerabilities for depression are more strongly expressed when coupled with the occurrence of heightened stress (Abramson et al., 2002).

The present study is the first to our knowledge to assess the role of rumination directly after the occurrence of naturalistic stress in influencing memory and depressive symptoms over time. This longitudinal EMA design serves as a refined test of the hypothesis that engaging in repetitive, ruminative thought after stress contributes to deeper encoding and improved retrieval of this negative information. The current research aims to better understand the specific mechanisms through which rumination contributes to depressed mood in order to ultimately inform treatment and prevention efforts. It is possible that targeting individuals' tendencies to engage in stress-reactive rumination directly after the experience of negative life events could help to reduce negative memory biases and therefore alleviate depressive symptoms and episode occurrence over time.

Method

Participants

One hundred twenty-two students were recruited from the Temple University psychology department online study pool. Participants were required to be age 18 or older, fluent in English, and cognitively able to complete all study components for inclusion. Cash or course credit was offered as compensation for study participation. Participants provided informed consent and all components of the research were

approved by the Temple University Institutional Review Board. One participant withdrew due to time constraints, resulting in a final sample of 121 participants (M_{age} = 21.74 years ± 5.21, range: 18 – 50). The current sample was 69.4% female and racially and ethnically diverse (54.5% White, 18.2% Black, 14% Asian, 9.1% Hispanic, and 4.2% Biracial).

Measures

Schedule of Affective Disorders and Schizophrenia, Lifetime (SADS-L, Endicott & Spitzer, 1978).

The current study utilized the depression module of the expanded version of the SADS-L semi-structured diagnostic interview (exp-SADS-L; see Alloy et al., 2012). This modified version included additional probes to allow for assessment of lifetime history of DSM–IV–TR (American Psychiatric Association, 2000) depressive disorders. The depression module of the exp-SADS-L was administered by student investigators who received extensive training in SADS administration, including assigned readings, training on case vignettes, role-playing, and conducting supervised live interviews. SADS-L interviews conducted by investigators trained using this protocol display high interrater reliability with kappas >.80 (Alloy et al., 2000).

California Verbal Learning Test (CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000).

The CVLT-II is an interviewer-administered verbal memory test in which the participant is asked to recall a list of 16 words after hearing them listed by the interviewer (List A); this process is repeated five times using the same list. They are then asked to recall a second list of 16 words (List B; distractor list) after which they must recall the

initial list again. Finally, following a 20 minute delay during which the participant is engaged in other tasks, they are asked to recall List A again. The CVLT-II was slightly modified in this study, in that participants were asked to recall List A again after a two-week delay when they returned to the lab. Similar modifications of the CVLT have been employed in studies of long-term memory (Walhovd et al., 2006). The current study utilized the total number of List A words recalled at two-week follow-up, not including repetitions, as a control measure of long-term memory for neutral stimuli.

Life Events Scale (LES) and Interview (LEI; Alloy & Clements, 1992; Safford, Alloy, Abramson, & Crossfield, 2007).

The LES includes 194 major and minor life events in a variety of domains relevant to young adults (e.g., school, work, romantic relationships), and assigns each event an a priori objective intensity rating ranging from 1 (*Mild*) to 3 (*Major*). The current version was abbreviated to only include the 135 LES events rated as negative. At the two-week follow-up session, individuals indicated which events they experienced during the weeklong EMA period. The validity of these event classifications was then evaluated through an experimenter-administered interview (LEI) to reduce subjective report biases. Standardized event criteria and probes are provided to the interviewer to help him/her determine event eligibility and confirm objective intensity ratings. The LES has shown excellent reliability and validity (Alloy & Clements, 1992; Safford et al., 2007).

Trait-level questionnaires.

Stress-Reactive Rumination Scale (SRRS; Robinson & Alloy, 2003). The SRRS is a 25-item questionnaire assessing the extent to which individuals direct their focus on

negative attributions and inferences, hopeless cognitions, and coping and problem-solving strategies following the experience of life stress. Participants rate their degree of focus from 0% (*Not focus on this at all*) to 100% (*Focus on this to a great extent*). The current study utilized the 9-item negative attributions and inferences subscale, which has demonstrated good test-retest reliability, internal consistency and validity in predicting major depressive episodes (Robinson & Alloy, 2003). For ease of interpretation, total scores were divided by ten in the current analyses, creating a response range of 0 to 90. The negative attributions and inferences subscale of the SRRS (referred to hereafter as the SRRS) had an $\alpha = .82$ in the current sample.

Ruminative Response Scale of the Response Styles Questionnaire (RRS; Nolen-Hoeksema & Morrow, 1991). The RRS contains 22 items assessing a person's tendency to think about the symptoms, causes, and consequences of their depressed mood. A subsequent psychometric analysis of the RRS, intended to better isolate the construct of ruminative thought from overall depressive symptomatology, resulted in the removal of 12 items and the creation of reflective and brooding subscales (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). The current study utilized the 5-item brooding subscale (RRS-B), which measures more passive, repetitive cognitions regarding one's depressed mood and is thought to represent maladaptive rumination. The RRS is internally consistent and moderately correlated with alternative measures of rumination (Nolen-Hoeksema & Morrow, 1991; Siegle, Moore, & Thase, 2004). The RRS-B had an $\alpha = .74$ in this sample.

Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). On a scale of 0 to 3, participants rate the degree to which they experienced a given symptom of

depression during the past two weeks. It is the most widely used self-report measure of depressive symptoms and has demonstrated excellent internal consistency and validity in undergraduates (Dozois, Dobson, & Ahnberg, 1998; Storch, Roberti, & Roth, 2004). The BDI-II had an $\alpha = .85$ in this sample.

EMA questionnaires.

Stress-Reactive Rumination Scale-State (SRRS-S). The SRRS-S is a modified 5-item version of the SRRS (Robinson & Alloy, 2003) created to measure participants' cognitive responses directly after the experience of stress during the EMA week. It includes one item per component of the negative inferences and attributions domain: Negative Attributions/Stable (*Think about how things like this always happen to you*), Negative Attributions/Global (*Think that the cause of the event will lead to additional stressful events in your life*), Negative Attributions/Internal (*Think about how the stressful event is all your fault*), Negative Inferences/Self (*Think about what the occurrence of the event means about you*), and Negative Inferences/Future (*Think about how the negative event will negatively affect your future*). The SRRS rating scale of 0 to 100% was presented as a slider bar on participants' smartphones. For ease of interpretation, total scores were divided by ten in the current analyses, creating a response range of 0 to 50. The SRRS-S had an $\alpha = .81$ in this sample and was moderately correlated with baseline SRRS ($r = .49$) and RRS-B scores ($r = .36$).

State Depression Scale (SDS). The SDS is a 3-item scale in which participants rated their level of depressed mood, anhedonia, and irritability on 7-point Likert scales ranging from 1 (*Not at all*) to 7 (*Very much*) at the time of the alert. The SDS is in part modeled after a scale used in a previous EMA study of rumination and depression

(Moberly & Watkins, 2008). The scale was presented as a slider bar on participants' smartphones. The SDS had an $\alpha = .77$ in this sample and was strongly correlated with baseline BDI score ($r = .50$).

Procedure

At the Time 1 (T1) in-laboratory assessment, participants completed the consenting process, SADS-L, CVLT-II, self-report questionnaires (SRRS, RRS, BDI-II), and a briefing on EMA procedure. Participants completed a sample text message alert sent to their smartphones and reviewed their responses with the researcher to ensure proper EMA functioning and procedure comprehension. Beginning one day after T1, participants received four smartphone alerts per day for seven days during a 12-hour period that they chose based on their anticipated schedule during the EMA week (e.g., 11 AM to 11 PM). Alerts were programmed at random times occurring a minimum of 90 minutes apart within four three-hour periods. Alerts were sent using the online text message reminder service OhDontForget.com and contained a link to a questionnaire on the customizable survey website FluidSurveys.com. Participants were instructed to complete each alert within 30 minutes of receiving the text message, and were informed that they would receive a cash or credit bonus if they completed at least 80% of EMA alerts within this window.

At each EMA alert, participants reported if any negative life events (NLEs) had occurred to them since the previous alert within the following categories: school, work, social relationships, money, health, other. Participants were able to indicate up to two events per category. For each NLE, participants wrote a brief description of the event and completed the SRRS-S. Participants also had the option to indicate the continuation of an

event reported at a previous alert; these events were labeled as chronic and all associated ratings were averaged into one composite score per event. Participants rated the subjective intensity of the event on a scale of 1 (*mild*) to 4 (*extreme*). To prevent participants from neglecting to report NLEs experienced in order to complete alerts faster, participants reporting no NLEs within the abovementioned categories were prompted to describe a neutral or positive event and completed an alternate version of the SRRS-S modified for neutral/positive events. This modified version was intended primarily to encourage accurate reporting and was not analyzed in the current study. Participants completed the SDS at each alert.

Two weeks after initiating the EMA procedure, participants returned to the laboratory for a follow-up session (T2). They were first asked to recall the list of 16 words from the CVLT-II administered at Day 1. They then completed a surprise free recall task in which they wrote down all negative life events they could remember having occurred during the EMA week on a blank seven-day calendar corresponding to the seven days of the EMA week. Following free recall, participants were provided with the LES and wrote down any additional events they remembered after reading through the event categories (e.g., *Significant fight or argument with boyfriend, girlfriend, or spouse*); this component served as a cued recall condition. The interviewer then completed the LEI, asking follow-up probes for all negative events reported during the EMA week to place each event within an LES category and confirm its objective intensity rating. This process served to standardize and categorize NLEs reported during the EMA week. The interviewer then confirmed which events recalled by the participant during the free recall and cued recall conditions corresponded with events reported during the EMA week. This

interview verified the accuracy of participant's recall during the free and cued recall conditions. Finally, participants completed the BDI-II and received 2/3 of their compensation. Two weeks after T2, participants completed the BDI-II remotely through the website FluidSurveys.com (T3). Following successful completion of T3, participants received the final 1/3 of their compensation and were debriefed.

Statistical Analyses

Prediction of event recall.

Hierarchical linear modeling analyses were performed using HLM 7 Hierarchical Linear and Nonlinear Modeling (Raudenbush, Bryk, & Congdon, 2010). NLEs (Level 1) were nested within individuals (Level 2), in order to differentiate between within-person and between-person variance in the prediction of event recall. Given that the outcome variable was dichotomous (whether or not an event was recalled), a Bernoulli model was used, employing full maximum likelihood via Laplace estimation.

The primary analyses tested whether the degree of SRR engaged in directly after event occurrence was predictive of NLE recall at T2. The Level 1 model can be described as:

$$\text{Prob}(\text{Recall}_{ti}=1|\pi_i) = \phi_{ti}$$
$$\log[\phi_{ti}/(1 - \phi_{ti})] = \eta_{ti}$$
$$\eta_{ti} = \pi_{0i} + \pi_{1i}(\text{SRRS-S}_{ti}) + \pi_{2i}(\text{Objective Intensity}_{ti}) + \pi_{3i}(\text{Subjective Intensity}_{ti}) + \pi_{4i}(\text{Chronicity}_{ti})$$

Recall_{ti} refers to whether or not NLE *t* was recalled, collapsed across the cued and uncued recall conditions, for individual *i*. Degree of SRR engaged in after experiencing that event, the objective and subjective intensity rating of that event *t* for individual *i*, and event classification as chronic or singular served as predictor variables. Objective intensity, subjective intensity, and chronicity were controlled for based on established

links between increased event severity and improved recall (Christianson & Loftus, 1987; Rubin & Kozin, 1984). Variables were person-mean centered to best capture the effect of deviations from individuals' mean levels during the EMA week, with the exception of the dichotomous chronicity variable. Depressed mood at time of event reporting during the EMA week (SDS score) was not a significant predictor of event recall, and therefore, was not controlled for in the model. The Level 2 model can be described as:

$$\pi_{0i} = \beta_{00} + \beta_{01}(\text{Aggregated SRRS-S}_i) + \beta_{02}(\text{Aggregated Objective Intensity}_i) + \beta_{03}(\text{Aggregated Subjective Intensity}_i) + \beta_{04}(\text{Aggregated Chronicity}_i) + r_{0i}$$

Aggregated SRRS-S, objective intensity, subjective intensity, and chronicity variables were included to account for average levels of these variables across all events endorsed for each individual *i*. These scores were controlled for at Level 2 to partition between-person variance, referring to differences in participants' average levels over the EMA week, from within-person variance, referring to fluctuations in scores from event to event within individuals at Level 1. Age, sex, history of MDD, CVLT recall at T2, T1 BDI-II, RRS-B, and SRRS scores, and T2 BDI-II were not significant predictors of event recall and were not controlled for in the model. All Level 2 variables were grand-mean centered. Allowing the slopes of the Level 1 predictors to randomly vary did not result in a significantly improved model fit ($\chi^2(9) = 3.67$, $p > .05$); therefore, these random effects were not retained in analyses. In order to assess the effect of event-level SRR over and above control variables, analyses were first run excluding SRR from the model.

Prospective prediction of depressive symptoms.

The presence of a hypothesized mediational relationship was assessed by testing for significant paths between 1) aggregated SRRS-S score and T3 depressive symptoms, 2) aggregated SRRS-S score and proportional recall of NLEs, and 3) proportional recall

of NLEs and T3 depressive symptoms. The proportional recall variable was calculated by dividing the total number of recalled NLEs by the total number of NLEs endorsed during the EMA week. In addition, the PROCESS macro (Hayes, 2013) was used to probe the interaction between number of NLEs endorsed and proportional recall. Analyses controlled for T2 depressive symptoms when predicting to T3 symptoms. The interaction term was group-mean centered and main effects of NLEs and recall were controlled for in the model. Age, sex, and time between T2 and T3 were not significant predictors of change in depressive symptoms, and thus, were not controlled for in the model.

Results

Preliminary Analyses

Participants responded to a total of 2,933 EMA alerts[5]. Five percent of alerts were completed more than 30 minutes after receipt and were removed from analyses, resulting in 2,791 alerts. Participants responded to 82% of alerts on time (23.07 alerts ± 3.78), which is comparable to response rates reported in similar EMA studies (Moberly & Watkins, 2008; Ruscio et al., 2015). Number of alerts completed did not significantly differ based on age, sex, history of MDD, BDI-II, SRRS, or RRS-B score. All 121 participants who completed the EMA week returned for their two-week follow-up session (T2; $M_{\text{days after T1}}$ = 14.41 ± 1.13), and 120 participants completed their final two-week follow-up online questionnaires (T3; $M_{\text{days after T2}}$ = 16.03 ± 5.25).

Participants reported experiencing at least one negative event at 46% of completed alerts, with a maximum of five NLEs being reported at any one alert. The current analyses included 1,144 NLEs. Participants endorsed an average of 9.45 ± 5.51

[5] Sample characteristics and descriptive statistics are also reported in Chapter 1.

NLEs during the EMA week, with two participants reporting no negative events (Range: 0-25). The majority of events were low in objective intensity, which is consistent with previous EMA studies of life stress (Ruscio et al., 2015); 83.1% of events were mild, 15.8% were moderate, and 1% were major events. Participants' subjective ratings of the intensity of their experienced events were significantly higher; 31.6% were reported as mild, 41.2% as moderate, 20.4% as major, and 6.8% as extreme. Of the events reported, 13.4% were coded as chronic, indicating that they were reported at more than one EMA alert. Of all NLEs, 43.5% were recalled at T2 (35.1% in uncued condition; 8.4% in cued condition). Participants recalled an average of 48.5% ± 20.3% of their endorsed NLEs.

There was a significant range in depressive symptoms within the sample as measured by the BDI-II across the study (M_{T1} = 11.88 ± 8.16; M_{T2} = 10.32 ± 8.20; M_{T3} = 9.15 ± 8.61; Range: 0-50). Forty-one percent of the sample met criteria for having experienced at least one major depressive episode in their lifetime (n = 50) and 17% (n = 21) had a history of multiple MDEs. Five participants were experiencing major depression at the time of study initiation. Rumination scores also varied within the sample with regards to baseline scores on the RRS-B (M = 10.48 ± 3.21) and SRRS (M = 39.83 ± 15.56). Participants recalled an average of 6.28 ± 3.17 CVLT-II words at T2.

Individuals with higher T1 BDI-II scores endorsed significantly more negative events during the EMA week (r = .38, p < .001); no associations emerged between negative event endorsement and T1 SRRS, RRS-B, or history of MDD. Although T1 BDI-II, SRRS, and RRS-B scores all were positively correlated with reporting higher *subjective* intensity ratings of negative events (rs > .26, ps < .01), only T1 BDI-II was

associated with experiencing more *objectively* intense events (r = .20, p < .05). Objective and subjective intensity ratings did not vary based on history of MDD.

Individuals with higher T1 BDI-II, SRRS, and RRS-B scores reported engaging in more SRR (SRRS-S; rs > .25, ps < .01) and experiencing increased depressive symptoms (SDS; rs > .26, ps < .01) following NLE occurrence during the EMA week; scores did not differ based on history of MDD. The EMA measures of SRR (SRRS-S; M = 21.34 ± 12.45) and depressive symptoms (SDS; M = 6.01 ± 3.96) displayed significant variability both within and between individuals, as measured by their intraclass correlations (SRRS-S ICC = .45; SDS ICC = .43), indicating that the use of HLM analyses was appropriate.

Prediction of Event Recall

Before including SRR in the model, event recall was significantly predicted by the objective and subjective intensity of the event as well as its chronicity (Bs > .24, ps < .05; Table 2.1), such that events that were rated as chronic or were higher in objective or subjective intensity relative to an individual's mean were more likely to be recalled at follow-up. Including SRRS-S score in the model significantly improved the model fit, $\chi^2(2) = 9.41$, $p < .01$. The degree of SRR engaged in directly after NLE occurrence significantly increased the odds that the event would be recalled at two-week follow-up (B = .02, OR = 1.02, p < .05; Table 2.1), such that a ten point increase in SRR score improved the odds of event recall by 20%. Event subjective intensity lost significance as a predictor of recall when SRR was added to the model; objective intensity and chronicity remained significant predictors. Follow-up analyses revealed no significant interactions

Table 2.1 Stress-reactive Rumination Predicts Negative Life Event Recall at Follow-up

	Without SRRS-S			*With SRRS-S*		
Predictor	*B*	*OR*	*95% CI*	*B*	*OR*	*95% CI*
Event-Level						
Objective intensity[a]	.96***	2.62	(1.86, 3.71)	.98***	2.67	(1.87, 3.81)
Subjective intensity [a]	.24*	1.27	(1.05, 1.53)	.12	1.13	(.91, 1.40)
Chronicity	.82***	2.28	(1.52, 3.43)	.83***	2.29	(1.51, 3.47)
Stress-reactive rumination (SRRS-S)[a]	---	---	---	.02*	1.02	(1.00, 1.04)
Individual-Level						
Intercept	-.37***	.69	(.60, .80)	-.37***	.69	(.60, .80)
Aggregated objective intensity[b]	.52	1.68	(.74, 3.81)	.37	1.45	(.63, 3.32)
Aggregated subjective intensity[b]	-.14	.87	(.63, 1.19)	.06	1.06	(.69, 1.64)
Aggregated chronicity[b]	.23	1.25	(.43, 3.63)	.11	1.12	(.37, 3.41)
Aggregated SRRS-S[b]	---	---	---	-.02	.98	(.96, 1.00)

Note. Analyses included 1,143 events. Recall outcome variable collapsed across cued and uncued recall conditions. SRRS-S = Stress-Reactive Rumination Scale-State.

* $p < .05$, ***$p < .001$

[a] = person-mean centered [b] = grand-mean centered

between SRRS-S score and either objective intensity, subjective intensity, or chronicity in predicting event recall at T2. The overall proportion of negative events recalled at T2 did not significantly differ based on T1 BDI-II, RRS-B, or SRRS score, T2 BDI-II, history of MDD, or number of CVLT-II words recalled.

Prospective Prediction of Depressive Symptoms

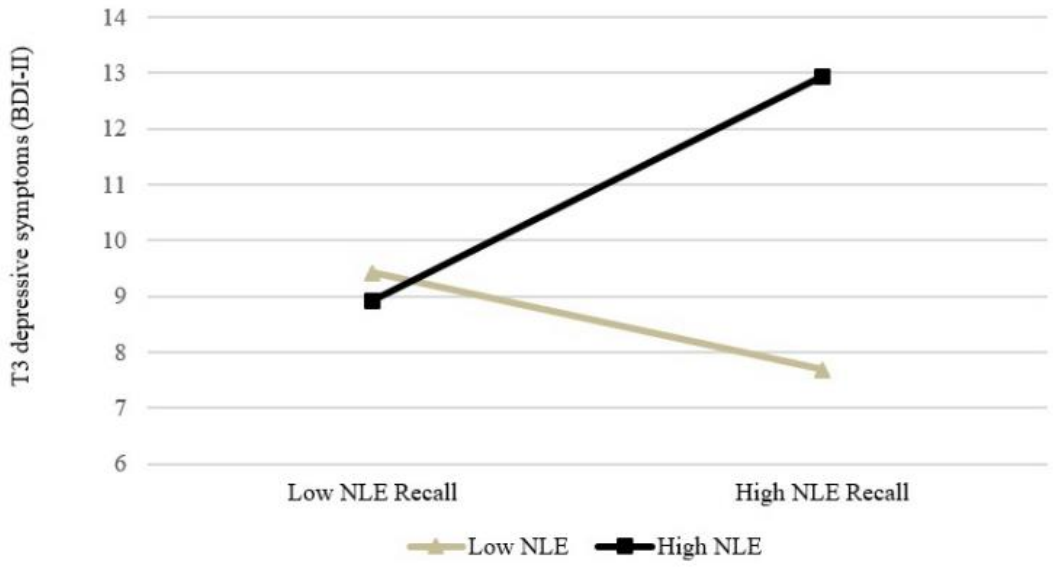
Neither aggregated SRRS-S score (B = -.08, p > .10) nor proportional recall of NLEs (B = -2.57, p > .10) independently predicted increases in T3 BDI-II score, and aggregated SRRS-S was not significantly correlated with recall (r = -.14, p > .10), precluding any follow-up tests of mediational relationships between variables. Therefore, the hypothesis that negative recall biases would interact with the experience of stress to increase depressive symptoms was tested outside of the proposed mediation model. There was a significant interaction between number of NLEs reported during the EMA week and the proportion of NLEs recalled at T2 in predicting depressive symptoms at T3, controlling for T2 depressive symptoms (B = 1.08, p < .05; Table 2.2, Figure 2.1). The effect was significant when proportional recall was high (75th percentile and above), such that individuals who endorsed and recalled more NLEs were significantly more depressed at follow-up compared to individuals who demonstrated high proportional recall but endorsed fewer NLEs during the EMA week (conditional effect = .41, t = 2.74, p < .01). Among those with low recall of NLEs, there was no significant difference in T3 depressive symptoms based on number of NLEs endorsed. The effect also was marginally significant among those with high numbers of NLEs (90th percentile and above), in that those who recalled more of their NLEs displayed greater increases in BDIII at follow-up than those who recalled fewer of their NLEs (conditional effect = 9.79, t = 1.84, p < .07). There was no significant effect when number of NLEs endorsed was low.

Table 2.2 Negative Life Events Interact with Proportional Event Recall to Predict T3 Depressive Symptoms (BDI-II)

Predictor	*B*	*SE*
Intercept	1.23	.81
T2 BDI-II	.81***	.06
NLE occurrence[a]	.20*	.10
NLE proportional recall[a]	2.92	3.03
NLE occurrence X proportional recall	1.08*	.47

Note. Analyses include 118 participants. NLE = negative life events; Proportional recall = NLEs recalled at T2/Total NLEs endorsed during EMA week.
* $p < .05$, ***$p<.001$

Figure 2.1 Negative Life Events Interact with Proportional Event Recall to Predict T3 Depressive Symptoms (BDI-II)



Note. Analyses include 118 participants. T2 depressive symptoms (BDI-II score) were controlled. NLE = negative life events; Recall = NLEs recalled at T2/Total NLEs endorsed during EMA week. High and low values ± 1 SD from grand means.

Discussion

To our knowledge, this is the first study to employ EMA methodology to test the hypotheses that 1) engaging in more stress-reactive rumination (SRR) directly after the occurrence of negative life events (NLEs) will increase the likelihood of their recall, and 2) that this improved recall of negative autobiographical information will prospectively predict increases in depressive symptoms. The current design provides a fine-grained analysis of the proposed theory that repetitive, ruminative thought strengthens the encoding and subsequent retrieval of negative information, thereby fueling negative memory biases and perpetuating depressed affect over time (Joormann et al., 2007; Joormann, 2010).

Negative events followed by increased levels of SRR relative to individuals' means were more likely to be recalled at two-week follow-up, such that a ten-point increase in SRR score improved the odds of the event being remembered by 20%. This effect held when controlling for the objective intensity rating of the event, as determined using standardized a priori criteria, as well as event chronicity. Of note, prior to including SRR in the regression equation, participants' subjective rating of the event's intensity was a significant predictor of recall. However, this effect became nonsignificant when SRR was added to the model, indicating that the degree to which one ruminates about a stressor is a stronger predictor of its recall than its perceived severity. Level of depressed affect experienced by participants at time of event reporting was not a significant predictor of event recall. Person-level characteristics, including age, sex, history of MDD, long-term memory for neutral stimuli, baseline depression and rumination scores, and

depressive symptoms at time of recall did not significantly influence the likelihood of an event being remembered.

These findings serve as the first direct evidence of a relationship between the degree of rumination engaged in following the experience of daily stress and the subsequent recall of that information. The current results support theoretical accounts suggesting that repetitive, ruminative thought strengthens the encoding and maintenance of negative information, contributing to long-term negative memory biases (Joormann et al., 2007; Joormann, 2010). These findings are particularly strengthened by their situation within an EMA framework assessing cognitive response to, and subsequent memory for, actual NLEs experienced outside of the laboratory during the course of participants' daily lives.

The results build on the extant body of literature demonstrating links between heightened rumination, whether measured at the trait level or prompted through in-lab induction, and improved memory for negative information. Previous studies have found such relationships with regards to recall of negative words (Hertel & El-Messidi, 2006; Moulds et al., 2007), including words participants were instructed to forget (Hertle & Gerstle, 2003; Joormann & Tran, 2009). Our findings are also consistent with social anxiety research examining the relationship between post-event processing, a form of repetitive, maladaptive thought in response to stress similar to rumination, and event recall (Brozovich & Heimberg, 2008; McEvoy et al., 2010). Engaging in increased post-event processing was associated with improved memory for an in-lab stressor, providing strengthened support for the relationship between repetitive thought following stress and

increased recall of that information over time (Cody & Teachman, 2010; Mellings & Alden, 2000).

Current findings extend this literature by demonstrating a relationship between rumination and the recall of daily life stress employing EMA methodology. The use of EMA provides multiple advantages in the study of NLEs and rumination. First, this design ensured that the stressors being assessed were uniquely relevant to each participant, as they were actual NLEs that participants reported during the course of their daily functioning outside of a controlled laboratory setting. By sending participants multiple alerts per day, the current design aimed to reduce retrospective recall biases in the reporting of life stress and SRR. This momentary sampling approach allowed for more accurate measurements of both NLE occurrence and the degree to which participants engaged in SRR directly after the stressor. Furthermore, the ability to collect multiple measurements of life stress, SRR, and depressive symptoms per participant increased the reliability of the data and allowed for sophisticated statistical analyses partitioning both within- and between-person variance. As such, the current study demonstrated that when individuals engaged in greater levels of SRR relative to their own means following the experience of stress, they were more likely to recall that stressor at follow-up. Thus, the current findings shed important light on the role that within-person variation in coping style plays in our memory for life stress over time.

Contrary to hypotheses, neither the aggregated degree of SRR engaged in during the EMA week nor the proportional recall of NLEs at follow-up was a significant independent predictor of increases in depressive symptoms. Therefore, no mediational relationship was found such that NLE recall explained the effects of SRR in increasing

follow-up depression scores. However, a significant interaction emerged between the number of NLEs reported during the EMA week and proportional recall of those events in predicting depressive symptoms at the T3 assessment. It was found that demonstrating higher rates of NLE recall was more detrimental among those who experienced greater levels of stress during the EMA week compared to those with lower stress. A marginally significant effect also was present among those with higher NLE endorsement; endorsing and subsequently recalling more stressors led to greater increases in depressive symptoms compared to those who endorsed high levels of NLEs but demonstrated poorer recall of those events.

It is somewhat surprising that aggregated SRR levels during the EMA week did not predict increases in depressive symptoms in the current sample. Follow-up analyses confirmed that trait-level SRR measured at T1 (SRRS score) also was not predictive of increases in T3 depression scores. It is possible that stronger relationships between SRR and depressive symptoms at follow-up would be observed when employing a depressed sample versus the current nonclinical sample. However, it is worth noting that relative daily increases in SRR were found to predict same-day depressive symptoms, controlling for symptoms on the previous day, suggesting that SRR may exert more proximal effects in increasing depressed mood over time (see Chapter 1). In a similar vein, aggregated SRR scores were not correlated with subsequent recall of NLEs, contrary to study predictions. However, given findings that event recall was predicted by increases in SRR relative to individual means and not by overall higher SRR levels, it is not particularly surprising that this correlation was nonsignificant. It is possible that other aspects of SRR, such as the difference between rumination in response to negative and

neutral/positive events may be more closely related to overall negative recall scores. For instance, individuals who demonstrate significantly greater degrees of repetitive thought following negative events compared to their response to neutral or positive events may possess stronger negative recall biases. Exploration of such hypotheses would be an important extension of the current research.

Although proportional recall of NLEs did not independently predict changes in depressive symptoms, its deleterious effects emerged in interaction with the experience of more NLEs during the EMA week. This finding is consistent with vulnerability-stress models of depression, positing that cognitive vulnerabilities are more strongly expressed following the occurrence of stressors (Abramson et al., 2002). The current results support the hypothesis that negative autobiographical memory biases are not merely a correlate of depression, but serve as an important vulnerability factor predictive of increases in depressive symptoms over time. Indeed, previous research has demonstrated that biased recall of emotional stimuli (e.g., word lists) prospectively predicted increases in depressive symptoms in a nonclinical sample (Connolly, Abramson, & Alloy, 2016). Furthermore, an earlier study demonstrated that individuals who reported experiencing more intrusive negative autobiographical memories displayed greater prospective increases in depressive symptoms over time (Brewin, Reynolds, & Tata, 1999). Although this design did not explicitly test recall of past events and instead relied on self-reported estimates of intrusive memory occurrence, it does suggest that increased negative autobiographical memory may serve as a risk factor for subsequent depression.

To our knowledge, this is the first study to utilize EMA methodology to measure biases in the recall of NLEs experienced in individuals' daily lives. This design presents a

compromise between 1) paradigms in which individuals recall predetermined negative information in the laboratory, which allows for standardization of stimuli but sacrifices personal relevance, and 2) studies in which individuals freely recall autobiographical memories, which assesses self-referent information but precludes the examination of cognitive processes during initial memory encoding. The current paradigm was further strengthened by its use of both a free and cued recall condition to best capture participants' subsequent memory for life stress. In addition, the inclusion of the standardized researcher-administered Life Events Scale and Interview (Alloy & Clements, 1992; Safford et al., 2007) helped to decrease subjectivity in the report of stress during the EMA week. Neutral memory was also assessed using the CVLT-II (Delis et al., 2000) in order to ensure that effects were specific to recall of negative autobiographical information and were not better explained by overall memory abilities.

Several limitations within the current design also should be noted. Despite the use of an EMA approach, the possibility of recall bias still remains given that participants were retrospectively reporting on the experience of NLEs and SRR since their last alert. However, as has been discussed elsewhere (Ruscio et al., 2015), there are flaws inherent in the alternative of having participants initiate questionnaire completion directly after the experience of a stressor, in that response rates would likely vary based on participant and event characteristics. Furthermore, although associations were found between 1) SRR and event recall and 2) negative memory bias and depressive symptoms, the full mediation model of SRR predicting event recall predicting depression was not significant, precluding comprehensive conclusions regarding the hypothesized relationship between SRR and recall biases in increasing depression over time.

In addition, the events experienced during the EMA week were overwhelmingly mild in intensity, as has been reported in a similar EMA study (Ruscio et al., 2015). As such, there may be a need for standardized life event interviews modified for EMA usage that would better capture variation in the types of life events reported during a typical week in participants' lives. The current design also did not include a measure of inhibitory control. Given theory that rumination stems from impairments in the inhibition of negative information (Joormann et al., 2007; Joormann, 2010), it would be important to assess whether current findings were related to trait-level inhibitory deficits, as would be expected. Finally, despite there being a wide range of depressive symptoms in the current sample, it aimed to represent a primarily nonclinical population in order to test the potential relationship between SRR, NLEs, and memory biases as *vulnerabilities* for depression. However, it would be important to test these relationships in clinically depressed samples to further understand the interplay between these cognitive factors.

The current findings invite exciting clinical extensions. These results suggest that engaging in greater rates of SRR relative to one's mean following the occurrence of stress is predictive of improved recall of NLEs, and that negative recall biases increase depressive symptoms over time. Findings indicate that, instead of depressive symptoms solely being influenced by broader and more stable individual-level traits, affect can vary based on event-level fluctuations in ruminative responses and recall. Therefore, results support the use of interventions aimed at reducing ruminative responses directly after the experience of stress, as this would be predicted to lessen subsequent event recall and potentially decrease depressive symptoms.

Cognitive bias modification (CBM) is a promising area of research aimed at targeting vulnerabilities including rumination and negative memory biases in order to ultimately improve mood outcomes (Hertel & Mathews, 2011). Indeed, training participants to engage in more concrete thought as opposed to rumination has been found to decrease depressive symptoms (Watkins, Baeyens, & Read, 2009). CBM also has been employed in depressed samples to decrease perseverative thought in response to negative stimuli (Joormann, Hertel, LeMoult, & Gotlib, 2009), and recent research has demonstrated that training participants to repetitively focus on positive, versus negative, information can have beneficial effects on mood state (Hertel, Amaris, Cottle, & Vrijsen, in press). Future research would benefit from integrating CBM and EMA methodology to assess the effects of cognitive training on response styles and subsequent mood when encountering daily stressors.

In sum, the current EMA study serves as the first direct evidence of a relationship between stress-reactive rumination following the experience of negative life events outside of the laboratory and improved subsequent recall of that information. Furthermore, increased memory for daily stressors, in combination with greater levels of reported stress during the EMA week, was predictive of increases in depressive symptoms at follow-up, controlling for previous symptoms. Thus, these findings provide important preliminary support for the hypothesis that engaging in rumination leads to strengthened encoding and retrieval of negative autobiographical memories over time, highlighting an important mechanism through which rumination may contribute to the development and maintenance of depression. Current results suggest that clinical interventions intended to alter ruminative responses directly after the experience of stress may help to decrease negative memory biases and alleviate depressive symptoms.

CHAPTER 3

SYSTEMATIC LITERATURE REVIEW

Rumination and Memory Biases as Vulnerabilities for Depression:

An Integrated Cognitive Risk Account

Abstract

Rumination and memory biases have been identified as important cognitive vulnerabilities for depression. The current review presents theory and research suggesting a potential interplay between these risk factors. A growing body of literature demonstrates links between rumination and the strengthened encoding and retrieval of negative memories, as well as the increased production of overgeneral memories. Although the majority of studies suggest a causal relationship in which rumination fosters memory biases, intervention research is also reviewed in which alterations in memory production may, in turn, influence rumination levels. Potential overarching mechanisms that may contribute to both rumination and memory biases are considered, including inhibition impairments and a tendency towards abstraction. Findings are integrated and recommendations for future areas of research are discussed.

A wealth of literature has identified cognitive mechanisms that may contribute risk for depression, ranging from biases in attention and memory, to difficulties in set-switching or cognitive flexibility, to engaging in rumination or possessing negative inferential styles. However, a major limitation of much of this research is its tendency to study these cognitive factors in isolation versus examining the potential interplay between vulnerability factors in predicting depression. Hirsch, Clark, and Matthews (2006) outlined the importance of this integration within their combined cognitive bias hypothesis, in which they postulated that differing vulnerability factors can influence and interact with each other and, in turn, can exert a stronger influence on the development and maintenance of psychopathology than if these biases worked in isolation. Whereas Hirsch et al. presented support for their hypothesis within the context of social anxiety, their theory has since been examined within a depression framework (Everaert, Koster, & Derakshan, 2012). Indeed, considerable evidence exists in support of an interplay between biases of attention, interpretation, and memory in the prediction and maintenance of depressed mood.

One particular relationship of interest is that between rumination and memory biases, two cognitive vulnerabilities that have been consistently linked to depression (Dalgleish & Watts, 1990; Williams et al., 2007; Wisco & Nolen-Hoeksema, 2008). It is possible that rumination, a repetitive, maladaptive thinking style in which one broods about negative self-referent information, may cause deepened encoding of this information and lead to subsequently increased recall of negative self-relevant information over time. This hypothesis has been proposed both indirectly and, more

recently, explicitly within a novel theory of the cognitive processes that contribute to depression (Joormann, 2010). Furthermore, in addition to perhaps being biased towards recalling negative information, it also has been found that the *quality* of memories may differ among those who ruminate, in that their memories are more overgeneral in nature. It has been hypothesized that an underlying tendency towards abstraction may contribute to both rumination and overgeneral memory retrieval (Williams et al., 2007; Watkins, 2015). The current review aims to integrate theory and evidence in support of this proposed interplay between rumination and memory biases in the prediction of depression. Better understanding the relationship between these cognitive vulnerabilities serves as an important step in characterizing the onset of depression and the best ways to intervene in its development and persistence over time.

1. Theoretical background

The concept that memories are influenced by an individual's unique perception and interpretation of a given event is longstanding. Some of the earliest evidence for this idea was presented in Frederic Bartlett's (1932) "War of the Ghosts" experiment, in which he demonstrated that individuals displayed preferential recall of story details that were more in line with their own past experiences and schema, and were more likely to forget or modify non-schema congruent information. This study serves as a very early example of the ways in which an individual's self-concept and experiences influence subsequent memory for information. Indeed, later researchers expounded on this idea, theorizing that information congruent with one's self-schema may be encoded deeper or more strongly than non-self-referent stimuli (Derry & Kuiper, 1981; Rogers, Kuiper, & Kirker, 1977).

As Rogers and colleagues (1977) postulated in their influential study of memory processes, "When self-reference is involved, it should provide a useful device for encoding or interpreting incoming information by virtue of accessing the extensive past experience abstracted in the self" (p. 679). Rogers et al. went on to demonstrate in a series of studies that individuals displayed improved recall of words that were encoded self-referentially, compared to words that were processed based on their semantic, structural, or phonemic qualities.

These early studies demonstrate a link between the self-relevance of events or stimuli and subsequent memory for this material, and in many ways can be integrated into a conceptualization of the relationship between ruminative thought processes and memory bias. As opposed to active problem-solving, rumination refers to a maladaptive cognitive style in which one repetitively and passively dwells on negative information about oneself (Nolen-Hoeksema, 1991). Rumination is a strong vulnerability factor for depression and is predictive of the onset, length, and number of depressive episodes experienced (e.g., Just & Alloy, 1997; Roberts, Gilboa, & Gotlib, 1998; Spasojević & Alloy, 2001). Although many studies have identified rumination as a contributor to depression, the mechanism through which this cognitive style confers risk is not fully understood. It has been proposed that rumination may contribute to depressed mood by increasing the availability of negative thoughts and memories (Wisco & Nolen-Hoeksema, 2008).

This link between rumination, memory, and depression has been outlined in Joormann's impaired cognitive control account of depression (Joormann, Yoon, &

Zetsche, 2007; Joormann, 2010). Joormann proposes that individuals with impaired inhibitory processes are less able to prevent negative information from entering working memory and subsequently have difficulties removing that information. As a consequence, these individuals are thought to engage in a higher degree of elaboration and rehearsal of this information, which can be conceptualized as the negative response style of rumination. Due to this maladaptive, repetitive processing of negative information, Joormann hypothesizes that this information will become more deeply encoded within one's long-term memory. In the final step of the model, it is proposed that having more negative information within long-term memory, coupled with a tendency to ruminate on this information, will serve to further perpetuate depressed mood. As individuals experience negative events and low mood, this chain of cognitive processes will occur again.

Rumination also has been thought to contribute to depression by increasing overgeneral memory production. The phenomenon of overgeneral autobiographical memory, in which individuals retrieve categorical memories (e.g., I always used to get hurt as a kid) when prompted to recall a specific memory (e.g., The day I fell off my bike and got hurt) after being presented with a cue word (e.g., "hurt")**,** has been consistently associated with depression (Williams et al., 2007). This tendency to generate more abstract, nonspecific memories has been linked to increased hopelessness, difficulty imagining future events, and impaired problem-solving abilities, and is considered to be an indicator of depression and a potential vulnerability factor for the disorder.

Overgeneral memory also predicts poorer long-term depression outcomes (see Williams et al., 2007 for a review).

It has been theorized that rumination may play a significant role in the development of overgeneral memories. Conway and Pleydell-Pearce (2000) proposed that overgeneral memories arise when individuals discontinue their search for specific memories while still within a higher-order, categorical level of memory. It has been thought that this process may represent a form of avoidance of negative affect, aiming to prevent engagement with specific, potentially painful memories from one's past by remaining at a level of abstraction. This hypothesis is further supported by strong demonstrated links between the experience of trauma and the production of overgeneral memories (Williams et al., 2007). In addition to avoidance, Williams et al. (2007) posited that rumination and impaired executive control also may contribute to the retrieval of overgeneral memories, as presented in their Capture and Rumination, Functional Avoidance, and Impaired Executive Control (CaRFAX) model of autobiographical memory. This model suggests that individuals who engage in rumination possess a well-developed, easily accessible network of abstract self-conceptualizations as a result of their tendency to engage in repetitive self-focused thought. When asked to generate a specific memory based on an emotional cue word, Williams et al. hypothesized that ruminators are more prone to become stuck within this abstract level of self-representation and subsequently produce an overgeneral memory, instead of advancing through the retrieval process and generating a specific memory. Furthermore, impairments in executive control and inhibitory abilities, which have been linked to

rumination, may make it increasingly difficult to detach from these abstract, negative thoughts and focus on the retrieval of a specific memory. In considering these theories, Watkins (2015) suggests that abstraction may serve as an underlying factor that leads to both ruminative thought and overgeneral memory production.

Despite addressing different facets of the relationship between rumination and memory biases, Joormann's impaired cognitive control account and Williams' CaRFAX model can, in part, be considered complementary theories. Importantly, both models place an emphasis on the role of impaired inhibitory processes in perpetuating rumination and memory biases (for a review of inhibition impairment and rumination, see Joormann et al., 2007). Joormann's model focuses more on the extent to which impaired inhibition among ruminators allows for deeper *encoding* of negative information, whereas the CaRFAX model focuses on the idea that these inhibitory deficits also may interfere with the subsequent *retrieval* of specific memories when prompted. Indeed, it is quite possible that ruminative thought influences memory processes at both the encoding and retrieval stages, as will be reviewed here.

Rumination, characterized by its repetitive nature and negative, abstract self-referent content, appears to possess many of the qualities that would foster negative and overgeneral memory biases, and, in turn, these biases may perpetuate ruminative thought processes. Although a collection of studies independently demonstrates the existence of this proposed relationship, these findings have yet to be the subject of an integrated review. The current effort aims to address this need, examining the varying ways that rumination and memory biases may together influence the development of depression.

This review first discusses studies that assess the relationship between rumination and increased encoding of negative information using a variety of cognitive paradigms. Studies are then reviewed in which rumination is linked to heightened recall of autobiographical memories. Next, research demonstrating links between rumination and increased overgeneral memory production is discussed, including data from the intervention literature. Findings are then integrated, and potential underlying mechanisms that may be responsible for these observed relationships are considered. Finally, avenues for future research are presented, aimed at better understanding this purported link between rumination and memory biases in contributing to depression.

2. Review of studies of rumination and memory biases

2.1. Rumination and encoding of negative information

As outlined in Joormann's (2010; Joormann et al., 2007) impaired cognitive control account, it is possible that the act of rumination may lead to negative information becoming more strongly encoded, which may serve as a crucial mechanism in increasing its likelihood of being later retrieved. Several studies have explored this question, in order to better understand this purported link between rumination and negative memory biases.

Moulds, Kandris, and Williams (2007) examined the link between rumination, the encoding of self-referent information and depression. In their study, 93 college students were classified as dysphoric or nondysphoric based on their self-reported depressive

symptoms as measured by the Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996); dysphoric participants had BDI-II scores of 13 or above and nondysphoric participants had scores of 5 or below. Participants were randomized to either a rumination or distraction condition, in which they underwent a well-established rumination or distraction induction procedure (Nolen-Hoeksema & Morrow, 1993; Lyubomirsky & Nolen-Hoeksema, 1993) referred to hereafter in this review as the "standardized rumination induction." In this task, participants are instructed to focus on a series of 45 items; in the rumination condition, these items involved emotions, depressive symptoms, and the self (e.g, "think about trying to understand your feelings"), whereas in the distraction condition, the items were external in nature (e.g., "think about clouds forming in the sky"). Participants in each condition spent eight minutes focusing on the items, after which they completed a self-referent memory task (Bradley & Mathews, 1988; Dalgleish, Spinks, Golden, & du Toit, 2004). In this task, participants first were instructed to think of an acquaintance they knew. They then were presented with three lists of eight positive and eight negative adjectives; half of the words were preceded by the word "self," whereas the remaining words were preceded by the word "other;" in the "self" condition, participants rated whether or not the word described them, whereas in the "other" condition, they rated whether the word described their acquaintance. Participants were told that their memory for these words would be tested following this rating component. After each list, participants completed a short counting interference task, and then recalled as many adjectives as they could from the list. Moulds et al. hypothesized that dysphoric individuals induced to ruminate would endorse more

negative self-referent words compared to participants in the remaining three conditions (dysphoric distraction, nondysphoric rumination, and nondysphoric distraction), as rumination would increase negative self-focus. Furthermore, they hypothesized that individuals in this condition also would recall more of the negative self-referent words, as, in line with the impaired cognitive control account, it was suggested that rumination may lead to deeper processing and encoding of this negative information.

Moulds et al. did not find that individuals in the dysphoric rumination group endorsed more negative self-referent words. However, they did find that dysphoric participants as a whole endorsed more negative self-referent words compared to the nondysphoric group, as did individuals in the rumination condition compared to the distraction condition. Interestingly, the authors' hypothesis with regards to recall was supported. A group by condition interaction indicated that dysphoric participants receiving the rumination induction recalled significantly more endorsed negative self-referent adjectives compared to those in the distraction condition; no differences were found between conditions for nondysphoric participants. It should be noted that all recall analyses controlled for the number of negative self-referent adjectives initially endorsed, in order to better isolate participants' recall abilities. This study provides support for the impaired cognitive control hypothesis by demonstrating that rumination occurring just prior to the presentation of negative self-referent stimuli increases subsequent recall of this information. Although dysphoric participants across both conditions endorsed more negative words as self-referent, only those who ruminated subsequently recalled more of these words. The authors postulated that the rumination induction led participants to

dwell on their endorsed negative words, leading to deeper encoding and improved retrieval, compared to participants encouraged to distract. Findings suggest that rumination may play an important role in perpetuating the negative memory biases and, in turn, negative self-schemas observed in depressed individuals.

A unique study by Hertel and El-Messidi (2006) provides further support for this claim. Thirty-two dysphoric participants with BDI (Beck, 1961) scores of 10 and above and 32 nondysphoric participants with BDI scores of 5 and below underwent a slightly modified version of the standardized rumination induction. They then completed a homograph task, in which they viewed 24 words presented individually on a computer screen for two seconds each. Twelve of these words were homographs; words with both a personal and impersonal meaning (e.g., bitter, blue, odd). Participants were asked to blurt out the first two words that came to mind when seeing each word. Responses were coded by two raters to determine whether participants had interpreted the cue word personally or impersonally. Participants were then given a blank sheet of paper and asked to recall as many words from the task as they could. As in the previously reviewed study, it was hypothesized that dysphoric participants in the rumination condition would recall more words that they interpreted personally compared to the dysphoric distraction group. Indeed, this hypothesis was supported, and no significant differences in recall were found between conditions for the nondysphoric group. Findings again indicate that dysphoric participants induced to ruminate show improved memory for negative, personally interpreted information, suggesting that rumination may lead to improved encoding of this material.

An alternative mode of measuring differences in the encoding of negative material assesses recollection versus familiarity towards task stimuli, in which recollection refers to remembering contextual details associated with the presentation of a stimulus, and familiarity refers to having seen a stimulus, but not recalling any additional related information (Yonelinas, 2002). As would be expected, recollection is thought to reflect deeper stimulus encoding compared to familiarity. Kuo et al. (2012) sought to examine whether trait rumination was associated with a greater rate of recollection versus familiarity of negative self-referent stimuli. Of note, the authors employed a nonclinical sample to assess this relationship independent of depressive symptoms. Ninety-seven female participants completed the Ruminative Responses Scale, a well-established self-report measure of rumination (RRS; Nolen-Hoeksema & Morrow, 1991), and then completed a computerized self-referent encoding task. In this task, participants viewed 75 positive, negative, and neutral adjectives, each presented twice and preceded by the phrase "You are." Participants were instructed to think of themselves as each word. Following a 20 minute delay, participants then were presented with each word again, along with never before presented words, and asked if each word was old or new. If participants indicated a word to be "old," they were asked if they remembered contextual details of being presented with the word (measuring recollection) or whether they knew the word was presented but did not remember any associated details of viewing the word (measuring familiarity). The authors found a significant interaction between rumination score and recollection, such that participants with higher levels of rumination recalled more negative words compared to neutral and positive words. This relationship remained

significant when controlling for state-level negative affect. Kuo et al. assert that findings support the link between rumination and deeper, elaborative encoding of negative stimuli. It is notable that Kuo et al. found significant results utilizing the described self-referent encoding task, given that most similar tasks involve participants making a personal judgment of whether or not a given word describes them, as these words would be hypothesized to be encoded more deeply. Although individuals scoring higher in rumination likely would have endorsed more of the negative adjectives as being self-referent, this information is not available given the design of the task and as such represents a limitation and avenue for future research.

Biases in encoding also have been examined in studies of directed forgetting, in which participants are presented with words that they are then told to forget in order to focus on so-called target stimuli. When they are then asked to recall all stimuli from both conditions, participants typically display improved recall of to-be-remembered words, and lowered recall of to-be-forgotten words (Johnson, 1994). However, it has been found that depressed individuals have demonstrated stronger memory for to-be-forgotten emotional stimuli compared to controls (Power, Dalgleish, Claudio, Tata, & Kentish, 2000). This phenomenon has been studied in relation to rumination, with researchers hypothesizing that individuals who ruminate may devote more attentional resources towards emotional information regardless of forgetting condition. This, in turn, would lead to increased encoding and therefore improved recall of to-be-forgotten emotional words compared to low ruminators.

Joormann and Tran (2009) explored this hypothesis in a study of 33 undergraduates divided into low and high rumination groups using a median split of scores on the RRS. Participants also completed the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), a self-report measure of depressive symptoms, followed by a computerized directed forgetting task. Participants were shown a series of 10 negative and 10 positive words, presented individually on the screen, and were instructed to indicate the word valence and memorize each word. After completing this set, participants were informed that this was a practice trial and to forget all words presented in order to prepare for the actual trials. They then completed the same procedure with a set of 20 new words. Finally, participants engaged in a free recall task in which they were asked to recall all words from both the to-be-forgotten (TBF) and to-be-remembered (TBR) conditions. It was found that participants in the high rumination group remembered more TBF words compared to the low rumination group. This effect did not vary by word valence, and remained when controlling for participants' depressive symptom scores. Joormann and Tran concluded that findings suggest a specific role of ruminative thought in the reduced forgetting of emotional stimuli. Results supported the hypothesis that rumination may be linked to difficulties inhibiting the processing of emotional information, leading to improved encoding and subsequent recall of these stimuli. Although Joormann and Tran's findings were not specific to negative information, the authors suggested that such a finding might emerge when examining this relationship within a dysphoric or depressed sample. Interestingly, high ruminators also were more likely than low ruminators to "recall" negative words that had never been

presented during the task. It was suggested that this may be due to high ruminators engaging in increased elaboration of the negative stimuli presented to them, causing them to form associations with related words not included in the task.

Hertel and Gerstle (2003) explored this directed forgetting hypothesis within a sample of 32 dysphoric (BDI scores of 9 and above) and 32 nondysphoric (BDI scores of 6 and below) participants. Rumination was measured using the self-report Rumination on Sadness Scale (RSS; Conway, Csank, Holm, & Blake, 2000). In their design, participants learned pairs of nouns and positive or negative adjectives (e.g., cozy chair; electric chair). They were then presented with the cue alone (e.g., chair) and had to say out loud its corresponding target word (e.g., cozy). In a second phase, participants were instructed to continue with this procedure for some of the words, but to "suppress" certain cues and try not to think about or say the corresponding target out loud. Half of the participants were assigned to suppress negative word pairings, whereas the other half suppressed positive word pairings. Finally, participants were asked to recall the target for each cue word, regardless of previous suppression instructions. The authors found that the dysphoric group demonstrated impaired suppression of TBF words compared to the nondysphoric group. Interestingly, when dividing the sample in half based on rumination score, the high rumination group demonstrated the same pattern of impaired directed forgetting, regardless of depression condition. Of note, BDI scores were not significantly different between rumination groups, suggesting that rumination effects were not primarily explained by depression score. This finding, along with the work of Joormann and Tran (2009), indicates the presence of a relationship between trait-level rumination and

impaired forgetting of emotional material, which may be the result of increased encoding among high ruminators. Interestingly, no significant valence effects emerged, even when employing a dysphoric sample in the current design. The authors suggested that perhaps a clinically depressed sample would be necessary to observe such valence-specific effects; indeed, the absence of studies employing clinical samples can be considered a limitation within this literature.

Wong and Moulds (2008) employed a rumination induction to further test this directed forgetting hypothesis, positing that dysphoric participants (BDI-II of 12 or above) would recall more TBF negative words after receiving a rumination induction, compared to dysphoric participants in the distraction condition. They also predicted that nondysphoric participants (BDI-II of 5 or below) would show standard directed forgetting effects regardless of condition. Eighty participants underwent the standardized rumination induction and then completed a computer task in which they were shown 30 positive, negative, and neutral words (e.g., cheerful, downcast, windows). Each word was presented for two seconds, followed by the instruction to either remember or forget the word. Participants were then told to write down as many words as they could remember from both the TBR and TBF conditions. Contrary to the previous two directed forgetting studies, Wong and Moulds did not find support for their hypotheses, as there was no difference in directed forgetting of positive, negative, or neutral words between the rumination and distraction conditions among dysphoric participants; there were also no notable differences in directed forgetting between the dysphoric and nondysphoric groups. The authors suggested that perhaps their low dysphoria cut-offs may have

contributed to these null findings, although it should be noted that the majority of the studies reviewed thus far have used similarly low dysphoria thresholds. It is possible that the current design did not allow for deep enough encoding of target stimuli. Whereas the previous two directed forgetting studies required participants to form associations with the presented words by either indicating word valence or learning word pairs, the task employed by Wong and Moulds only asked participants to either remember or forget the presented words. Future directed forgetting studies could aim to manipulate learning instructions to test whether differences in depth of encoding may be at play.

It is also important to consider that direct forgetting paradigms may, in fact, be better measures of retrieval, versus encoding, of emotional information. As Power et al. (2000) asserted, although recall of TBF words in standard directing forgetting tasks is lower than TBR words, it has been found that the *recognition* of these words remains intact. The authors discuss that, if these tasks were indeed capturing weaker encoding of the TBF words, they would be more poorly recognized as well. Instead, Power et al. proposed that the poorer recall of TBF words is the result of weaker retrieval processes, and that these processes happen to be stronger among dysphoric participants when accessing emotional information. This argument emphasizes the difficulty of isolating differing memory processes within experimental designs. In particular, it appears especially unwieldy to demonstrate that a task is measuring the encoding process, as in all studies reviewed thus far, memory is measured through free recall conditions that by their nature require the participant to *retrieve* information from memory. Indeed, Wong and Moulds (2008) note that in their study, participants reported engaging in varied

encoding strategies during the directed forgetting task; whereas some participants engaged in minimal word rehearsal until learning whether or not the target word was TBR or TBF, others engaged in more elaborative encoding upon seeing each word. Future studies would benefit from systematically manipulating encoding instructions across participants, or from gathering more specific data regarding participants' encoding strategies and assessing differences in subsequent retrieval performance.

In sum, a collection of studies employing differing paradigms have suggested a link between ruminative thought and increased encoding of emotional stimuli. Whereas some studies suggest the presence of a specific bias towards the encoding of negative stimuli (Hertel & El-Messidi, 2006; Kuo et al., 2012; Moulds et al., 2007), directing forgetting studies found biased encoding of both positive and negative information (Hertel & Gerstle, 2003; Joormann & Tran, 2009). Findings of biases towards emotional stimuli in general have been reported within many other studies of rumination and cognitive function, particularly in the overgeneral memory literature (Williams et al., 2007). Indeed, it has been hypothesized that even objectively positive stimuli may trigger negative associations in ruminators by highlighting discrepancies between their actual state and their desired state, leading to comparable effects across valence conditions (e.g., the word "happy" triggering thoughts that the individual is not as happy as they should be). It is also important to note that some studies suggest these observed effects of rumination only emerge among dysphoric samples (Hertel & El-Messidi, 2006; Moulds et al., 2007), whereas others demonstrate a specific relationship between biased encoding and rumination, independent of depressive symptoms (Hertel & Gerstle, 2003; Joormann

& Tran, 2009; Kuo et al., 2012). Although it is still unclear to what extent current dysphoria is necessary for observed rumination effects to emerge, these findings together provide strengthened support for the hypothesis that rumination contributes to observed memory biases, over and above what may be attributed to depressive symptoms.

2.2. Rumination and retrieval of autobiographical memories

In addition to leading to stronger encoding, engaging in self-focused, ruminative thought has been demonstrated to increase the subsequent recall of negative autobiographical memories. Perhaps the first evidence for this relationship emerged in a pair of studies on the relationship between self-focus and memory by Pyszczynski, Hamilton, and Herring (1989). The concept of self-focus employed in this study referred to the act of perseverating on the discrepancy between one's desired self and one's actual self among individuals with depression. This form of maladaptive, self-referent thought in many ways can be equated to the construct of rumination that would soon gain traction in the field (Nolen-Hoeksema, 1991). Pyszczynski and colleagues conducted two experiments to test the hypotheses that 1) negative memory biases observed in dysphoric individuals could be attributed to their degree of self-focus, and 2) these negative memory biases were specific to autobiographical memories, and would not emerge when participants were asked to generate memories about others. In their first study, 71 male and female undergraduates with BDI scores of 9 or above (depressed group) or 4 or below (nondepressed group) were assigned to one of two conditions: a self-focus condition, in which they were instructed to write a story using a collection of self-related

words such as *I, alone,* and *me,* and an external focus condition, in which words were included such as *he, together,* and *him.* Following this writing exercise, participants in both conditions were asked to recall ten events that they had experienced during the previous two weeks. When engaging in self-focus, depressed participants recalled more negative autobiographical memories than the nondepressed group; however, there were no significant differences in the valence of recalled memories between groups in the external focus condition. The authors interpreted these findings as demonstrating that the negative memory bias observed in depressed individuals is primarily the result of their increased level of self-focus. A follow-up experiment isolated this negativity bias to autobiographical memories, as the bias did not emerge when depressed participants in the self-focus condition were asked to recall events that had happened to someone they knew. These findings provided important preliminary evidence of a relationship between self-focused thought and increased availability of negative, autobiographical memories among dysphoric individuals.

This relationship received further support in later studies that utilized measures of rumination to capture the construct of negative self-focus. Lyubomirsky, Caldwell, and Nolen-Hoeksema (1998) expanded upon this question within a series of four experiments. In their first experiment, 72 college students were divided into dysphoric (BDI scores of 17 or higher) and nondysphoric (BDI scores of 4 and below) groups. Participants underwent the standardized rumination induction and then engaged in a free recall task in which they generated as many specific autobiographical memories as they could, and subsequently rated the valence of those memories. Results were in line with hypotheses;

dysphoric participants induced to ruminate rated their memories as more negative compared to the other three conditions (dysphoric distraction, nondysphoric distraction, and nondysphoric rumination). Furthermore, dysphoric participants in the rumination condition recalled less positive memories than the other three groups; interestingly, their recall was evenly split between positive and negative memories, whereas the remaining three groups displayed a bias for recalling positive memories. However, it could be argued that these results were influenced by a negative rating bias among the dysphoric individuals. A follow-up study was conducted to rule out this possibility, in which participants were asked to recall two positive and two negative autobiographical memories, with memory valence later being rated by two independent judges. The authors' initial hypothesis held; memories generated by participants in the dysphoric rumination group were judged to be more negative, and less positive, compared to the other groups. Further support was found in two additional studies, in which members of the dysphoric rumination group reported experiencing a list of negative events more frequently (e.g., having an argument with a friend), and spontaneously generated more negative memories in an unstructured condition.

A series of experiments conducted by McFarland and Buehler (1998) expanded the literature by manipulating both rumination and mood to examine associations with negative recall. In one experiment, rumination was measured using a subset of questions from the rumination scale of the Rumination-Reflection Questionnaire (Trapnell & Campbell, 1999), a self-report measure designed to capture the degree to which individuals engage in repetitive, passive self-focus. Ruminators and non-ruminators were

classified based on a median split. Participants underwent either a negative or neutral mood induction and then completed either a mood focus exercise (identifying one's current mood state using a selection of labels) or a distraction exercise (an anagram task). They then were asked to recall five memories from their high school years. It was found that ruminators in the negative mood condition were more likely to recall negative memories when focused on their mood; however, when in the distraction condition, their recall did not vary based on mood induction. Similar findings were reported in the authors' subsequent experiments, which manipulated both the measurement of rumination (using inductions versus self-report questionnaires) and recall (e.g., assessing more recent memories, restricting memory categories). By manipulating mood state in addition to self-focus, McFarland and Buehler strengthened the claim that this relationship between rumination and negative recall biases was linked to negative mood, and not perhaps some underlying associated third variable.

This early set of studies provided preliminary evidence that individuals who engage in ruminative thought, whether at the trait level or through an in-lab induction, are more likely to recall negative autobiographical memories. Pyszczynski et al. and Lyubomirsky et al. demonstrated that this relationship emerged specifically among dysphoric individuals, and McFarland and Buehler suggested that this link could also emerge when inducing negative mood in a community sample, providing further support for this proposed relationship. The aforementioned studies acknowledged that findings could be attributed either primarily to rumination or to negative affect, as none reported a significant effect of rumination within nondysphoric conditions. When considering the

effect of rumination, it was proposed that this form of repetitive thought may increase focus on depressed individuals' negative self-schema, which, in turn, may have made it easier to retrieve schema-congruent negative memories; similarly, engaging in distraction conditions would be thought to deactivate one's schema and, in turn, decrease this proposed memory bias. When considering the effect of low mood, it was proposed that rumination served primarily as conduit, directing greater attention towards the negative memories that were generated due to one's negative mood state (Lyubomirsky et al., 1998). Although these findings suggest that both rumination and dysphoria are necessary for negative autobiographical memory biases to emerge, the aforementioned designs unfortunately do not allow for a determination of which factor is most responsible for these biases. Future studies that assess the role of rumination in negative autobiographical memory retrieval, controlling for depressive symptoms or diagnoses, are warranted.

2.3. Rumination and retrieval of overgeneral memories

Although the previously reviewed studies provide evidence of the role that rumination may play in the increased retrieval of negative autobiographical memories, they do not address the *quality* of those recalled memories. Indeed, the relationship between rumination and overgeneral autobiographical memory has become an area of growing interest and examination. This link has been demonstrated at the correlational level, as self-reported rumination has been associated with both higher rates of categorical memory recall and lower levels of specific memory recall (Ramponi, Barnard, & Nimmo-Smith, 2004). A series of studies have sought to provide further support for

this relationship by demonstrating the ability to modify memory overgenerality by manipulating rumination levels. Watkins, Teasdale, and Williams (2000) divided 48 dysphoric participants (BDI above 14) into rumination or distraction conditions. Participants first completed a version of the Autobiographical Memory Test (AMT; Williams & Broadbent, 1986). In this task, participants were given 30 seconds to recall a specific autobiographical memory that came to mind when presented with six positive, six negative, and six neutral cue words. Memories were coded as specific if they lasted less than a day (e.g., I went camping one night in June), categorical if referring to a repeated event (e.g., We went camping every weekend last summer), and extended if lasting longer than a day (e.g., We went on a weeklong camping trip). Following AMT completion, participants underwent the standardized rumination or distraction induction, and then completed a second version of the AMT. Of note, categorical memories were used to measure overgeneral memories, as this subtype of memories, and not extended memories, have been shown to be more strongly linked to depression. Watkins et al. found a significant interaction between rumination group and change in categorical memory production between AMT administrations, in that members of the distraction group demonstrated decreases in categorical memory generation during the second AMT, whereas members of the rumination group did not display any change over administrations. The authors interpreted findings as preliminary evidence of the role rumination may play in fostering overgeneral memories in dysphoric individuals, although it should be noted that the absence of a nondysphoric control group is a weakness of the design. Importantly, this study demonstrates that the production of

overgeneral memories can be modified and reduced by engaging in distraction techniques, which could have important clinical implications. Findings also provide support for the hypothesis that overgeneral memory is the result of processes occurring at the point of memory *retrieval*, given that overgenerality could be influenced by an induction just prior to the recall procedure.

Watkins and Teasdale (2001) sought to further examine this relationship with the goal of better understanding what aspect of rumination may be particularly contributing to the formation of overgeneral memories. The authors isolated two distinct aspects of rumination to examine: its degree of self-focus and its analytical nature. Their study tested the four possible combinations of these components of rumination by creating a high self-focus/high analytical group (meant to mimic rumination), a low self-focus/low analytical group (distraction), a high self-focus/low analytical group (experiential self-focus), and a low self-focus/high analytical group (abstraction) (Watkins & Teasdale, 2001; Watkins, 2015). Thirty-six dysphoric participants (BDI above 14) were randomly divided into each of the four groups. The rumination and experiential self-focus groups underwent variants of the standardized rumination induction, with the following modifications: in the rumination group, to help encourage analysis, participants were asked to think about each rumination item (e.g., "Think about what your feelings might mean"), whereas participants in the experiential self-focus group were asked to focus their attention on the experience of each item, intended to reduce analysis (e.g., "Focus your attention on the experience of the physical sensations in your body"). The distraction group followed the same prompts set forth in the standardized distraction

induction (e.g., "Think about a raindrop sliding down a pane of glass"), whereas those in the abstraction group were presented with prompts designed to encourage greater analysis (e.g., "Think about how best to define equality"). Participants first completed a version of the AMT and a self-report mood measure, then completed their group-specific induction, and finally completed a second AMT and self-report mood questionnaire.

Watkins and Teasdale obtained several important results. First, they replicated the finding reported by Watkins et al. (2000), in that those in the distraction group demonstrated significantly decreased categorical memories at the second AMT administration. Second, they found a significant interaction between categorical memories and high levels of analysis, in that those in the low analysis groups (distraction and experiential self-focus) demonstrated significant decreases in categorical memories over time, whereas those in the high analysis groups (rumination and abstraction) demonstrated near-significant increases in categorical memories. This finding is important in isolating the *analytical* component of rumination as the primary contributor to the production of overgeneral memories versus its self-focused aspect. Interestingly, a significant interaction was found between high levels of self-focus and change in mood during the task, in that those in the low self-focus conditions demonstrated reductions in low mood and increases in happiness, whereas those in the high self-focus conditions displayed increases in low mood and decreased happiness. The authors therefore concluded that whereas the self-focused component of rumination was associated with increases in despondent mood, the analytical aspect of rumination was responsible for observed increases in overgeneral memory biases. They also suggested that this specific

relationship between analytical focus and overgeneral memory, distinct from the processes leading to low mood, may help explain why this pattern of memory retrieval continues to be observed even after individuals recover from depressive episodes, a finding that has been reported within the literature (e.g., Raes, Hermans, Williams, Beyers, Brunfaut, & Eelen, 2006).

Watkins and Teasdale (2004) later replicated these findings in a follow-up study that employed a more streamlined induction design to test for differences between high and low levels of analysis towards self-focused information; of note, this study was composed of 28 clinically depressed participants as determined using a structured diagnostic interview, which extended findings to a clinical population. An additional study replicated results among a clinical sample of adolescents, finding that those with diagnoses of major depression who underwent a rumination induction demonstrated increases in overgeneral autobiographical memory, whereas those in the distraction group did not display any significant change in memory production (Park, Goodyer, & Teasdale, 2004).

The studies reviewed thus far demonstrate the ability to decrease existing levels of overgeneral memory within dysphoric individuals by reducing their degree of rumination. Researchers sought to provide stronger evidence that rumination may play a causal role in the development of overgeneral memory by examining the potential for these biases to emerge in a nonclinical sample induced to engage in ruminative thought processes. Barnard, Watkins, and Ramponi (2006) manipulated degree of rumination using a "self-

related category generation task." This task was interwoven between the AMT; 36 nondysphoric participants first completed a section of the category generation task and then completed six AMT items, alternating in this pattern until they had completed all three sections of category generation and all 18 AMT items. Within each category generation section, participants were presented with five prompts that referred to one of three domains: working, relaxing at home, and socializing. Participants were given 30 seconds to generate as many examples as they could per prompt (e.g., "Generate as many different examples as possible of the feelings you typically have when working"). In an attempt to mimic perseverative, self-focused ruminative thought, the rumination group completed the five prompts per section within the same domain (e.g., completing all prompts with reference to the working domain). In contrast, those in the non-rumination group completed prompts that varied from domain to domain within each section.

The authors found a significant interaction, in that those in the rumination condition produced less specific autobiographical memories than those in the non-rumination group. Results were interpreted as providing the first evidence of the relationship between rumination and overgeneral memory existing in the reverse direction, in that inducing rumination in nondysphoric individuals who do not typically engage in this thinking style can lead to increases in overgeneral memory. Furthermore, the authors did not find that the rumination condition affected mood, suggesting that the relationship between rumination and memory specificity was not being explained by increases in depressive symptoms. Interestingly, the authors conducted a follow-up study in which they replaced the category generation task domains with impersonal themes

related to animals, and found no significant differences in memory specificity between groups. This result emphasized that rumination's effect on overgeneral memory only emerged when individuals perseverate on information related to the self. This finding is somewhat contrary to results reported by Watkins and Teasdale (2001), who found that it was rumination's analytic, and not self-focused component that influenced overgeneral memory. Although Barnard et al.'s findings suggest that self-focus does indeed influence overgeneral memory production, it could also be argued that both conditions of Barnard et al.'s design also encouraged analytic thought, which may have contributed to their results.

Raes, Watkins, Williams, and Ramponi (2008) further explored the potential to induce overgeneral memory in a nondysphoric sample using rumination induction. In their design, the authors sought to specifically examine the role that rumination's abstract, evaluative nature plays in fostering overgeneral memory, in contrast to more concrete forms of problem-solving. One hundred ninety-five college students were divided into rumination and non-rumination conditions. In the rumination condition, participants read a series of six negative and six positive scenarios that they were to imagine themselves undergoing (e.g., spilling coffee on a customer at work). They were instructed to concentrate on each scenario for one minute and then answer a series of abstract, evaluative questions designed to foster ruminative thought (e.g., "What do you think about yourself?"). Those in the non-ruminative condition were presented with pictures of the same scenarios, asked to imagine the details of what was occurring in each scene for one minute, and then answered a series of concrete, process-focused questions (e.g.,

"Was the coffee hot or cold when it was spilled?"). Participants then completed the Sentence Completion for Events from the Past Test (SCEPT; Raes, Hermans, Williams, & Eelen, 2007), a modified test of overgeneral memory intended to be a more sensitive measure of this construct within community samples. In this task, participants complete sentence stems such as ‘‘I still remember well how..." and their response is coded for specificity. Raes et al. found that those in the non-rumination group produced less overgeneral and more specific memories compared to the rumination group, suggesting that inducing more concrete versus abstract thought can reduce overgeneral memory production within a nonclinical sample.

It is important to note that none of the aforementioned studies report significant valence effects in overgeneral memory retrieval. It has been suggested that cue words of negative, positive, or neutral valence may be just as likely to potentially trigger rumination (Williams et al., 2007). For instance, as mentioned earlier in this review, the cue word "happy" may signal a discrepancy between the individual's desired mood state and their current depressed state, leading to ruminative thought processes and subsequently the retrieval of an overgeneral memory. Furthermore, as Williams et al. also noted, cue word valence does not necessarily dictate the valence of the subsequently recalled memory. For instance, a seemingly neutral word like "summer" may remind one participant of a fun vacation, whereas it may remind another individual of a break-up that occurred during that time of year. Therefore it plausible that findings may emerge across valence conditions, both within the overgeneral memory literature and among other areas of cognitive bias research.

Watkins et al.'s (2000) work served to demonstrate a relationship between rumination and overgeneral memory, whereas the studies that followed furthered this investigation by attempting to isolate the specific characteristics of ruminative thought that were responsible for this link. Watkins and Teasdale (2001) suggested that rumination's analytic nature may be leading to this relationship, whereas Barnard et al. (2006) pointed to the repetitive and self-focused aspects of this thought process. Finally, Raes et al. (2008) isolated rumination's abstract nature as a potential cause. It is possible that all of these components working in unison may best explain the relationship between rumination and overgeneral memory. However, designs that pit differing aspects against each other, such as Watkins and Teasdale's, will provide the most conclusive information as to what specific attributes of ruminative thought are most responsible for observed memory biases. The above findings are also important in their demonstration of a bidirectional causal relationship between rumination and overgeneral memory, in that evidence exists for both the ability to decrease levels of overgeneral memory by reducing already present rumination, as well as the ability to increase overgeneral memory by inducing rumination within nondysphoric samples.

Interesting findings from a clinical standpoint also have demonstrated that modifications in memory specificity may, in turn, influence rumination, suggesting that a reciprocal relationship may exist between these vulnerability factors. Ekkers, Korrelboom, Huijbrechts, Smits, Cuijpers, & van der Gaag (2011) tested a Competitive Memory Training (COMET) protocol within a sample of ninety-three older adults with clinical depression, in which participants were trained to recall more specific memories

of times when they confronted difficult situations without engaging in rumination. Although this training placed a greater emphasis on themes of acceptance and indifference towards difficult circumstances, their protocol did involve aspects of retrieving specific memories counter to participants' typical ruminative tendencies. COMET combined with treatment as usual was found to produce greater improvements in depressive symptoms and rumination compared to treatment as usual alone. Promising results also have emerged from Raes, Williams, and Hermans' (2009) Memory Specificity Training protocol (MEST), which is primarily intended to educate and train participants in the retrieval of more specific memories in order to decrease rumination and depression. In a preliminary study of ten inpatients engaged in a four-week training, MEST was demonstrated to increase memory specificity on the AMT, improve problem-solving abilities and decrease rumination scores. Importantly, these effects remained when controlling for observed changes in depressive symptoms, suggesting that improvements in mood were not primarily responsible for these findings. A follow-up study demonstrated MEST to be more effective than a no-treatment control group in increasing memory specificity and decreasing depressive symptoms at follow-up within a sample of depressed adolescents, although changes in rumination were not measured (Neshat-Doost et al., 2013); therefore, future examinations of this novel protocol are needed. In sum, these findings from the treatment literature provide evidence that the relationship between rumination and overgeneral memory may indeed be reciprocal, and that intervening from either direction may serve to improve depressive symptoms.

3. Synthesis and Future Directions

Together, the body of research presented in this review suggests the presence of an important relationship between ruminative thought and memory biases as contributors to depression. The majority of the reviewed studies demonstrated that ruminative thought may foster increased encoding and retrieval of negative memories, as well as increased overgeneral memory production. However, findings from the intervention literature indicated that increased memory specificity may reduce levels of rumination, providing support for a bidirectional relationship between these vulnerability factors. Results emphasize the importance of examining the interplay between cognitive vulnerabilities for depression, as it is very likely that many of these factors are interrelated and may confer added risk for the disorder when influencing or interacting with each other.

Whereas many of the reviewed studies sought to test a causal relationship between ruminative thought and memory bias, few of these studies employed prospective designs to test whether these risk factors were predictive of future depressive symptoms or diagnoses, with the exception of the aforementioned intervention studies. Given the hypothesis that rumination and memory biases are vulnerability factors for depression, it is particularly important to examine this relationship in its entirety, such as by testing whether the effect of rumination in predicting future depressive symptoms is mediated by negative or overgeneral memory biases. It is also possible that rumination and memory biases may interact in predicting depressive symptoms, as has been demonstrated in a recent prospective study within a community sample of adolescents (Hamlat, Connolly,

Hamilton, Stange, Abramson, & Alloy, 2014). Furthermore, these designs are necessary in order to better understand whether rumination and memory biases may *precede* depressive disorders. Indeed, many of the studies discussed within this review only found rumination to foster memory biases among already depressed or dysphoric samples. Therefore, it is yet unclear whether these observed effects are only present when individuals are currently in a dysphoric state, or whether rumination and memory biases may serve as risk factors for the initial development of depression. Overall, there is a great need for longitudinal designs in order to best assess the role that these vulnerability factors play in contributing to depressive symptoms over time.

An additional issue is whether the current paradigms used to examine biases in the encoding and retrieval of memories are adequately measuring their purported constructs of interest. Indeed, as mentioned earlier, all of the reviewed studies that examined the encoding of negative information utilized retrieval as their primary outcome, making it difficult to assess to what extent processes at the encoding stage were actually responsible for observed effects. Furthermore, these studies examined the encoding of single words; although this design allows for the standardization of experimental stimuli, the personal relevance of these words likely varied between participants, and the memorization of a word list is not particularly comparable to the encoding of experiences encountered during an individual's daily life. In contrast, the reviewed studies of autobiographical memory retrieval sacrificed standardization by allowing participants to freely recall any memories from their past; however, these studies are therefore restricted by their inability to analyze processes occurring at the

encoding stage, as the recalled memories are referencing content experienced perhaps many years prior, outside of the laboratory setting.

Given these weaknesses, it is possible that alternative experimental designs may more effectively examine the processes of rumination and memory bias in depression in a standardized, yet personally relevant, fashion. Ecological momentary assessment (EMA) may prove particularly useful within this field, in allowing participants to report on their experiences of events during their daily lives outside of the laboratory, as well as their levels of rumination following the occurrence of these events, using modes of data collection such as smartphones, tablets, or online surveys. Participants' memory for these events could subsequently be tested within the laboratory, assessing for patterns such as negative or overgeneral recall biases among individuals engaging in event-related rumination (e.g., Connolly & Alloy, in progress). Alternatively, participants could undergo a standardized stressful or negative experience in the laboratory, after which their event recall could be examined. This design has been employed within the social anxiety literature in order to examine the role of post-event processing on negative memory biases. Post-event processing refers to the rehashing of a social event after its occurrence, a behavior that has been strongly linked to social anxiety disorder and that has striking parallels to the construct of depressive rumination with regards to its repetitive, self-focused, and maladaptive qualities (Brozovich & Heimberg, 2008; McEvoy, Mahoney, & Moulds, 2010). Studies in which participants engaged in a stressful in-lab task, such as delivering a speech, and later were asked to recall aspects of the event, such as critiques they received from confederate judges, found varying degrees

of interrelationships between social anxiety, post-event processing following the stressful event, and subsequent negative memory biases (Cody & Teachman, 2010; Edwards, Rapee, & Franklin, 2003; Mellings & Alden, 2000). These studies provide encouraging evidence that such paradigms may be successful, and more ecologically valid, measures of the relationship between ruminative thought and the encoding and retrieval of negative, personally-relevant memories. Similar experimental designs are worthy of testing within a depression framework.

Future work would benefit from investigating the specific mechanisms that may be responsible for the deleterious effects of rumination and memory biases. As discussed earlier, studies of rumination have implicated a variety of its components, including its self-focused and repetitive qualities (Barnard et al., 2006; Pyszczynski et al., 1989), its analytic nature (Watkins & Teasdale, 2001), and its degree of abstraction (Raes et al., 2008). As Watkins (2015) hypothesized, it is possible that abstraction may represent an underlying, "unifying factor" that may lead to both ruminative thought and overgeneral memory production (p. 213). Studies employing novel measures intended to tap into wider-reaching constructs such as abstract thinking could help in pinpointing the particular mechanisms that may be most responsible for the manifestation of maladaptive cognitive styles, which could lead to more effective and targeted intervention strategies. Intriguingly, Watkins and colleagues, motivated by this abstraction hypothesis, have examined the efficacy of a concreteness training intervention in which participants are instructed to engage in more concrete, specific, and detail-oriented thought when faced with difficult situations. This training protocol was found to be more effective at reducing

rumination and depressive symptoms compared to no treatment, a sham training control group, or treatment as usual (Watkins, Baeyens, & Read, 2009; Watkins et al., 2012). Although this protocol provided greater reductions in rumination compared to relaxation training within a clinically depressed sample, change in depressive symptoms was not significantly different between groups (Watkins et al., 2012).

As suggested by Joormann's impaired cognitive control account (2010) and Williams' CaRFAX model (2007), inhibition impairment may be another important underlying mechanism that contributes to both rumination and memory biases. Inhibitory deficits may make it difficult for individuals to detach from the self-focused, negatively-valenced information that comprises ruminative thought, which could lead to deepened encoding of this information and increased retrieval of negative memories. Individuals with such deficits also may display similar difficulties attempting to navigate away from abstracted self-perceptions when instructed to retrieve a specific memory, resulting in overgeneral memory biases. The literature would benefit greatly from integrated examinations of the ways in which inhibitory deficits may underlie rumination, memory biases, and depressive symptoms. Such research would be further strengthened by attempts to link inhibitory impairments, as measured by behavioral tasks of executive functioning, with corresponding patterns of neural activity in order to identify common biomarkers that may underlie these cognitive risk factors for depression.

In addition, future research should examine the specific ways in which rumination and negative memory biases may, in turn, contribute to increases in depressive

symptoms. For instance, both of these vulnerabilities have been linked to impairments in problem-solving abilities (Lyubomirsky & Nolen-Hoeksema, 1995; Williams et al., 2007). Furthermore, Raes et al.'s Memory Specificity Training (2009) was shown to increase specific memory generation as well as decrease levels of rumination and depression and increase problem-solving skills, suggesting a potential interplay between all of these factors. Studies that examine whether impaired problem-solving mediates demonstrated relationships between rumination, memory biases, and depressive symptoms would help in better understanding the negative impact conferred by these cognitive risk factors and would further clarify necessary points of intervention.

Current findings indicate that rumination and memory biases are important and interrelated cognitive risk factors for depression. Although research has demonstrated relationships between ruminative thought and the encoding and retrieval of negative information, as well as the production of overgeneral memories, many questions regarding the nature of these links remain unanswered. Future investigation is needed in order to best characterize the mechanisms that underlie these relationships and that eventually lead to increases in depression. A more elaborated understanding of the interplay between these cognitive processes will allow for specific and targeted interventions aimed to reduce the burden of this debilitating disorder.

BIBLIOGRAPHY

Abramson, L.Y., Alloy, L.B., Hankin, B.L., et al. Cognitive vulnerability-stress models of depression in a self-regulatory and psychobiological context. In: Gotlib, I.H., Hammen, C.L., eds. *Handbook of Depression.* New York: Guilford Press; 2002.

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., . . . Lapkin, J. B. (2000). The Temple-Wisconsin cognitive vulnerability to depression project: Lifetime history of axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 109*(3), 403-418.

Alloy, L. B., Abramson, L. Y., Murray, L. A., Whitehouse, W. G., & Hogan, M. E. (1997). Self-referent information-processing in individuals at high and low cognitive risk for depression. *Cognition & Emotion,* 11, 539–568.

Alloy, L. B., Bender, R. E., Whitehouse, W. G., Wagner, C. A., Liu, R. T., Grant, D. A.,... Abramson, L. Y. (2012). High behavioral approach system (BAS) sensitivity, reward responsiveness, and goal-striving predict first onset of bipolar spectrum disorders: A prospective behavioral high-risk design. *Journal of Abnormal Psychology*, 121, 339 –351.

Alloy, L.B., & Clements, C.M. (1992). Illusion of control: invulnerability to negative affect and depressive symptoms after laboratory and natural stressors. *Journal of Abnormal Psychology*. 1992, *101(*2), 234-45.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Armey, M. F., Schatten, H. T., Haradhvala, N., & Miller, I. W. (2015). Ecological momentary assessment (EMA) of depression-related phenomena. *Current Opinion in Psychology*, 4, 21-25.

Barnard, P. J., Watkins, E. R., & Ramponi, C. (2006). Reducing specificity of autobiographical memory in nonclinical participants: The role of rumination and schematic models. *Cognition & Emotion*, 20(3), 328-350.

Bartlett, F. C. S. (1932). Remembering: A study in experimental and social psychology. Cambridge, Eng: The University Press.

Beck, A.T., Steer, R.A., Brown, G.K. (1996). *Manual for the Beck Depression Inventory-II*. San Antonio, TX: Psychological Corporation.

Beck, A.T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961) An inventory for measuring depression. *Archives of General Psychiatry,* 4, 561-571.

Birnbaum, H. G., Kessler, R. C., Kelley, D., Ben-Hamadi, R., Joish, V. N., & Greenberg, P. E. (2010). Employer burden of mild, moderate, and severe major depressive disorder: Mental health services utilization and costs, and work performance. *Depression and Anxiety*, *27(1)*, 78-89.

Bradley, B. P., & Mathews, A. (1988). Memory bias in recovered clinical depressives. *Cognition and Emotion*, 2, 235-245.

Brewin, C. R., Reynolds, M., & Tata, P. (1999). Autobiographical memory processes and the course of depression. *Journal of Abnormal Psychology*, 108(3), 511-517.

Brozovich, F., & Heimberg, R. G. (2008). An analysis of post-event processing in social

anxiety disorder. *Clinical Psychology Review,* 28(6), 891-903.

Christianson, S.A., & Loftus, E. (1987). Memory for traumatic events. *Applied Cognitive Psychology*, 1, 225–239.

Cody, M. W., & Teachman, B. A. (2010). Post-event processing and memory bias for performance feedback in social anxiety. *Journal of Anxiety Disorders*, 24(5), 468-479.

Connolly, S.L, Abramson, L.Y., & Alloy, L.B. (2016). The relationship between information processing biases, cognitive vulnerabilities, and depressive symptoms in adolescents: Evidence from a self-referent encoding task. *Cognition & Emotion*, 30(3), 550-560.

Connolly & Alloy (under review). Rumination interacts with life stress to predict depressive symptoms: An ecological momentary assessment study.

Conway, M., Csank, P.A.R., Holm, S.L., & Blake, C.K. (2000). On assessing individual differences in rumination on sadness. *Journal of Personality Assessment*, 75, 404-425.

Conway, M. A., & Pleydell-Pearce, C. W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review*, 107, 261–288.

Dalgleish, T., Spinks, H., Golden, A. M., & du Toit, P. (2004). Processing of emotional information in seasonal depression across different cognitive measures. *Journal of Abnormal Psychology*, 113, 116-126.

Dalgleish, T., & Watts, F. N. (1990). Biases of attention and memory in disorders of anxiety and depression. *Clinical Psychology Review*, 10(5), 589-604.

Delis, D.C., Kramer, J.H., Kaplan, E., & Ober, B.A. (2000). California Verbal Learning Test: Second Edition. San Antonio, TX: Psychological Corporation.

Derry, P. A, & Kuiper, N. A. (1981). Schematic processing and self-reference in clinical depression. *Journal of Abnormal Psychology*, *90*(4), 286–297.

Dozois, D.J., Dobson, K.S., Ahnberg, J.L. (1998). A psychometric evaluation of the Beck Depression Inventory-II. *Psychological Assessment*, 10(2):83-89.

Edwards, S. L., Rapee, R. M., & Franklin, J. (2003). Postevent rumination and recall bias for a social performance event in high and low socially anxious individuals. *Cognitive Therapy and Research, 27*(6), 603-617.

Ekkers, W., Korrelboom, K., Huijbrechts, I., Smits, N., Cuijpers, P., & van der Gaag, M. (2011). Competitive memory training for treating depression and rumination in depressed older adults: A randomized controlled trial. *Behaviour Research and Therapy*, 49(10), 588-596.

Endicott, J., & Spitzer, R.A. (1978). A diagnostic interview: The Schedule for Affective Disorders and Schizophrenia. *Archives of General Psychiatry*, *35*, 837-844.

Everaert, J., Koster, E. H. W., & Derakshan, N. (2012). The combined cognitive bias hypothesis in depression. *Clinical Psychology Review, 32*(5), 413–424.

Feldner, M. T., Leen-Feldner, E. W., Zvolensky, M. J., & Lejuez, C. W. (2006). Examining the association between rumination, negative affectivity, and negative affect induced by a paced auditory serial addition task. *Journal of Behavior Therapy and Experimental Psychiatry*, 37(3), 171-187.

Genet, J. J., & Siemer, M. (2012). Rumination moderates the effects of daily events on negative mood: results from a diary study. *Emotion (Washington, D.C.)*, *12*(6), 1329–39.

Hamlat, E.J., Connolly, S.L., Hamilton, J.L., Stange, J.P., Abramson, L.Y., & Alloy, L.B. (2014). Rumination and overgeneral autobiographical memory in adolescents: An integration of cognitive vulnerabilities to depression. *Journal of Youth and Adolescence, 44(4),* 806-818.

Hammen, C. (2005). Stress and depression. *Annual Review of Clinical Psychology*, 1(1), 293-319.

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York: The Guilford Press.

Hertel, P., Amaris, M., Cottle, J., & Vrijsen, R. (in press). Cognitive bias modification: Retrieval practice to simulate and oppose ruminative memory biases. *Clinical Psychological Science.*

Hertel, P. T., & El-Messidi, L. (2006). Am I blue? Depressed mood and the consequences of self-focus for the interpretation and recall of ambiguous words. *Behavior Therapy*, 37(3), 259.

Hertel, P. T., & Gerstle, M. (2003). Depressive deficits in forgetting. *Psychological Science*, 14(6), 573-578.

Hertel, P. T., & Mathews, A. (2011). Cognitive bias modification: Past perspectives, current findings, and future applications. *Perspectives on Psychological Science*, 6(6), 521-536.

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and Interpretations in Social Phobia: Support for the Combined Cognitive Biases Hypothesis. *Behavior Therapy*, *37*, 223–236.

Hoorelbeke, K., Koster, E., Demeyer, I., Loeys, T., & Vanderhasselt, M.A. (2016). Effects of cognitive control training on the dynamics of (mal)adaptive emotion regulation in daily life. *Emotion.* Advance online publication.

Johnson, H. M. (1994). Processes of successful intentional forgetting. *Psychological Bulletin*, 116, 274–292.

Joormann, J. (2010). Cognitive Inhibition and Emotion Regulation in Depression. *Current Directions in Psychological Science*, *19*, 161–166.

Joormann, J., Hertel, P., LeMoult, J., & Gotlib, I.H. (2009). Training intentional forgetting of negative material in depression. *Journal of Abnormal Psychology*, 118, 34–43.

Joormann, J. & Tran, T. (2009). Rumination and intentional forgetting of emotional material. *Cognition & Emotion*, 23(6), 1233-1246.

Joormann, J., Yoon, K. L., & Zetsche, U. (2007). Cognitive inhibition in depression. *Applied and Preventive Psychology*, *12*, 128–139.

Just, N., & Alloy, L. B. (1997). The response styles theory of depression: Tests and an extension of the theory. *Journal of Abnormal Psychology*, *106(2)*, 221-229.

Kessler, R. C., Merikangas, K. R., & Wang, P. S. (2007). Prevalence, comorbidity, and service utilization for mood disorders in the United States at the beginning of the twenty-first century. *Annual Review of Clinical Psychology*, 3137-158.

Kuo, J. R., Edge, I. G., Ramel, W., Edge, M. D., Drabant, E. M., Dayton, W. M., & Gross, J. J. (2012). Trait rumination is associated with enhanced recollection of negative words. *Cognitive Therapy and Research*, 36(6), 722-730.

Lyubomirsky, S., Caldwell, N. D., & Nolen-Hoeksema, S. (1998). Effects of ruminative and distracting responses to depressed mood on retrieval of autobiographical memories. *Journal of Personality and Social Psychology*, 75(1), 166-177.

Lyubomirsky, S., & Nolen-Hoeksema, S. (1993). Self-perpetuating properties of dysphoric rumination. *Journal of Personality and Social Psychology*, 65(2), 339-349.

Lyubomirsky, S., & Nolen-Hoeksema, S. (1995). Effects of self-focused rumination on negative thinking and interpersonal problem-solving. *Journal of Personality and Social Psychology, 69*, 176-190.

Marchetti, I., Koster, E., & De Raedt, R. (2013). Rest-related dynamics of risk and protective factors for depression: A behavioral study. *Clinical Psychological Science, 1*(4), 443-451.

Matt, G. E., Vázquez, C., & Campbell, W. (1992). Mood-congruent recall of affectively toned stimuli: A meta-analytic review. *Clinical Psychology Review*, 12(2), 227-255.

McEvoy, P. M., Mahoney, A. E. J., & Moulds, M. L. (2010). Are worry, rumination, and post-event processing one and the same? Development of the repetitive thinking questionnaire. *Journal of Anxiety Disorders*, 24(5), 509-519.

McFarland, C., & Buehler, R. (1998). The impact of negative affect on autobiographical memory: The role of self-focused attention to moods. *Journal of Personality and Social Psychology*, 75(6), 1424-1440.

Mellings, T. M. B., & Alden, L. E. (2000). Cognitive processes in social anxiety : the effects of self- focus, rumination and anticipatory processing. *Behaviour Research and Therapy, 38*, 243–257.

Moberly, N. J., & Watkins, E. R. (2008a). Ruminative self-focus, negative life events, and negative affect. *Behaviour Research and Therapy*, *46*(9), 1034-1039.

Moberly, N. J., & Watkins, E. R. (2008b). Ruminative self-focus and negative affect: an experience sampling study. *Journal of Abnormal Psychology*, *117*(2), 314–23.

Mor, N., Marchetti, I., & Koster, E. H. W. (2013). The Momentary Ruminative Self-focus Inventory (MRSI): Validation and Psychometric Evaluation. *Manuscript submitted for publication*.

Moulds, M. L., Kandris, E., & Williams, A. D. (2007). The impact of rumination on memory for self-referent material. *Memory*, *15*(8), 814–21.

Neshat-Doost, H. T., Dalgleish, T., Yule, W., Kalantari, M., Ahmadi, S. J., Dyregrov, A., & Jobson, L. (2013). Enhancing autobiographical memory specificity through cognitive training: An intervention for depression translated from basic science. *Clinical Psychological Science*, 1(1), 84-92.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100(4)*, 569-582.

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(1), 115-121.

Nolen-Hoeksema, S. & Morrow, J. (1993). Effects of rumination and distraction on naturally occurring depressed mood. *Cognition and Emotion*, 7, 561–570.

Park, R. J., Goodyer, I. M., & Teasdale, J. D. (2004). Effects of induced rumination and distraction on mood and overgeneral autobiographical memory in adolescent major depressive disorder and controls. *Journal of Child Psychology and Psychiatry*, 45(5), 996-1006.

Power, M. J., Dalgleish, T., Claudio, V., Tata, P., & Kentish, J. (2000). The directed forgetting task: Application to emotionally valent material. *Journal of Affective Disorders*, 57, 147–157.

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2003). Simple intercepts, simple slopes, and regions of significance in HLM 2-way interactions. Retrieved from http://www.quantpsy.org/interact/hlm2.htm.

Pyszczynski, T., Hamilton, J. C., Herring, F. H., & Greenberg, J. (1989). Depression, self-focused attention, and the negative memory bias. *Journal of Personality and Social Psychology*, *57*(2), 351–357.

Radloff, L. S. (1977). The CES-D scale. A self-report depression scale for research in the general population. *Journal of Applied Psychological Measurement*, 1, 385-401.

Raes, F., Hermans, D., Williams, J. M. G., Beyers, W., Brunfaut, E., & Eelen, P. (2006). Reduced autobiographical memory specificity and rumination in predicting the

course of depression. *Journal of Abnormal Psychology*, 115, 699–704.

Raes, F., Hermans, D., Williams, J. M. G., & Eelen, P. (2007). A sentence completion procedure as an alternative to the Autobiographical Memory Test for assessing overgeneral memory in nonclinical populations. *Memory*, 15, 495–507.

Raes, F., Watkins, E. R., Williams, J. M. G., & Hermans, D. (2008). Non-ruminative processing reduces overgeneral autobiographical memory retrieval in students. *Behaviour Research and Therapy*, 46(6), 748-756.

Raes, F., Williams, J. M. G., & Hermans, D. (2009). Reducing cognitive vulnerability to depression: A preliminary investigation of MEmory specificity training (MEST) in inpatients with depressive symptomatology. *Journal of Behavior Therapy and Experimental Psychiatry*, 40(1), 24-38.

Ramponi, C., Barnard, P., & Nimmo-Smith, I. (2004). Recollection deficits in dysphoric mood: An effect of schematic models and executive mode? *Memory,* 12(5), 655-670.

Raudenbush S. W., Bryk A. S., Cheong Y. F., Congdon R. T., du Toit M. (2011). HLM 7: Hierarchical linear and nonlinear modeling. Chicago, IL: Scientific Software I nternational.

Roberts, J. E., Gilboa, E., & Gotlib, I. H. (1998). Ruminative response style and vulnerability to episodes of dysphoria: Gender, neuroticism, and episode duration. *Cognitive Therapy and Research*, *22(4)*, 401-423.

Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research, 27*(3), 275-291.

Rogers, T. B., Kuiper, N. a, & Kirker, W. S. (1977). Self-reference and the encoding of personal information. *Journal of Personality and Social Psychology*, *35*(9), 677–688.

Rubin, D. C., & Kozin, M. (1984). Vivid memories. *Cognition*, 16(1), 81-95.

Ruscio, A. M., Gentes, E. L., Jones, J. D., Hallion, L. S., Coleman, E. S., & Swendsen, J. (2015). Rumination predicts heightened responding to stressful life events in major depressive disorder and generalized anxiety disorder. *Journal of Abnormal Psychology*, 124(1), 17-26.

Safford, S.M., Alloy, L.B., Abramson, L.Y., & Crossfield, A.G. (2007). Negative cognitive style as a predictor of negative life events in depression-prone individuals: a test of the stress generation hypothesis. *Journal of Affective Disorders*, *99*(1-3):147-54.

Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological Momentary Assessment. *Annual Review of Clinical Psychology*, *4*(1), 1–32.

Siegle, G. J., Moore, P. M., & Thase, M. E. (2004). Rumination: One construct, many features in healthy individuals, depressed individuals, and individuals with lupus. *Cognitive Therapy and Research, 28*(5), 645-668.

Spasojević, J., & Alloy, L. B. (2002). Who becomes a depressive ruminator? Developmental antecedents of ruminative response style. *Journal of Cognitive Psychotherapy, 16(4),* 405-419.

Stone, A. A., Schwartz, J. E., Neale, J. M., Shiffman, S., Marco, C. A., Hickcox, M., . . . Cruise, L. J. (1998). A comparison of coping assessed by ecological momentary assessment and retrospective recall. *Journal of Personality and Social Psychology, 74*(6), 1670-1680.

Storch, E.A., Roberti, J.W., Roth, D.A. (2004). Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory-Second Edition in a sample of college students. *Depression and Anxiety, 19*(3), 187-9.

Takano, K., & Tanno, Y. (2010). Concreteness of thinking and self-focus. *Consciousness and Cognition*, 19(1), 419-425.

Takano, K., & Tanno, Y. (2011). Diurnal variation in rumination. *Emotion, 11*(5), 1046-1058.

Trapnell, P. D., & Campbell, J. D. (1999). Private self-consciousness and the five-factor model of personality: Distinguishing rumination from reflection. *Journal of Personality and Social Psychology*, 76(2), 284-304.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination Reconsidered: A Psychometric Analysis. *Cognitive Therapy and Research*, 27(3), 247-259.

Walhovd, K. B., Fjell, A. M., Dale, A. M., Fischl, B., Quinn, B. T., Makris, N., . . . Reinvang, I. (2006). Regional cortical thickness matters in recall after months more than minutes. *NeuroImage*, 31(3), 1343-1351.

Watkins, E. (2015). Overgeneral autobiographical memories and their relationship to rumination. In L.A. Watson & D. Berntsen (eds.), *Clinical Perspectives on Autobiographical Memory (199-220).* Cambridge: Cambridge University Press.

Watkins, E. R., Baeyens, C. B., & Read, R. (2009). Concreteness training reduces dysphoria: Proof-of-principle for repeated cognitive bias modification in depression. *Journal of Abnormal Psychology*, 118(1), 55-64.

Watkins, E., Moberly, N. J., & Moulds, M. L. (2008). Processing mode causally influences emotional reactivity: Distinct effects of abstract versus concrete construal on emotional response. *Emotion*, 8(3), 364-378.

Watkins, E. R., Taylor, R. S., Byng, R., Baeyens, C., Read, R., Pearson, K., & Watson, L. (2012). Guided self-help concreteness training as an intervention for major depression in primary care: A phase II randomized controlled trial. *Psychological Medicine*, 42(7), 1359-1371.

Watkins, E., & Teasdale, J. D. (2001). Rumination and overgeneral memory in depression: Effects of self-focus and analytic thinking. *Journal of Abnormal Psychology*, 110(2), 353-357.

Watkins, E., & Teasdale, J. D. (2004). Adaptive and maladaptive self-focus in depression. *Journal of Affective Disorders*, 82(1), 1-8.

Watkins, E., Teasdale, J. D., & Williams, R. M. (2000). Decentering and distraction reduce overgeneral autobiographical memory in depression. *Psychological Medicine*, 30, 911-920.

Wenze, S. J., & Miller, I. W. (2010). Use of ecological momentary assessment in mood disorders research. *Clinical Psychology Review*, *30*(6), 794–804.

Williams, J. M. G., Barnhofer, T., Crane, C., Hermans, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. Psychological Bulletin, 133(1), 122-148.

Williams, J. M., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology*, 95, 144–149.

Wisco, B. E., & Nolen-Hoeksema, S. (2008). Ruminative response style. In K. S. Dobson, D. A. Dozois (Eds.) , *Risk factors in depression* (pp. 221-236). San Diego, CA US: Elsevicader Aemic Press.

Wong, A. C. M., & Moulds, M. L. (2008). Depressive Rumination and Directed Forgetting: An Examination of Encoding Style. *Cognitive Therapy and Research*, *32*(1), 1–10.

Yonelinas, A. P. (2002). The nature of recollection and familiarity: A review of 30 years of research. *Journal of Memory and Language*, 46, 441–517.