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

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Author manuscript

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## Abstract

The current research utilized ecological momentary assessment (EMA) methodology to test the hypotheses that: 1) engaging in greater rumination following stress (stress-reactive rumination; SRR) would lead to improved stressor recall, and 2) this improved memory for stress would predict increases in depressive symptoms. One hundred twenty-one participants received smartphone alerts in which they reported on their experience of negative life events (NLEs) as well as SRR and depressed mood after event occurrence. NLEs followed by increased SRR were more likely to be recalled two weeks later. Furthermore, individuals who endorsed and recalled more stressors displayed increased depressive symptoms. Contrary to hypotheses, no evidence was found for a mediational effect in which SRR predicted depressive symptoms and was mediated by memory for NLEs. Current findings demonstrate a relationship between rumination following stress and the subsequent recall of those stressors, and support the role of negative event recall as a vulnerability factor for depression.

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; Spasojevi & 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). Increased recall of negative information is a well-established correlate of major depression (see Matt, Vázquez, & Campbell, 1992, for a review) that also has been associated with depressive symptoms in

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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 the potential role of negative memory processes as an important vulnerability factor for depression.

Drawing from the field of cognitive psychology, 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 therefore would fuel the recall of negative information and perpetuate depressed mood over time. Indeed, it has long been demonstrated within the memory literature that repeated exposure to a stimulus increases its likelihood of being recalled (Hintzman & Block, 1971). Therefore, it would follow that engaging in ruminative thought, in which negative information about oneself is repetitively processed, would improve subsequent memory for this information over time.

Several studies have investigated whether rumination may lead to negative information being 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 subsequently 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 processes for negative information as factors influencing depressed mood. These findings invite further inquiry in order to address methodological limitations in

the abovementioned studies. Importantly, longitudinal approaches were not used to test whether these risk factors were predictive of future depressive symptoms or diagnoses, and whether their effects may be exacerbated by the experience of increased stress, in line with diathesis-stress models of depression (Abramson et al., 2002). Indeed, several of the studies only found rumination to foster increases in recall of negative information 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 stress, rumination, and memory may serve as a risk factor for increases in depressive symptoms and episodes over time. 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, 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 recall abilities. 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 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 ecological 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, trait rumination, and memory ability for neutral material, 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 it is possible that 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 would be amplified by the experience of stress. This hypothesis is 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.

The present study is the first to our knowledge to assess the role of rumination directly after the occurrence of naturalistic stressors 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 improves retrieval of this negative

information. The current research draws from theories of cognition and memory to better understand the specific mechanisms through which rumination contributes to depressed mood.

## 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  $\pm 5.21$ , 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, conducting supervised live interviews, and frequent case conferencing in order to achieve consensus regarding diagnostic impressions. SADS-L interviews conducted by investigators trained using this protocol display high interrater reliability with kappas >.80 (Alloy et al., 2000). The current research operationalized history of depression as meeting criteria for at least one major depressive episode within the participants' lifetime.

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 4 (*Extreme*)<sup>1</sup>. 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 LEI was administered by student investigators who received intensive training in interview administration, including conducting supervised live interviews and frequent case conferencing in order to achieve consensus regarding life event classifications. 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.

<sup>&</sup>lt;sup>1</sup>A priori objective intensity ratings were derived from criteria set forth within the Bedford College Life Events and Difficulties Schedule (LEDS; Brown & Harris, 1978), adapted by Scott Monroe within the Pittsburgh Life Events and Difficulties Schedule (Monroe & Roberts, 1990), upon which the current LES was based. Scores were based on the degree of threat and unpleasantness imposed by a given event category.

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**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 5item version of the SRRS (Robinson & Alloy, 2003) created to measure participants' cognitive responses directly after the experience of stressors 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 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. Of note, the exact date of event occurrence did not have to be correct for an event to be considered accurately recalled. 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:

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

Recall<sub>*ti*</sub> 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 SRRS-S over and above control variables, analyses were first run excluding Level 1 SRRS-S and Level 2 aggregated SRRS-S from the model.

**Prospective prediction of depressive symptoms**—The presence of a hypothesized mediational relationship was assessed using bootstrapping within the PROCESS macro (Hayes, 2013) to test for a significant indirect effect of proportional recall of NLEs in predicting the relationship between aggregated SRRS-S score 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 was used to probe the interaction between number of NLEs endorsed and proportional recall in predicting T3 depressive symptoms. 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. 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  $\pm$  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;  $M_{days after T1} = 14.41 \pm 1.13$ ), and 120 participants completed their final two-week follow-up online questionnaires (T3;  $M_{days after T2} = 16.03 \pm 5.25$ ).

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  $9.45 \pm 5.51$  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 (e.g., migraine headache, difficulty paying bills, ongoing relationship problems, severe illness or hospitalization of family member). Of all NLEs, 43.5% were recalled at T2 (35.1% in uncued condition, n = 401; 8.4% in cued condition, n = 96). Participants recalled an average of 48.5%  $\pm$  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 (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. These rates are consistent with results of previous rigorous longitudinal studies, assessing the incidence of a major depressive episode up to age 30 within the Oregon Adolescent Depression Project (51% of sample; Rohde, Lewinsohn, Klein, Seeley, & Gau, 2012) and up to age 32 in the Dunedin Study (41% of sample; Moffitt et al., 2010). The EMA measures of SRR and depressive symptoms 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. Additional descriptive statistics are provided in Table 1.

#### Prediction of Event Recall

Before including SRRS-S 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), 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 = . 01; Table 2), such that a ten-point increase in SRR score improved the odds of event recall by  $20\%^2$ ,<sup>3</sup>. Event subjective intensity lost significance as a predictor of recall when SRR was added to the model; objective intensity and chronicity remained significant predictors. SRRS-S score also significantly predicted event recall when no variables were controlled for at Level 1 or Level 2 (B = .03, OR = 1.03, p = .001). Follow-up analyses revealed no significant interactions between SRRS-S score and 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**

Mediation analyses found a nonsignificant total effect of aggregated SRRS-score on T3 BDI-II score (effect = -.08, p = .12) as well as a nonsignificant direct effect (effect = -.09, p = .10). The indirect effect on T3 BDI-II through proportional recall of NLEs also was nonsignificant (effect = .006, 95% CI [-.004, .04]). Therefore, the hypothesis that negative event recall 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 = .02,  $R^2$  change = .013; Table 3, Figure 1). The effect was significant when proportional recall was high (90<sup>th</sup> percentile), 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 (90<sup>th</sup> percentile), in that those who recalled more of their NLEs displayed greater increases in BDI-II score 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.

<sup>&</sup>lt;sup>2</sup>Current analyses collapsed the recall outcome variable across the cued and uncued conditions. When only predicting to memories recalled in the uncued condition, SRR was marginally significant (B = .01, OR = 1.01, p = .087).

<sup>&</sup>lt;sup>3</sup>Participants ruminated significantly more about dependent stressors (events that in part are related to characteristics or behavior of the individual, e.g. a fight with significant other) than independent stressors (events to which an individual would not be thought to contribute, e.g. a snowstorm); t(1141) = -10.60, p < .001. SRR level remained a significant predictor of event recall when event dependence rating was controlled for (B = .02, OR = 1.02, p < .05). There were no significant differences in rumination when comparing interpersonal to non-interpersonal stressors (t(1142) = 1.64, p = .15).

## 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 integrates cognitive and memory theory to provide a fine-grained analysis of the hypothesis that repetitive, ruminative thought strengthens the subsequent retrieval of negative information, thereby fueling negative recall processes 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. This effect became nonsignificant when SRR was added to the model, indicating that the degree to which one ruminates about a stressor may be a stronger predictor of its recall than its perceived severity. However, it must be noted that the SRR and subjective intensity variables were strongly correlated (r = .62); therefore, the loss of significance of the subjective intensity variable may be due to shared variance. 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 direct evidence of a relationship between the degree of rumination engaged in following the experience of daily naturalistic stress and the subsequent recall of that information. The current results support theoretical accounts suggesting that repetitive, ruminative thought contributes to long-term increases in memory retrieval of negative material (Joormann et al., 2007; Joormann, 2010). It is possible that rumination increases retrieval by strengthening the initial encoding and maintenance of negative information. However, it must be noted that the current design was unable to explicitly test this hypothesis, and future fine-grained examinations of the encoding of negative memories are therefore necessary. Furthermore, results complement the broader memory literature demonstrating that repetition of stimuli increases the likelihood of subsequent recall (Hintzman & Block, 1971). 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.

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 lives 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 a stressor, 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 from T2 to T3. Therefore, no mediational relationship was found such that NLE recall explained the effects of SRR in increasing follow-up depression scores, precluding interpretations that recall of naturalistic stress is a mechanism through which rumination exerts its deleterious effects on mood. However, in line with hypotheses, 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 in a clinically depressed 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 Connolly & Alloy, in press).

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. Baseline trait measures of stress-reactive and brooding rumination also did not significantly predict recall of NLEs, in opposition to previous research demonstrating relationships between trait rumination and subsequent memory for negative information. However, it is important to emphasize the substantial methodological differences within the current design compared to previous work, including its use of ecological momentary assessment to measure the recall of personally relevant, generally mild negative life events occurring outside of a structured laboratory setting during a prescribed one-week period. As such, previous findings of relationships between trait rumination and memory for pre-set lists of negative words, or the retrieval of autobiographical, perhaps more emotionally salient memories occurring across the lifespan, are not directly comparable. In addition, as mentioned above, the use of a nonclinical sample may have further contributed to the lack of an observed association between these variables in the current design.

Although proportional recall of NLEs did not independently predict changes in depressive symptoms from T2 to T3, its deleterious effects were significant among those who experienced more NLEs during the EMA week, as predicted. This finding is consistent with vulnerability-stress models of depression positing that cognitive vulnerabilities are more strongly expressed following increases in stressors (Abramson et al., 2002), and suggests that improved memory for stress is particularly detrimental when there is a greater amount of stress to be remembered. The current results support the hypothesis that increased negative autobiographical memory retrieval is not merely a correlate of depression, but serves as an important vulnerability factor predictive of increases in depressive symptoms over time. Indeed, previous research has demonstrated that heightened recall of emotional stimuli (e.g., self-referent words) prospectively predicted increases in depressive symptoms in a nonclinical adolescent sample (Connolly et al., 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, the present study is the first to identify the proportional recall of naturalistic stress as a prospective predictor of depressive symptoms.

Findings are distinct from those within the overgeneral memory (OGM) literature, which proposes that retrieving more abstract and broad negative autobiographical memories, as opposed to specific memories, is a risk factor for depression (Williams, 1996; Sumner, 2010). Indeed, recent work found an interaction between stress, trait rumination, and OGM retrieval in prospectively predicting depressive symptoms among adolescents (Hamlat et al., 2015). The structure of the current task inherently encouraged specificity in memory recall, by constraining participants to remember events from a prescribed one-week period and providing them with a calendar to log their memories, and therefore, cannot be directly compared to OGM designs in which participants are given a word and asked to freely recall an associated memory from their lives. As such, it is possible that rumination may both 1) improve memory for negative information that occurred during a prescribed time period and was retrieved in a structured fashion, and 2) contribute to overgeneral retrieval styles when engaging in unstructured recall of memories occurring across the lifespan. Future research employing EMA designs over longer time periods could help to illuminate potential differences in specific versus overgeneral memory retrieval among ruminators, and may benefit from more unstructured retrieval prompts at follow-up (e.g. "tell us what happened over the EMA period," to better code for overgeneral versus specific retrieval style, versus "list as many memories as you can from the EMA period on this calendar" which inherently encourages specificity in recall.)

The current study utilized EMA methodology to measure the recall of NLEs experienced in individuals' daily lives. This design represents 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 across the lifespan, which assesses self-referent information but precludes the measurement of cognitive processes that may have occurred at the time of 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 stressors during the EMA week. Memory for neutral information also was 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 and future directions 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) event recall and depressive symptoms, the full mediation model of SRR predicting event recall predicting depression was not significant. Therefore, it cannot be concluded from the current findings that SRR is predictive of subsequent increases in depressive symptoms over time and that this relationship is explained in part by negative event recall. Future research, perhaps employing clinically depressed samples, is necessary in order to expand upon the present test of this mediational hypothesis, with the goal of potentially linking the observed relationship between rumination and recall with the subsequent development of depressive symptoms.

The events experienced during the EMA week were generally mild in intensity, as has been reported in a similar EMA study (Ruscio et al., 2015). As such, future studies may benefit from the use of 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. Furthermore, despite there being a wide range of depressive symptoms and histories in the current sample, with 41% of participants having experienced at least one major depressive episode in their lifetime, it would be important to test these relationships in currently depressed samples to further understand the interplay between these cognitive factors, as noted above. Finally, while the SADS-L and LES measures employed in this study have demonstrated acceptable interrater reliability in previous examinations, these statistics were not calculated within the current research, which represents a methodological limitation.

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. This recall, in combination with greater stress exposure, was found to increase depressive symptoms over time. 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. Although the current research did not find a direct link between rumination and prospective changes in depressive symptoms, a relationship was found between event recall and shifts in symptom levels over time. As such, interventions intended to decrease recall of negative life events may help to decrease depressive symptoms.

Cognitive bias modification (CBM) is a promising area of research aimed at targeting vulnerabilities including rumination and negative memory processes 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, memory for daily stress, and subsequent mood.

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, greater levels of reported stress during the EMA week, in combination with increased recall of these stressors, was predictive of increases in depressive symptoms at follow-up when controlling for previous symptoms. Thus, these findings provide important preliminary support for the hypothesis that engaging in rumination leads to strengthened retrieval of negative autobiographical memories over time, and that proportional recall of naturalistic stress may serve as a risk factor for the development of depressive symptoms. Current results support the development and use of clinical interventions that target rumination and memory processes with the goal of ultimately alleviating depressive symptoms.

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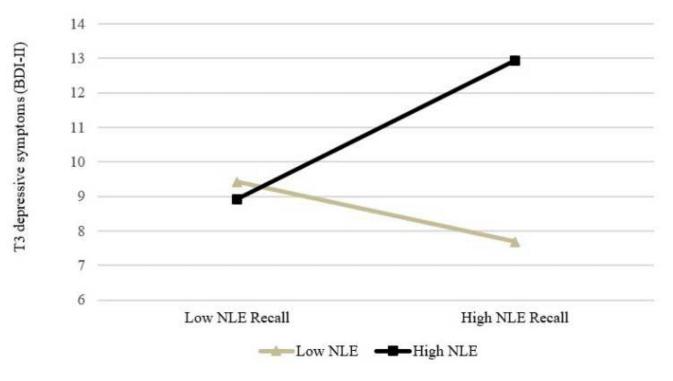
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## Figure 1.

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  $\pm 1$  SD from grand means.

Table 1

**Descriptive Statistics** 

|                |    |  | 1     | 7       | c       | t                  | ,                  | •    |                                   | •       | Y                       | 10   | П               | 12                 |
|----------------|----|--|-------|---------|---------|--------------------|--------------------|------|-----------------------------------|---------|-------------------------|------|-----------------|--------------------|
| Trait Measures | -  | T1 Depressive Symptoms (BDI-II)                |       | .80 *** | .78 *** | .49 <sup>***</sup> | .36 <sup>***</sup> | .07  | 16†                               | .38***  | .27 **                  | .16† | .27 **          | .48 ***            |
|                | 7  | T2 BDI-II                                      |       |         | .81 *** | .42 ***            | .25 **             | .10  | $16$ <sup><math>\div</math></sup> | .39 *** | .25 **                  | .14  | .29 **          | .44                |
|                | З  | T3 BDI-II                                      |       |         |         | .39***             | .24 **             | 60.  | 18*                               | .41     | $.18$ $^{\prime\prime}$ | .14  | .15             | .38 ***            |
|                | 4  | T1 Brooding Rumination (RRS)                   |       |         |         |                    | .63 ***            | 01   | .04                               | .04     | .34 ***                 | .14  | .35 ***         | .26**              |
|                | S  | T1 Stress-reactive Rumination (SRRS)           |       |         |         |                    |                    | 06   | 13                                | .04     | .28**                   | .13  | .43 ***         | .34 ***            |
|                | 9  | TI CVLT-II                                     |       |         |         |                    |                    |      | .05                               | .08     | 12                      | 90.  | .04             | 11                 |
|                | 7  | Negative life event proportional recall        |       |         |         |                    |                    |      |                                   | 44 ***  | 08                      | 02   | 14              | $18^{*}$           |
| EMA Measures   | 8  | # Negative Life Events Endorsed                |       |         |         |                    |                    |      |                                   |         | .19*                    | 60.  | $.16^{\dagger}$ | .35 ***            |
|                | 6  | Average Life Event Subjective Intensity        |       |         |         |                    |                    |      |                                   |         |                         | .14  | .62 ***         | .46 <sup>***</sup> |
|                | 10 | 10 Average Life Event Objective Intensity      |       |         |         |                    |                    |      |                                   |         |                         |      | 05              | .11                |
|                | 11 | 11 Average Stress-reactive Rumination (SRRS-S) |       |         |         |                    |                    |      |                                   |         |                         |      |                 | .53 ***            |
|                | 12 | 12 Average Depressive symptoms (SDS)           |       |         |         |                    |                    |      |                                   |         |                         |      |                 |                    |
|                |    | W  | 11.88 | 10.32   | 9.15    | 10.48              | 39.83              | 6.28 | 0.48                              | 9.45    | 1.95                    | 1.17 | 20.29           | 7.23               |
|                |    | SD   | 8.16  | 8.2     | 8.61    | 3.21               | 15.56              | 3.17 | 0.20                              | 5.51    | .48                     | .19  | 9.22            | 3.18               |

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California : State State. SUS 12/10tal negative life events endorsed during EMA week. SKKS-S = Stress-Reactive Rumination Scale-= negative life events recalled at Verbal Learning Test. Proportional recall Depression Scale. Ž

 $\begin{array}{c} \dot{r} \\ p < .10, \\ * \\ p < .05, \\ ** \\ p < .01, \\ *** \\ p < .001. \end{array}$ 

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| Life Event Recall at Follow-up |
|--------------------------------|
| еI                             |
| Negativ                        |
| nination Predicts N            |
| Rumination                     |
| Stress-reactive Run            |

|  | Wi     | Without SRRS-S | C-CNT             | 5      |      | With SRRS-S       | IS     | SKKS-S Only | Duly              |
|--|--------|----------------|-------------------|--------|------|-------------------|--------|-------------|-------------------|
| Predictor  | в      | OR             | 95% CI            | B      | OR   | 95% CI            | B      | OR          | 95% CI            |
| Event-Level                                      |        |                |                   |        |      |                   |        |             |                   |
| Objective intensity <sup>a</sup>                 | .96    |                | 2.62 (1.86, 3.71) | .98    |      | 2.67 (1.87, 3.81) |        |             |                   |
| Subjective intensity <sup>a</sup>                | .24 *  | 1.27           | (1.05, 1.53) .12  | .12    | 1.13 | (.91, 1.40)       |        |             |                   |
| Chronicity                                       | .82    | 2.28           | 2.28 (1.52, 3.43) | .83    | 2.29 | (1.51, 3.47)      |        |             |                   |
| Stress-reactive rumination (SRRS-S) <sup>a</sup> | ł      | 1              |                   | .02*   | 1.02 | (1.00, 1.04)      | .03*   | 1.03        | 1.03 (1.02, 1.04) |
| Individual-Level                                 |        |                |                   |        |      |                   |        |             |                   |
| Intercept  | 37 *** | 69.            | (.60, .80)        | 37 *** | 69.  | (.60, .80)        | 27 *** | LT.         | (.68, .87)        |
| 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 | 1.06 (.69, 1.64)  |        |             |                   |
| Aggregated chronicity $b$                        | .23    | 1.25           | 1.25 (.43, 3.63)  | .11    | 1.12 | (.37, 3.41)       |        |             |                   |
| Aggregated SRRS-S $^{b}$                         | I      | I              | 1                 | 02     | 86.  | (.96, 1.00)       |        |             |                   |

bgrand-mean centered

#### Table 3

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

| Predictor                            | В       | SE   |
|--------------------------------------|---------|------|
| Intercept                            | 1.23    | .81  |
| T2 BDI-II                            | .81 *** | .06  |
| NLE occurrence <sup>a</sup>          | .20*    | .10  |
| NLE proportional recall <sup>a</sup> | 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,

\*\*\* r p<.001