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Co-variation between stressful events and rumination predicts depressive symptoms: An eighteen months prospective design in undergraduates

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Abstract

Rumination is a maladaptive form of emotion regulation and seems to be the cognitive mechanism linking stress to depressive symptoms. However, it remains to be investigated whether individuals' variation in rumination in relation to the occurrence of stressful events (e.g., phasic co-variation between stressful events and rumination) prospectively predict the experience of depressive symptoms in lengthy follow-up moments. In this eighteen months prospective design, a large unselected sample of undergraduates was tested before, during, and after a period with prominent naturally occurring stressful events. The multilevel results show that the co-variation of stressful events and ruminative thinking predicts the experience of depressive symptoms at 3 and 15 months follow up moments, also when statistically controlling for baseline depressive symptoms. Moreover, the data demonstrate that the phasic elevations of rumination in relation to the occurrence of stressful events are more predictive of depressive symptoms compared with the stable aspects of rumination measured at one occasion. At the clinical level, the current findings seem to suggest a process-oriented intervention to target the phasic ruminative cognitions where individuals need to learn to control rumination exactly at moments of stress.

Keywords: Stressful events, rumination, depressive symptoms, prospective design

Stressful life-events are known to initiate negative, self focused thoughts, rendering individuals vulnerable for depression (e.g., for a review, see Monroe, Slavich, & Georgiades, 2014). In the processing of these stress-induced negative cognitions, depressive rumination – passively and repetitively contemplating symptoms and consequences of distress (Nolen-Hoeksema, 1991; 2000) - is considered a key mechanism of action. Although individuals often ruminate to understand negative feelings and problems, rumination does not enhance clarity and instead has detrimental effects on emotional well-being (Nolen-Hoeksema, Luybomirski, & Wisco, 2008). Considerable empirical support has shown that rumination is a maladaptive form of emotion regulation and has linked this thinking style to numerous maladaptive outcomes (e.g., Nolen-Hoeksema, 1991). Indeed, if these abstract and self-evaluative cognitions regarding the symptoms of distress are recursively activated and rehearsed, symptoms of distress are likely to be maintained (rather than overcome) and become depressogenic (Robinson & Alloy, 2003; Papageorgiou & Wells, 2004; Watkins & Nolen-Hoeksema, 2014). Therefore, rumination may be a cognitive mechanism that mediates the association between stress and depressive symptoms/depression (Ruscio et al., 2015).

Even though many theoretical models exist, the principal theory on depressive rumination is the Responses Styles Theory (RST; Nolen-Hoeksema, 1991), which hypothesizes that depressive rumination is a stable and habitual, trait-like tendency to engage in repetitive self-focused thoughts in response to depressed mood (see Nolen-Hoeksema et al., 2008). Prospective and longitudinal studies provide strong evidence that individual differences in rumination, measured as stable trait, predict the future onset, duration and recurrence of a major depressive episode (e.g., Nolen-Hoeksema et al., 2008), also after controlling for baseline depressive symptoms (e.g., Spasojevic & Alloy, 2001). Moreover, rumination is found to be an important mediating factor in the longitudinal stress-depression relationship in a community sample (e.g., Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013), by

enhancing negative, mood congruent thinking (Nolen-Hoeksema et al., 2008). Within this context, rumination is distinct from neuroticism, a personality trait that refers to a relatively stable tendency to respond with negative emotions in the face of stress. Neuroticism has already been found to be a moderator of the relationship between stress and depressive symptoms (e.g., Hutchinson and Williams, 2007). Yet, prior research has also shown that rumination can be meaningfully distinguished from neuroticism and is found to mediate the relation between neuroticism and depressive symptomatology (Nolan, Roberts, & Gotlib, 1998).

In most prior research, rumination is measured as a stable trait at a single moment in time (typically with questionnaires), and is reported to predict the associations between stress and depressive symptoms (e.g., Abela & Hankin, 2011; Driscoll, Lopez, & Kistner, 2009; Cox, Funasaki, Smith, & Mezulis 2012). Given that the association between rumination, stress and depression are often measured at a single occasion, recall biases confound the associations found between those constructs. Therefore, research has turned to the investigation of rumination more proximal to its occurrence using online reports (e.g., in diary studies, experience sampling studies). A great advantage of this shift in research paradigms is that rumination can be linked more directly to the occurrence of negative or stressful events (e.g., Genet & Siemer, 2012; LeMoult, 2013; see also Smith & Alloy, 2009). This way, rumination can be assessed as a more transient construct, namely a phenomenon that occurs within individuals, across time, and in relation to other time-varying phenomena (e.g., affect, stress). It remains to be investigated, however, whether individuals' variation in rumination in relation to stressful events (i.e., the association between the occurrence of stressful events and rumination measured at multiple moments over time) prospectively predicts the experience of depressive symptoms at a later point in time (e.g., months or a year later). This way, between-subjects longitudinal changes in depressive symptoms can be related to the phasic/dynamic

within-person variation of rumination in relation to stress (i.e., co-variation of stressful events and ruminative thinking) on a more day-to-day level. This way, measurement takes place more proximal to events (which reduces biases through retrospective report).

Moreover, it remains to be investigated whether within-subjects co-variation between stress and rumination predict depressive symptoms at a later time point, above and beyond the habitual tendency to ruminate, as measured for example as a stable trait at baseline. Robinson and Alloy (2003) already provided some evidence that stress-reactive rumination predicted depressive symptoms, even when baseline rumination was controlled for. This seems to suggest that stress reactive elevations of rumination may be more predictive of depressive symptoms compared with the stable aspects of rumination measured at one occasion. This implies that measuring rumination in relation to stressful events over time would more optimally predicts depressive symptoms at a later point in time compared with a single measurement of habitual rumination. Nevertheless, to date, only few studies investigated rumination in relation to stressful events using multiple measurements across time, usually only assessing a general tendency to ruminate (e.g., Robinson and Alloy, 2003), or not including lengthy follow-up assessments (e.g., Ruscio et al., 2015).

Hence, in the current study, an eighteen months prospective design, including a multi-wave assessment of stressful events and rumination, was utilized. This study was performed in a large unselected sample of undergraduates before, during, and after a mid-term exam period (a period with prominent naturally occurring stressful events). Depressive symptoms were measured at baseline (2 months before the exam period) and at 3 and 15 months follow-up (post exam period). Stressful events and rumination were measured weekly during the exam period at multiple times. We hypothesize that the co-variation between self-reported stressful events and rumination within students across the exam period (i.e., the strength with which rumination and events are linked during this stressful period) predicts depressive

symptoms 3 as well as 15 months later, even after controlling for baseline levels of depressive symptoms and trait rumination.

Methods

In this study, a longitudinal design was used including multiple test moments (i.e., intensive wave) during a time of elevated naturalistic stress (i.e., exams; see also Fox et al., 2010; Vanderhasselt et al., 2012) to predict depressive symptoms in times of normal naturalistic stress.[1]

Participants

An unselected group of 92 undergraduate students of Ghent University (20M/72F) with a mean age of 20.27 (*SD*=2.04) participated in this study. Participants were recruited via the university website and received a financial compensation for their participation.

Material

In the present study, internal consistency of all the questionnaires was very good at each assessment moment (see Table 1).

Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996, Dutch translation by Van der Does, 2002). The BDI-II was administered to evaluate depressive symptoms. The BDI-II is a widely used self-report questionnaire consisting of 21 multiple choice format items (4 point scale), to assess the presence and severity of cognitive, motivational, affective, and somatic symptoms of depression. Past reports demonstrated established reliability and validity in clinical and non-clinical samples (Hautzinger, Bailer, Worall, & Keller, 1995).

Ruminative Response Scale (RRS) (Nolen-Hoeksema & Morrow, 1991; Dutch translation by Raes & Hermans, 2007). The RRS was administered to measure ruminative

[1] This study is conducted part of a larger longitudinal project, assessing different measures of cognitive control, various questionnaires as well as genetic information. Results on the role of cognitive emotion regulation strategies on the relation between dysfunctional attitudes and depressive symptoms were presented in an earlier manuscript (see Vanderhasselt et al., 2014).

thinking styles. The RRS consists of items that describe responses to a depressed mood, related to focussing on the self, on symptoms, and on the origin and consequences of the distress. This self report questionnaire consists of 22 questions to which participants respond on a 4-point Likert scale how often they engage in these responses (i.e. 1 = almost never, 2 = sometimes, 3 = often, 4 = most of the times). Instructions of the RRS (administered weekly during the exams period) were "how they responded over the last week".

Adverse Events Questionnaire (AEQ, Carver, 1998). This is a self-report questionnaire especially designed for a student population and is intended to track the occurrence of adverse events that commonly occur in students' lives. This instrument is designed specifically for a college population (for items, see Carver, 1998) and includes academic and relationship domains, occurrence of negative events in any other domain, and the accumulation of minor problems. Participants are asked to indicate if they have had a "relatively major bad experience" in academic, relationships or other aspect of life during last week by answering 'No' = 0 or 'Yes' = 1.

Procedure

All participants received a complete description of the study and provided written informed consent (protocol approved by the local ethics committee of Ghent University). Participants completed questionnaires at six time points, both in the laboratory as well as via e-mail communication. Fist during the initial laboratory session (T1), participants completed the questionnaires on site (i.e., in the lab): BDI-II and RRS (instruction: 'in general'). Approximately 8 weeks later, all participants were preparing for and performing their examinations. During that period, participants completed the AEQ and RRS (instruction: 'over the last week') during four consecutive weeks (T2-T5). The questionnaires were sent out weekly at fixed days, and communication went via e-mail. Participants were instructed to

complete the questionnaires on the same day or the day after receipt, and asked to think of the last week when filling in the questions.

Three months after the end of the exams (T6), a first follow-up assessment was carried out. At that moment, we asked participants come to the lab and complete the BDI-II to measure depressive symptoms. Finally, one year later (T7, 15 months after the exam period), participants filled in the BDI-II again via e-mail to report their depressive symptoms. Thus, the BDI was measured at baseline, as well as 3 and 15 months after the end of the exams[2]. This way, we were able to prospectively predict depressive symptomatology, taking into account baseline levels of depressive symptoms. Trait rumination was measured at baseline, and phasic rumination was assessed four times - together with a measure of the occurrence of stressful events - during the exams. This way, we were able to investigate the dynamic co-variation between rumination and stressful events, taking into account baseline/trait levels of rumination.

Statistical Approach

We used multilevel modeling (MLM) and regression analyses to answer the question of this study. All analyses were carried in SAS 9.2. Outliers were defined as values +/- 3 *SD* above/below the mean and were winsorized by replacing scores more than 3 *SD*s above/below the sample mean with values equal to the mean +/- 3 *SD*s[3].

In a first step, we estimated the within-person association between rumination and the occurrence of events during the exam period using MLM. This procedure was chosen to account for the hierarchical nature of the data with occasions nested in individuals.

[2] The whole study lasted for 18 months (i.e., 1,5 years): baseline testing (T1) was performed 2 months before the exams, exam period lasted for 1 month (T2-T3-T4-T5), the first follow-up assessment (T6) was performed 3 months after the end of the exams (i.e., 6 months after the initial baseline testing) and the second follow-up assessment (T7) was performed 15 months after the end of the exams (i.e., 18 months after the initial baseline testing).

[3] Using this procedure, we detected 7 individuals with outlying values; depressive symptoms at T1, >M+3SD: three individuals, depressive symptoms at T2, >M+3SD: one individuals, depressive symptoms at T3, >M+3SD: two individuals, rumination-event association, >M+3SD: three individuals. There were no outlying values at the within-person level.

Rumination was treated as the outcome variable in the analysis and the occurrence of stress was treated as the level-1 predictor. That is, we estimated the increase in rumination at occasions with stressors in comparison to occasions without stressors. The fixed effect of this model reflects the average increase in rumination in the sample. Additionally, we specified a random effect, which means that we expected variation across participants in the strength of the association between rumination and event occurrence. This is an important issue with regard to our hypothesis—we expect these individual differences to be of predictive value for future depressive symptoms (i.e., participants with stronger associations are predicted to have higher future levels of depressive symptoms).

To test the latter assumption, we obtained estimates from MLM for the within-person rumination – event association for each individual (i.e., MLM random effects estimates). In a second step, we used these estimates as predictors in regression analyses. Particularly, we predicted depressive symptoms at 3 months (T6) and 15 months (T7) after the exam period (2 separate analyses) by the random effect estimates. We included both baseline trait rumination, measured with the RRS, and depressive symptoms prior to the exam period (T1) as additional predictors in these regression models. This reveals whether individual differences in the process of stress related rumination predict depressive symptoms over and above individual differences in the global tendency to ruminate (i.e., trait rumination). Additionally, including depressive symptoms at T1 excludes the possibility of capturing preexisting associations with our analysis, rather than prospective effects.

Results

Descriptive statistics for all study variables are presented in Table 1. Most of the participants had Belgian nationality (95.7% of the sample). Half of the undergraduates were in their second bachelor (45,2%), and the other half (54,8%) was in their third bachelor. Exams

were spread over four weeks of the exam period, demonstrated by the fact that about 75% of the students completed at least one exam after the first week (T2) and the last assessment (T5) was up to 4 days after their last exam in 57,5% of the students.

We first examined the association between rumination and the occurrence of stressful events throughout the exam period. Results from MLM revealed that rumination was higher in weeks during which participants experienced a stressful event, estimate = 2.66, *SE* = 0.84, *t* = 3.16, *p* = .002. According to a comparison of the residual variance of this model with an empty model (i.e., a model without any predictor), stressful events explained 31% of variance in rumination across the four time points throughout the exam period.

Next, we predicted depressive symptoms at a 3-month (T6) and 15-month (T7) follow-up from the association between rumination and stressful experiences throughout the exam period (T2-T5), while also including both trait rumination and depressive symptoms at baseline (T1) into the model. The results are presented in Table 2[4]. The results indicate that students with a relatively strong association between stressful events and rumination throughout the exam period had higher levels of depression three months after the exam period (T6). The unique effect (R-square unique) of ruminative reactions on future depressive symptoms (T6) was 4.4%. Similarly, these students also had higher levels of depression at 15-month follow-up (T7), with R-square unique = 3.6%. Of note, trait rumination (T1) did not predict future depressive symptoms (T6, T7) in this latter multilevel model. Please keep in mind that the effects in multiple regression are partial regression effects. When analysed separately, trait rumination (T1) is moderately correlated with depressive symptoms at

[4]We repeated the model without trait rumination at T1. The association between rumination and stressful events also predicted depressive symptoms at the 3- and 15-month follow up (*p* = .003 and *p* = .02, respectively). Moreover, we repeated these models replacing the total rumination scores with the brooding subscores (at T1), which is considered the most depressogenic type of rumination (Treynor et al., 2003). These analyses revealed similar results.

baseline (T1), $r = .32$, $p = .002$ and at the 3-month follow-up (T6), $r = .32$, $p = .002$, and only weakly correlated with depressive symptoms at the 15-months follow-up (T7), $r = .19$, $p = .08$.

Follow-up analysis

The MLM that was used to model the association between rumination and stress does not allow disentangling the order of the two variables across time. In order to elucidate the temporal order of the variables, which would be important information in consideration of interventions, we ran a cross-lagged panel model. Such a model allows for the simultaneous investigation of lagged and cross-lagged effects or two or more variables. More specifically, we specified a model with two time series, rumination and stress (4 time points each). Both time series had autoregressive effects (lag1 and lag2) to model stability in both variables across time. Furthermore, we included cross-lagged effects in which we simultaneously predicted (a) rumination at T by stress at T-1 as well as (b) stress at T by rumination at T-1. Similarity constraints were set for all effects (the lag1 effects of stress, the lag1 effects of rumination, the lag2 effects of stress, the lag2 effects of rumination, the cross-lagged effects of stress on rumination, and the cross-lagged effects of rumination on stress).

Results from the time-lagged analyses revealed the following: the model fit of this model was acceptable according to the CFI (.98) and RMSEA (.07). Importantly, none of the cross-lagged effects were significant (i.e., the effects of rumination on stress and vice versa; p-values $> .25$). That is, neither did previous stress predict current rumination nor did previous rumination predict current stress. However, the concurrent associations (co-variation) of stress and rumination (at T2, T3, and T4) were significant (and all lagged effects were significant, too).

Discussion

The current study sought to examine whether depressive symptoms are best predicted by tonic levels of rumination (i.e., trait) or phasic levels of rumination (i.e., more dynamic fluctuations in relation to stressful event). For this purpose we used a prospective design that tracked depressive symptoms up to 1.5 years (i.e., eighteen months) after initial testing. Overall, rumination was higher during weeks during which participants experienced a stressful event. The multilevel results show that the co-variation of stressful events and ruminative thinking predicted the experience of depressive symptoms at both follow-up moments (3 and 15 months). This effect remained even when statistically controlling for baseline depressive symptoms.

In this study, participants were asked about the occurrence of a relatively major bad experience in the past week, without asking them whether they perceived the experience as stressful, and rumination was measured independently. In contrast, prior studies instructed participants to report their tendency to ruminate about negative inferences following stressful events (e.g., Nolen-Hoeksema & Morrow, 1991; e.g., "Think about how the stressful event was all your fault," Robinson & Alloy, 2003). This latter instruction could have directed participants to report their rumination when they are feeling (dis)stressed in general (i.e., impact of timing and context on the assessment). This current study's design, on the contrary, illuminates whether naturally occurring stressful experiences that are co-occurring with a perseverative focus on one's distress and its causes/consequences measured at multiple moments in time, predict individual differences in depressive symptoms. It should be noted that - even though the retrospective bias was minimized as much as possible in this study - the repeated phasic measurement of rumination in the exam period still included a retrospective evaluation given the instruction to refer to the last week when evaluating feelings distress/or

negative mood. Therefore, an even more proximal assessment (last hour/day instead of week) of stress and rumination could be used in future research.

The habitual tendency to ruminate (measured at T1) was associated with depressive symptoms (at T1, T6, and T7), such that individuals who report the tendency to ruminate display elevated depressive symptoms across time. This is in line with the well-known association between trait rumination and the onset of major depressive episodes (Nolen-Hoeksema, 2000), even though the current participants were non-depressed undergraduates. However, when trait rumination (T1), depressive symptoms (T1), and phasic levels of rumination in co-occurrence of stressful events (T2-T5) were all entered in one model, the phasic levels of rumination emerged as the strongest predictor, explaining a significant proportion of variance in depressive symptoms at follow-up (T6, T7). The current findings thus underscore that the variability in rumination in relation to stressful events uniquely predicts depressive symptoms. Even though rumination is often seen as a relatively stable response style (Nolen-Hoeksema et al., 2008), it is also a transitive, state-like, phenomenon that fluctuates according to the presence or absence of a certain triggers (Smith & Alloy, 2009). Based on our results, we can conclude that these stress related fluctuations in rumination play a stronger role than the trait tendency to ruminate when predicting depressive symptoms.

According to theoretical considerations, one would expect a temporal order of the occurrence of stress and rumination across time, and even bidirectional effects. Unfortunately, our main findings do not inform about the temporal order of rumination and stress during the exam period, and thus within-person mechanisms in the manifestation of stress and rumination. Our additional cross-lagged panel analysis revealed that neither did rumination predict stress across time, nor did stress predict rumination across time. This suggests that the co-variation of stress and rumination was most predictive instead of other sequential effects of

rumination or stress. Our interpretation of this finding is that the exam period holds much temporal variation in terms of stressful events and rumination across subjects, which do not necessarily predict each other in a sequential order. We therefore interpret our results as the phasic co-variation between rumination and stressful events, to emphasize the dynamical variability of rumination across weeks, which do not inform about the within subjects mechanisms of the development of rumination in reaction to stress (or the other way around).

Even though stress and the risk for depression are strongly linked (for a review, see Monroe et al., 2014), not all individuals that are exposed to stress develop depressive symptoms. Individuals are not passive respondents to stressful events, but actively try to cope with the stressor, sometimes in a maladaptive way (e.g., rumination). The current findings demonstrate that rumination in association with stressors plays an important role in predicting depressive symptoms. Based on the vast amount of research, rumination is found to prompt ineffective problem solving (Nolen-Hoeksema et al., 2008), and guide the appraisal of problems as overwhelming and unsolvable (Lyubomirsky et al. 1999). This, in turn, could exacerbate low mood in the short-term and explain its role in the etiology of depression in the long-term.

Given that interventions on rumination are imperative in treatment of depression (Wells & Papageorgiou, 2003), the present results have implications at the clinical level. The current findings highlight that cognitive interventions should target phasic, reactive ruminative thoughts, in addition to the treatment of a habitual tendency to ruminate. Hence, interventions should focus on ruminative cognitions in response to stressful events, frustration, negative thoughts or feelings. This process oriented way to intervene could involve taking into account the ecological day-to-day dynamic interplay between stress and rumination, where individuals need to learn to control rumination exactly at moments of stress. Moreover, meta-cognitive beliefs about the usefulness of rumination as a coping

strategy to deal with negative affect, induced by stressful events, could be targeted in order to make individuals more resilient to the onset and persistence of depressed mood.

Despite the strengths of the study, including a combination of a longitudinal design with an intensive measurement period as well as the lengthy follow-ups, some limitations need to be discussed. Although rumination as measured throughout the exam period conceptually implies that individuals with high levels rumination focus their affective experiences, mood was not measured directly. A future study should incorporate an affect measurement to establish dynamic links between stress, affect, and rumination. That way it could be examined in more detail whether rumination is a reaction to the occurrence of stressful events, or whether affective distress and a ruminative response are preceding the occurrence of distress and prospectively predict depressive symptoms in healthy undergraduates. Second, this study has worked with a largely homogenous non-clinical sample that limits the generality of the interpretations. Moreover, albeit we speak of depressive symptoms, we do not have any diagnostic information. That is, we cannot claim that the association between rumination, stressors, and depressive symptoms as observed in this study eventually results in a depressive episode, especially as the depressive symptoms scores are rather low in this population. The low base rate of depressive symptoms in the current sample may call into question the clinical significance of the findings. However, depressive symptoms, as measured with the BDI-II, are relevant as they refer to the individual differences in depressive symptomatology, which is predictive of the development of full-blown depressive episodes. It should be noted that the variability of the BDI-II scores across participants was substantial, both within and between subjects. Third, phasic and tonic aspects of rumination were measured at different points in time, with phasic (T2-T5) rumination being measured more closely to the follow-up periods (T6, T7) than trait rumination (tonic, T1). One might argue that, because phasic rumination was measured at a later time point than

trait rumination it could predict depressive symptoms better. However, trait rumination is considered a stable habit where the exact moment of measurement is less likely to influence scores. Moreover, these differences in timing are highly unlikely to still influence depressive symptoms measured eighteen months later. Finally, neuroticism was not measured in the current study, even though it might have informed of how rumination predicted the relation between stress and depressive symptoms, above and beyond neuroticism as a trait vulnerability factor for depression.

To conclude, the dynamic co variation of increased ruminative thinking when experiencing more stressful events across time (i.e., phasic rumination) uniquely predicts individual differences in depressive symptoms, above and beyond a stable trait to ruminate and baseline depressive symptoms. In clinical practice, meta-cognitive beliefs to use rumination as a coping strategy to negative affect, induced by stressful events, could be targeted in order to make individuals more resilient to the onset and persistence of depressed mood.

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Table 1.

Descriptive Statistics of all Study Variables. The values represent mean score across participants at that moment in time (T1, T6, T7) or the average score across participants across four moments in time (T2, T3, T4, T5).

	N	*M*	*SD*	*Cronbach's α*
Depressive symptoms, baseline (T1)	92	6.16	6.29	.88
Depressive symptoms, 3-months follow-up (T6)	89	4.99	5.57	.89
Depressive symptoms, 15-months follow-up (T7)	82	4.68	3.31	.88
Trait rumination, baseline (T1)	92	42.08	12.80	.94
Rumination average across the exam period (T2-T5)	92	35.28	10.48	
Stressor occurrence across the exam period (T2-T5)	92	0.23	0.28	

T1*: baseline measurement at two months before the start of the exams period;* ***T2-T5****: four weekly measurements during the one month exam period;* ***T6****: three months after the end of the exams period;* ***T7****: fifteen months after the end of the exams period.*

Table 2.

Results from Regression Analyses.

	Estimate	***SE***	***t*-value**	***p***
Criterion:				
Depressive symptoms at 3-month follow-up (T6)				
Intercept	1.35	1.86	0.72	0.47
Depression at baseline (T1)	0.33	0.09	3.64	0.001*
Trait rumination at baseline (T1)	0.04	0.04	0.94	0.35
Rumination - stressor association (T2-T5)	1.87	0.74	2.54	0.01*
Criterion:				
Depressive symptoms at 15-month follow-up (T7)				
Intercept	2.49	2.01	1.24	0.23
Depression at baseline (T1)	0.15	0.09	1.59	0.12
Trait rumination (T1)	0.03	0.05	0.67	0.51
Rumination - stressor association (T2-T5)	1.62	0.79	2.05	0.04*

** significant; follow-up analyses revealed that gender as a covariate of potential interest was no significant predictor of depressive symptoms at T6 or T7 (p-values >.25). Including gender in the model predicting depressive symptoms at T7 resulted in a change of the p-value for the rumination-stressor association from .04 to .06.*