Journal of Abnormal Psychology
2006, Vol. 115, No. 4, 699–704

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0021-843X/06/$12.00 DOI: 10.1037/0021-843X.115.4.699

Reduced Autobiographical Memory Specificity and Rumination in Predicting the Course of Depression

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Reduced autobiographical memory (AM) specificity is a known vulnerability factor for depression. AM specificity was investigated as a predictor of depression with the Autobiographical Memory Test (J. M. G. Williams & K. Broadbent, 1986). When baseline depression scores were partialed, reduced AM specificity to negative cue words predicted higher levels of depression at 7-month follow-up. Once rumination was taken into account by means of the Rumination on Sadness Scale (M. Conway, P. A. R. Csank, S. L. Holm, & C. K. Blake, 2000), AM specificity no longer predicted depression, suggesting that the predictive value of AM specificity observed in previous studies might be—at least partly—explained as an effect of rumination. Further mediation analyses indeed revealed support for rumination as a mediator of the relation between reduced AM specificity and poor outcome of depression.

Keywords: depression, rumination, autobiographical memory

Given depression's high prevalence and recurrence rates (Blazer, Kessler, McGonagle, & Swartz, 1994; National Institute of Mental Health Consensus Development Conference Statement, 1985), it is no surprise that increased emphasis is being placed on early detection and prevention of (relapse into) depression. Of particular interest is the search for trait-markers which could serve as predictors of depression. However, unequivocal findings about vulnerability factors are relatively rare. Most variables are often not specific to depression or tend to wane once depression has remitted. A variable that may qualify as a vulnerability factor is specificity of autobiographical memory (AM).

Twenty years of research using the Autobiographical Memory Task (AMT; Williams & Broadbent, 1986) has shown that people with major depressive disorder (MDD) have more difficulty retrieving specific memories than nondepressed people (Williams, 1996). In the AMT, respondents are asked to retrieve a *specific memory* to cues (e.g., *happy*, *alone*). A specific memory refers to a particular personal event that did not last longer than one day (e.g., "my grandmother's funeral last year"). Compared with controls, people with depression respond relatively more often with *overgeneral* or *categoric memories* that summarize across categories of similar events (e.g., "the times I have to say goodbye," rather than "that very Sunday when I kissed my son goodbye when he left for Paris"). Past research has shown that this lack of AM specificity or overgeneral memory (OGM) does not disappear once the depression remits, with level of AM specificity remaining stable over time even when the severity of depressive symptoms declines (e.g. Brittlebank, Scott, Williams, & Ferrier, 1993).

The extent of this deficit has, moreover, been found to predict clinical outcome in depression. Brittlebank et al. (1993) were the first to report that patients with MDD who retrieved more OGMs on the AMT had a poorer prognosis at 7-month follow-up in terms of higher scores on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), even when initial symptom levels were taken into account. These findings were later replicated by Peeters, Wessel, Merckelbach, and Boon-Vermeeren (2002), who found that a lower number of specific memories was predictive of a worse prognosis, over and above initial symptomatology (though see Brewin, Reynolds, & Tata, 1999, for a nonreplication). Dalgleish, Spinks, Yiend, and Kuyken (2001) further extended these findings to a sample of patients with seasonal affective disorder, reporting that OGM significantly predicted depression at follow-up.

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J. Mark G. Williams is supported by the Wellcome Trust GR067797. We thank Luc Hamelinck, Koen Demyttenaere, Sieglinde Meganck, Bart Jansen, Bernard Sabbe, Carmen De Grave, Stefaan Vertommen, and Guido Pieters for their assistance in conducting this study.

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Although many studies have shown that OGM is a clear characteristic of depression (Williams, 2004) and that it appears to predict a more chronic course, little is known about the underlying mechanisms that might explain how OGM leads to such an unfavorable course. One possible way in which OGM may impede recovery from depression is through a spiraling, reciprocal relationship with depressive rumination (Williams, 1996, 2004). *Rumination* refers to a constant dwelling on one's depressive symptoms and its causes and consequences (Nolen-Hoeksema, 1991). Williams (1996) suggested that the retrieval of OGMs is "encouraged by and itself encourages ruminative self-focus" (p. 261). There is now experimental evidence in support of this claim. Watkins and colleagues (e.g., Watkins & Teasdale, 2001, 2004) showed that reducing rumination, either using distraction or an experiential self-focus manipulation, reduced OGM in depressed patients. Further, pilot work in our lab has shown that the induction of an overgeneral AM retrieval style, relative to a specific retrieval style, increases the degree to which ruminators' mental mode is consistent with ruminative thinking (Raes, Hermans, Williams, Geypen, & Eelen, in press).

How does OGM lead to rumination? According to Williams' notion of "mnemonic interlock" (Williams, 1996, p. 261), some people's memory retrieval gets truncated at an intermediate stage of retrieval and the tendency to stop the memory search is (negatively) reinforced because it reduces the negative affect attached to specific painful memories. Such intermediate memories are, however, closely related to categoric labels or self-descriptors (e.g., "I've always disappointed other people"). The consequence of persistent use of such an overgeneral retrieval style, with one categoric self-label iteratively leading to another, is a ruminative self-focused processing style. Further, research has clearly shown that overgeneral recall has a deleterious effect on effective problem solving (e.g., Goddard, Dritschel, & Burton, 1996). As such, the persistent use of such an overgeneral retrieval style, in particular when one is trying to solve perceived problems (either past losses or failures, or depressed mood itself), typically results in an endless stream of attempts to think through such problems, which is precisely what rumination is.

It is important to note here that rumination itself has also been intensively investigated in relation to the course of depression in a body of literature separate from that on AM specificity. From that distinct research tradition, rumination has emerged as a clear predictor of a poor prognosis in depression (Nolen-Hoeksema, 2004). The fact that reduced AM specificity is associated with rumination and that rumination has shown to be a reliable predictor of depression outcome suggests that the relation between reduced AM specificity and a poor outcome in depression may be mediated by rumination.

The present study had two aims: first, to investigate the predictive value of reduced AM specificity for depression outcome; second, to investigate whether any effect of lack of memory specificity on depression outcome could be explained by rumination (i.e., whether rumination acts as a mediator). To our knowledge, no prospective longitudinal study with adult MDD patients has so far included both a measure of AM specificity and a measure of depressive rumination. There is one recent prospective study by Park, Goodyer, and Teasdale (2005) with depressed adolescents in which a measure of depressive rumination and a measure of memory specificity were included. However, in that study, neither memory specificity nor rumination were found to independently predict persistent depression. The authors argued that this might have been due to the fact that their sample consisted of adolescents. The present study aimed to investigate in a depressed adult sample whether any effect of reduced memory specificity on depression outcome could be explained by rumination. No study has directly investigated the role of rumination as a possible mediator of the relationship between AM specificity and depression outcome. This is important because the processes underlying the link between reduced AM specificity and poor depression outcome remain unclear.

Method

Participants

Twenty-eight patients (19 women, 9 men) participated from three hospitals in Belgium: University Hospitals Leuven (Leuven; $n = 15$), St-Norbertus (Duffel; $n = 9$), and St-Jozef (Kortenberg; $n = 4$). Inclusion criteria were a *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) diagnosis of MDD without psychotic features and aged 18 to 65 years. Patients were interviewed with the mood modules of the Structured Clinical Interview for *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996; Dutch version: Van Groenestijn, Akkerhuis, Kupka, Schneider, & Nolen, 1999). Diagnostic information on comorbidity was obtained from medical records and from the team's psychiatrist. Exclusion criteria were electroconvulsive therapy in the previous 6 months, bipolar disorder, substance abuse, or organic brain disease. Mean age of the sample was 40.2 years ($SD = 11.6$; range = 21–65). Twenty-one participants were inpatients and 7 outpatients. Nine participants presented a major depressive episode for the first time; the others had suffered recurrent episodes of depression. The most common comorbid diagnoses in the sample were personality disorders ($n = 14$) and anxiety disorders ($n = 2$). At first assessment, all but 4 patients were receiving antidepressant medication.

Materials

AMT. An extended version of the Dutch AMT was used (de Decker, Hermans, Raes, & Eelen, 2003). Participants were asked to recall a specific memory to 18 cue words. Words were presented orally in a fixed order, with nine positive and nine negative words alternating: *happy, sad, safe, angry, interested, clumsy, successful, emotionally hurt, surprised, lonely, relaxed, guilty, proud, scared, pleasurable, cowardly, carefree,* and *lazy*. Positive and negative cues were matched for familiarity, imageability, and emotional intensity. The instructions state that a specific memory refers to one particular personally experienced event that happened on a particular day at least 1 week before. Participants were instructed not to retrieve the same memory twice. They were given 60 s for each cue. If the first response was not a specific memory, participants were verbally prompted to describe a particular time or a particular event. The prompting procedure was repeated until the participant retrieved a specific memory or until the time limit was exceeded. To familiarize participants with the procedure, we gave participants three practice words (*enjoy, friendly,* and *naughty*).[1]

Each response was later coded as a specific memory or a nonspecific memory. The latter were further qualified as a categoric memory (e.g., "watching my favorite sitcom"), an extended memory (e.g., "My sabbatical leave during the 1974-75 academic year"), no memory (e.g., verbal associations), omission, same event (referring to an event already mentioned), or incorrect specific (referring to an event of the past week). Using this

[1] The Dutch words can be obtained from Filip Raes.

scoring procedure, we obtained good reliability ($K = .96$; Raes, Hermans, de Decker, Eelen, & Williams, 2003).

Although earlier studies focused on one particular type of autobiographical error made—that is, categoric memories—later studies have found that a number of other memory errors (e.g., semantic associates) can also contribute to nonspecificity in memory, so the present study examined both OGMs and the total number of nonspecific memories arising from any source.

Rumination on Sadness Scale (RSS). The RSS (Conway, Csank, Holm, & Blake, 2000) is a 13-item questionnaire measuring rumination on sadness. Items are rated on a 5-point scale (1 = *not at all*, 5 = *very much*) for the extent to which they reflect the participant's responses to sadness. All items are preceded by the frame sentence, "When I feel sad, down, or blue. . . ." Sample items are "I repeatedly analyze and keep thinking about my sadness," and "I get absorbed in thinking about why I am sad and find it difficult to think about other things." The RSS has demonstrated good internal consistency ($\alpha = .91$), high concurrent validity with the Ruminative Response Scale (Nolen-Hoeksema & Morrow, 1991; $r = .81$), and good convergent and discriminant validity (for more details, see Conway et al., 2000; Luminet, 2004). The Dutch translation by Raes, Hermans, and Eelen (2003) was used ($\alpha = .84$).

HRSD. The HRSD is a widely used observer-rated instrument for measuring severity of depressive symptoms and consists of 21 items. The Dutch version by Schotte (1996) was used ($\alpha = .70$).

Procedure

Participants were tested individually. Following the SCID and after written informed consent, participants were assessed with the HRSD. Next, they completed the RSS and AMT. Follow-up testing took place 7 months later and consisted of the HRSD, RSS, and AMT.

Data Analysis

Stability of scores was evaluated by repeated measures analysis of variance. Multiple regression analyses were performed to evaluate prediction of depression at follow-up. Following recent recommendations by MacKinnon, Lockwood, and Williams (2004), a nonparametric, resampling approach (bootstrapping procedure; see Preacher & Hayes, 2004b) was used to test a mediation model in which rumination is hypothesized to mediate the relation between reduced AM specificity and depression outcome. At follow-up, depression severity data were available on 24 of the original 28 participants: One patient had been given ECT, and 3 patients refused to participate further.

Results

AMT Performance and Its Stability

Approximately 50% of the first responses to AMT cues at baseline were specific (Table 1), which is comparable to earlier reports (Brittlebank et al., 1993; Peeters et al., 2002). About 29% of the first responses were OGMs, which is comparable to Brewin et al. (1999), but lower than the percentage reported by Brittlebank et al. (1993; 54%). Information on the stability of scores is provided in Table 2. AM specificity was relatively stable (Table 2); level of depression significantly decreased with time. There was a tendency for rumination scores to drop over time, but not significantly.

AM Specificity and the Prediction of Depression Severity at Follow-Up

A multiple regression was carried out, with HRSD scores at 7 months as the dependent variable. The number of specific memories to negative cues and to positive cues, the number of OGMs to negative cues and to positive cues, and HRSD scores at baseline were entered as the five independent variables. The overall model was significant, $R^2 = .32$, $F(5, 18) = 3.19$, $p < .05$. There were significant partial effects of both baseline HRSD score, $t(18) = 2.67$, $p < .05$, $\beta = .48$, and specific memories to negative cues, $t(18) = -2.72$, $p < .05$, $\beta = -.91$. There was no significant effect of AM specificity to positive cues, $t(18) = 0.67$, $p = .51$, $\beta = .15$, and no significant effect of overgeneral AM to negative cues, $t(18) = -1.68$, $p = .11$, $\beta = -.52$, or to positive cues, $t(18) = 1.17$, $p = .26$, $\beta = .33$.[2]

Table 1
Mean Number and Standard Deviations of All Different Response Categories for the Autobiographical Memory Test (AMT) at Baseline (n = 28)

AMT response category	All cues		Positive cues	Negative cues
	M	*SD*		
Specific	9.14	3.22	4.54	4.61
Categoric	3.46	2.44	1.71	1.75
Extended	1.79	1.23	0.89	0.89
No memory	1.25	1.29	0.36	0.89
Omission	1.89	1.50	1.29	0.61
Same event	0.36	0.56	0.14	0.21
Incorrect specific	0.11	0.31	0.07	0.04

Rumination and the Prediction of Depression Severity at Follow-Up

A multiple regression was performed, with HRSD scores at follow-up as the dependent variable. Rumination and HRSD scores at baseline were entered as the independent variables. The model was significant, $R^2 = .44$, $F(2, 21) = 8.27$, $p < .01$; with a significant partial effect of baseline rumination score, $t(21) = 2.98$, $p < .01$, $\beta = .51$; and no significant effect of baseline HRSD score, $t(21) = 1.71$, $p = .10$, $\beta = .29$. This regression was then used as a first step in a multiple hierarchical regression. On the next step, the number of specific memories to negative cues was entered, alongside baseline rumination and HRSD score. Memory specificity did not add to the prediction of follow-up HRSD scores, $\Delta R^2 = .04$, $p = .24$. Thus, once rumination was taken into account, the effect of number of specific memories to negative cues was no

[2] The observed association between AM specificity and follow-up depression scores cannot be accounted for by chronicity or recurrence of the depression because none of the AMT memory indices correlated significantly with either chronicity (duration of current episode) or recurrence (number of previous episodes), with largest $p = .18$ and largest $r = -.28$. Furthermore, when both duration of current episode and number of previous episodes were entered in the regression model, AM specificity to negative cues still came out as a significant predictor of follow-up HRSD depression scores.

Table 2
Mean Scores and Standard Deviations on the HRSD and RSS and for Autobiographical Memory Specificity at Baseline and Follow-Up

	Baseline (n = 28)		Follow-up (n = 24)		Repeated measures analysis of variance	
Measure	*M*	*SD*	*M*	*SD*	*F*	*p*
HRSD	22.18	4.74	14.92	8.79	16.05	< .001
RSS	46.46	9.45	43.87	11.57	2.46	.13
AMT all cues	9.14	3.17	10.32	4.29	1.27	.27
AMT positive	4.54	1.91	5.09	2.29	0.93	.35
AMT negative	4.61	1.69	5.23	2.47	0.72	.41

Note. Analyses are limited to 23 and 22 patients for the Rumination on Sadness Scale (RSS) and Autobiographical Memory Test (AMT) data, respectively. HRSD = Hamilton Rating Scale for Depression.

longer significant, $t(21) = -1.22$, $p = .24$, $\beta = -.22$.[3] Baseline rumination score was the only significant predictor of follow-up HRSD scores, $t(21) = 2.16$, $p < .05$, $\beta = .41$. The effect of baseline HRSD score did not reach significance, $t(21) = 1.71$, $p = .10$, $\beta = .29$. The condition index and variance inflation factors indicated that multicollinearity was not a problem (highest variance inflation factor = 1.38).

Mediation Analyses

Consistent with Baron and Kenny's (1986) definition of mediation, the results of the above reported regression analyses suggest that rumination mediates the relationship between reduced AM specificity and depression. That is, both rumination and reduced specificity are significant predictors of higher levels of depression, but once depression is regressed on both variables only rumination remains significantly related to depression outcome, whereas the relationship between reduced AM specificity to negative cues and depression was reduced to nonsignificance.

As recommended by MacKinnon, Lockwood, and Williams (2004) for those cases in which sample sizes are small, we used a nonparametric resampling method (bias-corrected bootstrap) with 5,000 resamples to derive the 95% confidence interval (CI) for the indirect effect of memory specificity to negative cues via the hypothesized mediator (rumination) on depression (HRSD scores) at follow-up when baseline HRSD scores were partialed (for more details, see MacKinnon et al., 2004; Preacher & Hayes, 2004b). To that end, we used the SPSS Macro provided by Preacher and Hayes (2004a). The true indirect effect was estimated to lie between –2.6408 and –0.1126 with 95% CI. Because zero is not in the 95% CI, we can thus conclude that the indirect effect is significantly different from zero at $p < .05$ (two-tailed). The direct effect of memory specificity was no longer significant ($p = .24$).

Discussion

The results of this study show that when baseline depression levels were taken into account (a) reduced AM specificity to negative cues predicted a poorer outcome in depression and (b) the inclusion of rumination as a mediator reduced the direct relationship between AM specificity and depression at follow-up to nonsignificance.

The data replicate earlier reports that a higher level of memory specificity at admission predicts less depression at 7-month follow-up, even when initial depression levels were partialed (Brittlebank et al., 1993; Dalgleish et al., 2001; Peeters et al., 2002). In the present study, it was specificity to negative cues that was related to depression outcome, replicating Peeters et al. (2002). Brittlebank et al. (1993) and Dalgleish et al. (2001), however, found that depression at follow-up was related to level of specificity to positive cues. It is a typical observation in the AMT literature that effects sometimes are related to negative cues only, sometimes to positive cues only. In fact, a recent meta-analysis has come to the conclusion that depressed people are less specific to both positive and negative cues (Van Vreeswijk & de Wilde, 2003). We agree with Dalgleish et al. (2003) that this valence effect is likely not due to the valence of the cue or the valence of the corresponding memory, but rather to the word itself: "It may be the case that OGMs are elicited by cues reflecting particular autobiographical themes rather than by cues of differing valence" (p. 219). In line with Peeters et al. (2002) and Mackinger and Svaldi (2004), it was a reduced amount of specific memories and not an elevated level of OGMs (e.g., Brittlebank et al., 1993) that was associated with depression outcome in the present study. The absence of such relationship for overgenerality in the present study was most likely due to the lower proportion of OGMs retrieved in the current sample compared with previous samples.

With respect to our second objective, we found that rumination also predicted an unfavorable outcome for depression, which concurs with a large amount of research published over the past decade (Nolen-Hoeksema, 2004). The main reason why we had included rumination was that previous research had shown rumination to be related to reduced AM specificity (see the introduction). Therefore, we wanted first to verify whether reduced AM specificity would still add to the prediction of depression, once rumination is partialed. Results showed that it did not, suggesting that the effect of reduced AM specificity was at least partly explained by its relation with rumination. Secondly, by including rumination, we wanted to further test whether rumination acts as a

[3] As expected, AM specificity was significantly negatively related to rumination, $r(28) = -.43$, $p < .05$. This correlation remained significant when depression scores were partialed (HRSD), $r(28) = -.44$, $p < .05$.

mediator between reduced AM specificity (to negative cues) and poor outcome in depression. Mediation analyses indeed showed that rumination does significantly mediate at least a portion of the AM specificity association with depression. These results are a first indication of rumination as one possible mechanism or pathway by which reduced AM specificity exerts its negative effects on the course of depression.

Given the intimate relationship between a ruminative thinking style and OGM, we suggest that they might represent two different aspects reflecting a common underlying cognitive process. This cognitive process would be a discrepancy-driven process that attempts to use analytical thinking (rumination) and overly general self-referent knowledge (OGM) to solve problems related to the self or current mood. Researchers in the field of rumination increasingly agree that such analytical rumination might indeed be conceptualized as some form of problem solving (e.g., McLaughlin, Sibrava, Behar, & Borkovec, in press; Segal, Williams, & Teasdale, 2002). In the case of depressive rumination, the typical problems that are perceived are the negative mood itself, past losses or failures, and current problems (e.g., McLaughlin et al., in press). For example, feeling depressed can be perceived as discrepant from a desired state, namely no longer feeling depressed or feeling happy. It is the perception of such a mismatch that makes depressed or depression-prone people shift to rumination to tackle these problems. However, such mismatches lead to often fruitless attempts to reduce them, which in turn lead to more undesired negative mood (Carver & Scheier, 1990; see also Segal et al., 2002). This discrepancy-based processing thus uses (a) verbal–abstract analytic reasoning, with questions such as "where did it all go wrong" and (b) self-relevant material, in the form of categoric statements ("having no friends," "feeling dreadful," "feeling good," "being around other people"). Both these aspects interact and mutually fuel each other in a spiral interaction (Williams, 1996, 2004; Williams et al., in press).

An important limitation of the present study was its relative small sample size. However, it should be noted that to test the crucial hypotheses regarding mediation and indirect effects, we did not use only regression analyses but also the nonparametric resampling approach (bootstrapping procedure), which has been specifically designed to draw valid and robust conclusions in such small samples that do not meet the assumption of multivariate normality (MacKinnon et al., 2004; Preacher & Hayes, 2004b). Another possible limitation of the current study was that our sample of MDD patients was characterized by a low number of comorbid anxiety disorder diagnoses (less than 10%), whereas typically higher percentages are reported for patients with major depression who also suffer from a current anxiety disorder (e.g., Sartorius, Ustun, Lecrubier, & Wittchen, 1996). This may thus limit to some extent the generalizability of the present findings to depressed populations with higher rates of comorbid anxiety states. Nevertheless, this study provides important preliminary evidence about the possible cognitive processes that combine to produce serious long-term depression.

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Received April 11, 2005
Revision received May 2, 2006
Accepted May 30, 2006 ■