Testing the CaR–FA–X Model: Investigating the Mechanisms Underlying Reduced Autobiographical Memory Specificity in Individuals With and Without a History of Depression

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Abstract:

Reduced autobiographical memory specificity (AMS) is an important cognitive phenomenon in major depressive disorder (MDD), but knowledge about mechanisms is lacking. The CaR–FA–X model of Williams and colleagues (2007) proposed that 3 processes contributed to reduce AMS: capture and rumination (CaR), functional avoidance (FA), and impaired executive control (X). However, the entire CaR–FA–X model has not been tested. We addressed this gap in the literature by investigating contributions of the CaR–FA–X mechanisms to reduced AMS, alone or in interaction, in a subset of young adults (N = 439) from the Northwestern–UCLA Youth Emotion Project. Participants were classified as those with (n = 164) and without (n = 275) a history of MDD at AMS assessment. They completed measures of: AMS; rumination (the brooding factor; CaR); childhood, adolescent, and early adulthood adversity (FA); avoidant coping (FA); and verbal fluency (X). Using structural equation modeling, we found greatest support for associations between reduced AMS and the capture and rumination, and impaired executive control mechanisms. In those with and without a history of MDD, brooding and verbal fluency interacted to contribute to reduced AMS. For participants without a history of MDD, lower verbal fluency (indicating impaired executive control) was associated with reduced AMS among those high on brooding. For participants with a history of MDD, lower verbal fluency was associated with reduced AMS among those low on brooding. The first finding was consistent with the CaR–FA–X model but the latter was not. Implications for conceptualizations of reduced AMS and its mechanisms are discussed.

Keywords: reduced autobiographical memory specificity | overgeneral autobiographical memory | CaR-FA-X model | mechanisms | depression

Article:

Reduced autobiographical memory specificity (AMS) refers to the finding that when asked to generate a specific autobiographical memory in response to a cue word, some individuals are less specific or more overgeneral in their recall than others (see Sumner, 2012; Williams et al., 2007,

for reviews). This cognitive phenomenon, also known as overgeneral autobiographical memory, has strong associations with certain emotional disorders. In particular, research has shown that individuals with depression exhibit lower levels of AMS than nondepressed controls (e.g., Williams et al., 2007). This phenomenon appears to be relatively specific to depression, although it has also been associated with posttraumatic stress disorder (PTSD) and acute stress disorder (ASD; see Moore & Zoellner, 2007, for a review).

Reduced AMS has been proposed to be a trait-like risk factor for depression (Williams et al., 2007). Consistent with this notion, reduced AMS has been found to predict increases in depressive symptoms over time in nonclinical samples (e.g., van Minnen, Wessel, Verhaak, & Smeenk, 2005) and a slightly but significantly worse course of depressive disorder (see Sumner, Griffith, & Mineka, 2010, for a meta-analysis). Furthermore, some (but not all) studies have found reduced AMS in individuals in remission from depression compared to nondepressed controls (e.g., Mackinger, Pachinger, Leibetseder, & Fartacek, 2000), thereby suggesting that reduced AMS is not merely a correlate of depressive symptoms.

Mechanisms Underlying AMS: The CaR–FA–X Model

There is evidence of associations between reduced AMS and each of the CaR–FA–X mechanisms. Our review of the extant literature (Sumner, 2012) found greatest support for associations between reduced AMS and analytical, evaluative ruminative processing, as well as impaired executive control (especially deficits in inhibition and updating and maintaining information in working memory). There also was evidence for reduced AMS as a cognitive avoidance strategy, although there was less support for early trauma playing an important role in the functional avoidance mechanism. One limitation of the literature on the CaR–FA–X model is that each of the mechanisms has been examined for the most part in isolation. Only a few studies have considered how multiple mechanisms may simultaneously relate to reduced AMS (see Sumner, 2012, for a review). For example, Barnhofer, Crane, Spinhoven, and Williams (2007) and Sumner, Griffith, and Mineka (2011) investigated aspects of both the capture and rumination and impaired executive control mechanisms. However, there are no empirical tests of the entire CaR–FA–X model.

Aims of the Current Study

We addressed this gap in the literature by examining the extent to which these three CaR–FA–X mechanisms contribute to reduced AMS, alone or in interaction, using data from a longitudinal study of risk for emotional disorders—the Northwestern–UCLA Youth Emotion Project (YEP). The YEP included measures of the following aspects of the CaR–FA–X mechanisms: (1) rumination, specifically, the brooding factor that measures a maladaptive form of analytical, evaluative ruminative processing (CaR); (2) childhood, adolescent, and early adulthood adversity and avoidant coping (FA); and (3) verbal fluency, one component of executive control (X). In this study, we examined cross-sectional associations between AMS and the CaR–FA–X mechanisms using structural equation modeling.

Consistent with the theory of Williams et al. (2007), we hypothesized that greater brooding and avoidant coping and lower verbal fluency would be associated with reduced AMS. We also hypothesized that adversity would be positively related to avoidant coping. In accordance with the emphasis on early trauma in the development of the functional avoidance mechanism (Williams et al., 2007), we predicted that this relationship would be stronger for those who had experienced

childhood adversity as opposed to late adolescent/early adulthood adversity. In addition, adversity was predicted to relate to greater brooding and impaired executive control. Given the hypothesized relationships between adversity and different aspects of the CaR–FA–X mechanisms, we examined whether avoidant coping, brooding, and verbal fluency might mediate associations between adversity and AMS. The cross-sectional nature of our data on the CaR–FA–X mechanisms precluded a strong test of mediation but nevertheless allowed us to explore potential indirect effects.

Williams et al. (2007) posited that the three CaR–FA–X mechanisms may interact with one another in contributing to reduced AMS but specific interactions were not clearly delineated. Based on theory and findings in the literature, we hypothesized and tested three interactions among these mechanisms. First, we examined a potential interaction between capture and rumination with impaired executive control. We hypothesized that individuals who engaged in maladaptive ruminative processing (brooding) might only succumb to capture errors and exhibit reduced AMS if they were also low on executive control due to difficulties in inhibiting inappropriate overgeneral responses. Second, we tested for an interaction between functional avoidance and impaired executive control. The functional avoidance hypothesis would posit that avoidance of retrieving specific memories becomes maladaptive when it is applied inflexibly to all autobiographical memories, instead of only distressing ones (e.g., Debeer, Raes, Williams, & Hermans, 2011). Therefore, individuals who engage in cognitive avoidance and have executive control deficits (and therefore reduced cognitive flexibility) might be most likely to exhibit reduced AMS. Third, we investigated a potential interaction between adversity and cognitive avoidance to further study the functional avoidance mechanism. One explanation for failures to observe significant associations between adversity and reduced AMS (cf. Moore & Zoellner, 2007) is that the experience of adversity also may need to be accompanied by cognitive avoidance for reduced AMS to develop. A combination of early adversity and avoidant tendencies might best capture those individuals who would avoid retrieving specific memories to regulate affect.

We investigated the mechanisms underlying reduced AMS separately in participants with and without a history of major depressive disorder (MDD) at AMS assessment. There is a theoretical and empirical basis for possible differences in the predominant influence on AMS in different samples. Williams et al. (2007) described how the CaR–FA–X mechanisms may be impacted by emotional disorders (e.g., MDD), and the extent to which the CaR–FA–X processes are relevant to different populations is thought to influence their relative contributions to AMS. For example, Dalgleish, Rolfe, Golden, Dunn, and Barnard (2008) posited that affect regulation may be a key mechanism in trauma-exposed populations whereas executive control deficits may be particularly important in depressed samples given the robust impairments in executive control observed in these individuals. There is initial empirical support for these distinctions. Using a modified AMT that pitted the functional avoidance and impaired executive control mechanisms against one another, Dalgleish et al. (2007, 2008) found that the functional avoidance mechanism primarily explained reduced AMS in trauma-exposed individuals with PTSD symptoms, whereas the impaired executive control mechanism primarily contributed to reduced AMS in individuals with depressive symptoms. These studies supported the notion that the mechanisms of the CaR–FA–X model may not necessarily operate in a "one-size-fits-all" manner.

To our knowledge, this is the first study to examine these three CaR–FA–X mechanisms and AMS in individuals with and without a history of MDD. Some a priori predictions were formulated based on theory and prior findings. Given the central role that executive control plays in retrieving specific memories regardless of the presence of psychopathology (Williams et al.,

2007), we hypothesized that impaired executive control would relate to reduced AMS in those with and without a history of MDD. In addition, some researchers have posited that capture and rumination may lead to reduced AMS when cues activate highly elaborated networks corresponding to negative self-schemas (Dalgleish et al., 2003). Given the role of negative self-schemas in depression, we hypothesized that brooding (our measure of capture and rumination) might relate more strongly to reduced AMS in those with (vs. without) a history of MDD.

Method

Participants

Participants were from a larger sample of young adults in a 10-year longitudinal study (baseline plus 7 to 9 years of follow-up, depending on cohort) of risk for emotional disorders (the YEP; see Zinbarg et al., 2010). High school juniors in suburban Chicago or Los Angeles were recruited in three cohorts from 2003 to 2005. At screening, participants completed the Eysenck Personality Questionnaire Neuroticism scale (EPQ–R–N; Eysenck & Eysenck, 1975). We oversampled high EPQ–R–N scorers (those in the upper tertile) to obtain a behavioral high-risk sample for the development of emotional disorders (59% of the original sample of 627 participants were high EPQ–R–N scorers).

From 2009 to 2011, 466 participants completed an AMS assessment. Participants with a history of clinically significant bipolar disorder (n = 9), PTSD (n = 13), ASD (n = 3), major depression due to a general medical condition (n = 1), substance-induced mood disorder (n = 1), or psychotic symptoms (n = 5) were excluded from analyses. Twenty-seven individuals met one or more of these exclusion criteria, resulting in a final sample size of 439. As in the original YEP sample, participants in this subsample were predominantly female (68.3%) and racially and ethnically diverse (49.4% White, 14.4% Hispanic/Latino, 13.2% African American, 4.3% Asian American, 0.7% Pacific Islander, 12.5% multiracial, and 5.5% other). Mean age at AMS assessment was 22.4 years (SD = 0.9, range = 20–25).

We compared associations between reduced AMS and measures of the CaR–FA–X mechanisms in two groups: individuals with (n = 164) and without (n = 275) a history of MDD at the AMT. We focused on those with a history of MDD given that only six participants were in a current major depressive episode at the AMT. Table 1 presents participant characteristics as a function of MDD history. Differences between participants with and without a history of MDD were generally consistent with those observed in the broader depression literature (e.g., Gotlib & Hammen, 2009). For example, compared to those without a history of MDD, participants with a history of MDD had higher neuroticism based on responses to the EPQ–R–N questionnaire administered at screening. Participants with a history of MDD were also more likely than those without a history of MDD to report a current or past history of psychiatric comorbidity (both in terms of anxiety disorders and alcohol and/or nonalcohol substance use disorders). Furthermore, there was a trend for a higher percentage of females in the group with, compared to those without, a history of MDD. The two groups did not differ significantly on socioeconomic status at baseline based on the Hollingshead index of social position (Hollingshead, 1975), on the percentage of White participants, or on age at the AMT.

Table 1. Participant Characteristics for Individuals With and Without a History of MDD at the AMT

Characteristic	History of MDD % (n)	No History of MDD %(n)	p value
Female gender	74 (121)	65 (179)	.06
White race/ethnicity	61 (100)	58 (159)	.52
History of anxiety disorders at the AMT[a]	38 (62)	14 (38)	<.001
History of substance abuse disorders at the AMT[b]	29 (47)	18 (49)	.01
	M (SD)	M (SD)	p value
EPQ–R–N total score at screening	14.3 (3.7)	11.1 (4.4)	<.001
Hollingshead SES index at baseline[c]	48.5 (13.2)	48.9 (12.2)	.77
Age at the AMT	22.5 (0.9)	22.4 (0.9)	.21

Note. History of MDD: n 164; no history of MDD: n 275. MDD major depressive disorder; AMT Autobiographical Memory Test; EPQ–R–N Eysenck Personality Questionnaire Neuroticism scale; SES socioeconomic status.

[a] Anxiety disorders assessed included generalized anxiety disorder, obsessive– compulsive disorder, social anxiety disorder, panic disorder, and agoraphobia without panic.

[b] Substance abuse disorders assessed included alcohol and nonalcohol abuse and dependence.

[c] Mean Hollingshead index scores for both groups corresponded to minor professionals (e.g., insurance agents, sales representatives), workers at medium-sized businesses, and technical workers (Hollingshead, 1975).

Materials and Tasks

RRS. The Ruminative Responses Scale (RRS) of the Response Style Questionnaire (Nolen-Hoeksema & Morrow, 1991) is a well-validated 22-item measure that assesses the tendency to respond to depressed mood by focusing on oneself, one's symptoms, and the causes and consequences of depressed mood. Ratings are made on a 1 (almost never) to 4 (always) scale. The current study used the five-item brooding subscale to measure analytical, evaluative ruminative processing (see Treynor et al., 2003). This subscale had good internal consistency ($\alpha = .85$)

COPE Inventory. The COPE (Carver, Scheier, & Weintraub, 1989) is a well-validated 53-item coping inventory that was used to measure avoidant coping in the current study. It comprises 14 scales assessing different coping dimensions, including denial, behavioral disengagement, and mental disengagement (see Carver et al., 1989). A version with eight additional items on emotional processing and expression (Stanton, Kirk, Cameron, & Danoff-Burg, 2000) was used. When completing this measure, participants described their "most stressful or traumatic" experience in the past year and indicated whether they used different strategies to deal with it on a 1 (I don't do this at all) to 4 (I do this a lot) scale. Research suggests that the denial, behavioral disengagement, and mental disengagement subscales (each with four items) load on a single Disengagement factor (Carver et al., 1989). We used these subscales to measure avoidant coping. These items had good internal consistency ($\alpha = .87$).

AMT. The Autobiographical Memory Test (AMT; Williams & Broadbent, 1986) uses a cuing methodology to elicit autobiographical memories. The AMT was administered twice, approximately 9 months and 5 years after the baseline assessment (see Griffith et al., 2009; Sumner, Griffith, Mineka, Rekart, et al., 2011, for published data from the first AMT when participants were 16–18 years of age, Sumner, Vrshek-Schallhorn et al., 2013 for published data from the second AMT; Sumner, Mineka et al., 2013, for published data from the first and second AMTs). A subsample of 333 participants was invited to complete the first AMT, whereas all

remaining participants were invited to complete the second AMT. The current study used data from the second AMT (N = 439). Compared to data from the first AMT, data from the second AMT provided both a larger sample size for analysis and a measure of AMS that was collected close in time to, rather than several years before, the measures of the CaR–FA–X mechanisms.

Participants were instructed to retrieve a specific autobiographical memory in response to cue words and had 30 s to respond on each trial. Up to seven practice items were administered; participants had to retrieve two consecutive specific memories or complete all practice trials before proceeding to the test trials. Feedback was given only on practice items. There were 12 test trials alternating between positive (safe, ambitious, peace, hope, brave, interested) and negative (disappoint, inferior, hurt, frustrated, tense, regret) cues. Cues were selected from word sets used in previous studies and from the MRC Psycholinguistic Database (Coltheart, 1981), and positive and negative cue words were matched on concreteness, imageability, usage frequency, and familiarity. Responses were made orally and audio-recorded. Responses were scored as specific memories (events that occurred at a particular time and place and lasted less than 1 day), extended memories (events lasting more than 1 day), categorical memories (summaries/classes of events), semantic associates (semantic information but no personal memory), or omissions (no response). Interrater reliability of this scoring approach was good overall; mean kappas for within-site (n = 46) and cross-site (n = 46) reliability were .77 for the second AMT. We used dichotomous variables (specific vs. nonspecific responses) as measures of AMS (see Griffith et al., 2009, for rationale). These responses had acceptable internal consistency ($\alpha = .69$).

COWAT. The Controlled Oral Word Association Test (COWAT; e.g., Strauss, Sherman, & Spreen, 2006) is a measure of verbal fluency and has been widely used as a classic executive control measure (e.g., Dalgleish et al., 2007; Swan & Carmelli, 2002). The task assesses several aspects of executive control, including organization, initiation, maintenance, and interference control. It requires the ability to organize verbal retrieval, initiate and maintain a verbal search set, and inhibit inappropriate responses. In three 60-s trials, participants were asked to generate as many words as possible starting with the letter F on the first trial, A on the second, and S on the third. We used the total numbers of correct responses for each of the three trials as measures of executive control. These totals had high internal consistency ($\alpha = .83$).

CTI. The Childhood Trauma Interview (CTI; Fink, Bernstein, Handelsman, Foote, & Lovejoy, 1995) is a semistructured interview assessing adversity in six domains of early experience: separations and losses; neglect; emotional abuse or assault; physical abuse or assault; witnessing violence; and sexual abuse or assault. The interview evaluated the severity, frequency, and duration of different adverse experiences from birth through age 16. Interviewers rated each adversity for severity on a 1 (minimal/mild) to 6 (very extreme/sadistic) scale. In addition, interviewers scored the frequency of each adversity using a 1 (once or a few times over a number of years) to 6 (at least daily) scale. The duration of the adversity, participant age at the start and end of the experience, and type of perpetrator also were documented.

Given little consensus in the literature on how to calculate CTI summary scores, we developed a new scoring approach for the YEP (see Vrshek-Schallhorn et al., 2014, for details). We created separate indexes for (1) minor and relatively more severe adversity and (2) adversity during childhood versus adolescence. Based on qualitative aspects of the different severity levels and on severity score distributions, we characterized severity scores of 1 to 2 as minor adversities and scores of 3 to 6 as major adversities. Count variables for the numbers of minor and major adversities were calculated and summed across the six CTI domains; this was done separately for early/middle childhood (ages 0 to 9 years) and preadolescence/adolescence (9 to 16 years). This

produced four aggregate variables: minor and major adversity in childhood and minor and major adversity in adolescence. These aggregate indexes had good interrater reliability. Intraclass correlation coefficients (ICCs; two-way random effects model, absolute agreement) for the four aggregate variables ranged from .82 to .92 for within-site reliability (n = 47) and from .72 to .94 for cross-site reliability (n = 47). We used aggregate scores for major adversity in childhood and/or adolescence as measures of early adversity. Only major adversities were used to capture adverse experiences because many of the minor adversities were quite normative (e.g., being home alone as a teenager for a few hours, quarreling between siblings).

LSI. Whereas the CTI was used as a measure of adversity in childhood/early adolescence, the Life Stress Interview (LSI) was used as a measure of adverse experiences in late adolescence/early adulthood. The LSI (Hammen, 1991) is a semistructured interview assessing stress in the past 12 months in 10 domains: close friendship, social life, romantic relationships, family relationships, neighborhood, school, work, finances, personal health, and family members' health. Interviewers rated chronic stress in each domain on a 1 (superior conditions) to 5 (exceptionally poor conditions) scale. Independent teams of two or more LSI-trained individuals rated episodic stressors for threat severity on a 1 (minimal/no negative impact) to 5 (severe negative impact) scale based on only objective information about episodic stressors (e.g., duration and impact of the event).

In this study, information on episodic stressors represented adversity experienced during late adolescence/early adulthood, specifically from the age of 16 (the period assessed at the YEP baseline) until the second AMT. Threat severity ratings ≥ 2.5 (indicating moderate-to-severe stress) were summed for each yearly LSI from baseline until the AMT administered approximately 3 to 7 years later (M = 5.38 years, SD = 0.78). As noted earlier, moderate-to-severe episodic stressors were selected to capture adversity because many of the lower severity stressors were quite minor (e.g., moving within the same city, starting an advanced degree program). Interrater reliability for episodic stress ratings in the YEP has been adequate. ICCs (two-way random effects model, absolute agreement) for threat severity ratings ranged from .69 to .76 for the LSIs administered at baseline through the fourth annual follow-up assessment.

SCID. The Structured Clinical Interview for DSM–IV (SCID; First, Spitzer, Gibbon, & Williams, 2002) was used to assign Axis I diagnoses at baseline and annual follow-up assessments. SCID data were utilized to classify participants as those with or without lifetime history of MDD at the second AMT. Kappas and adjusted kappas for clinically significant MDD diagnoses for the baseline and first four annual follow-up assessments ranged from .56 to .83 and .84 to .94, respectively.

Procedure

At the baseline assessment between 2003 and 2005, participants completed a lifetime SCID and LSI for the past 12 months. Trained advanced graduate students and Bachelor's-level research assistants administered the SCID and LSI at baseline and annual follow-up interviews. Follow-up interviews assessed Axis I psychopathology and life stress since the previous interview. Starting approximately 9 months after baseline, a random subsample (n = 333) completed the AMT. Between 2009 and 2011, the AMT was re-administered, along with the COWAT, CTI, RRS, and COPE. All remaining participants were eligible for these measures. Trained undergraduates, graduate students, and Bachelor's-level research assistants administered the AMT, COWAT, and CTI via phone (these tasks were administered during the same assessment for most cases). The

AMT was always administered prior to the COWAT, which was always administered prior to the CTI. The RRS and COPE were completed online via personal computers at the participants' convenience. The present study used RRS and COPE data that were provided within 3 months of the AMT. Of the 439 AMT completers included in the present study, 332 completed the RRS, 329 completed the COPE, 439 completed the COWAT, and 424 completed the CTI. There were no significant differences between AMT completers who did and did not complete these other measures on gender, race/ethnicity, history of MDD, or AMS, all ps > .05.

Analytic Approach

Structural equation modeling (SEM) was used to test the CaR–FA–X model using the Mplus software (version 6.11; Muthén & Muthén, 1998–2010). Missing data were accommodated using full information maximum likelihood. First, measurement models for brooding, avoidant coping, verbal fluency, and AMS latent variables were estimated for those with and without a history of MDD at AMS assessment. As described below, we attempted to use a causal indicator measurement model for the childhood/early adolescent and late adolescent/early adulthood adversity latent variables, whereby history of adversity was defined by the experiences represented by these indicators (Bollen & Bauldry, 2011). Causal indicator coefficients are structural coefficients; thus, measurement models for the adversity latent variables were not examined. We assessed measurement invariance in participants with and without a history of MDD using multiple group comparison to ensure that latent constructs were being measured in a similar way in these groups. Evidence of at least configural invariance (equivalence in basic factor structure) was required before comparing structural models (Horn & McArdle, 1992). If configural invariance was supported, then we tested for metric invariance (equivalence in factor loadings across groups). Chi-square ($\chi 2$) difference tests were calculated to test whether the fits of these nested models differed significantly.

We used the comparative fit index (CFI), root mean square error of approximation (RMSEA), and standardized root-mean-square residual (SRMR) fit indexes to evaluate models with continuous indicators. As per Hu and Bentler (1999), the following cutoffs were used to assess fit: CFI ≥ .95, RMSEA ≤ .06, and SRMR ≤ .08. A robust (mean- and variance-adjusted) method of weighted least squares estimation (WLSMV; Muthén & Muthén, 1998–2010) was used for models with categorical indicators. For these models, CFI, RMSEA, and weighted root-mean-square residual (WRMR) were used to evaluate fit. WRMR values ≤ .90 were used as a cutoff (Yu, 2002).

After assessing measurement invariance, structural models with mechanism main effects and the hypothesized interactions were examined separately for those with and without a history of MDD. Interaction latent variables were defined by the product of indicators of the mechanism latent variables in the interaction and were calculated with a maximum likelihood estimator with robust standard errors using a numerical integration algorithm (Muthén & Muthén, 1998–2010). This method produces the Akaike information criterion (AIC) and sample-size adjusted Bayesian information criterion (SABIC) fit statistics. These statistics are used to compare models, with smaller values indicating better fit.

Table 2. Descriptive Statistics for the CaR–FA–X Mechanism and AMS Latent Variable Indicators

Latent variable indicator	History of MDD	No history of MDD	Range in full sample
Brooding			
RRS5 "What I am doing to deserve this?"	1.87 (0.97)	1.58 (0.77)	1–4
RRS10 "Why do I always react this way?"	2.11 (1.02)*	1.57 (0.77)	1–4
RRS13 "Wish a situation had gone better"	2.60 (0.84)*	2.11 (0.86)	1–4
RRS15 "Why do I have these problems?"	1.98 (0.98)*	1.58 (0.82)	1–4
RRS16 "Why can't I handle things better?"	2.35 (0.99)*	1.72 (0.79)	1–4
Avoidant coping			
C4 "Admit to myself that I can't deal with it"	1.88 (0.92)	1.59 (0.77)	1–4
C11 "Act as though it hasn't happened"	1.68 (0.91)	1.58 (0.79)	1–4
C13 "Say 'this isn't real'"	1.52 (0.86)	1.39 (0.71)	1–4
C15 "Pretend it hasn't happened"	1.47 (0.79)	1.44 (0.74)	1–4
C26 "Refuse to believe it has happened"	1.31 (0.66)	1.36 (0.67)	1–4
C29 "Daydream about other things"	2.32 (1.08)	2.12 (0.95)	1–4
C30 "Give up trying to deal with it"	1.66 (0.90)	1.55 (0.80)	1–4
C31 "Do something else"	2.51 (1.04)	2.34 (0.97)	1–4
C33 "Give up the attempt to cope"	1.51 (0.81)	1.51 (0.79)	1–4
C34 "Sleep more than usual"	1.78 (1.03)	1.60 (0.84)	1–4
C38 "Reduce effort put into dealing with it"	1.84 (0.88)	1.75 (0.87)	1–4
C39 "Turn to work/other activities"	2.54 (1.02)	2.42 (0.91)	1–4
Childhood/early adolescent adversity			
Number of major events in childhood	1.76 (2.57)	1.19 (2.12)	0–16
Number of major events in early adolescence	3.36 (3.78)*	1.88 (2.60)	0–20
Late adolescent/early adulthood adversity			
Total severity for SLEs at baseline[a]	2.49 (3.54)*	1.26 (2.28)	0–16.5
Total severity for SLEs at 1-year follow-up	1.96 (2.73)	1.31 (2.33)	0–17.5
Total severity for SLEs at 2-year follow-up	2.24 (3.18)	1.54 (2.76)	0–20.5
Total severity for SLEs at 3-year follow-up	2.78 (3.65)*	1.53 (2.44)	0–17.0
Total severity for SLEs at 4-year follow-up	2.98 (3.66)*	1.50 (2.48)	0–18.0
Total severity for SLEs at 5-year follow-up	2.25 (3.16)*	1.08 (1.86)	0–15.5
Total severity for SLEs at 6-year follow-up	1.94 (2.86)	1.05 (1.93)	0–11.5
Total severity for SLEs at 7-year follow-up	0.78 (1.21)	0.66 (1.32)	0–5.0
Verbal fluency			
COWAT F trial total correct	13.51 (4.30)	12.76 (4.46)	3–30
COWAT A trial total correct	11.24 (3.71)	11.14 (4.08)	2–23
COWAT S trial total correct	15.08 (4.12)	14.45 (4.49)	3–27
AMS			
AMT Trial 1 Safe	0.71 (0.46)	0.66 (0.47)	0–1[b]
AMT Trial 2 Disappoint	0.86 (0.35)	0.79 (0.41)	0–1
AMT Trial 3 Ambitious	0.67 (0.47)	0.68 (0.47)	0–1
AMT Trial 4 Inferior	0.66 (0.47)	0.53 (0.50)	0–1
AMT Trial 5 Peace	0.74 (0.44)	0.69 (0.47)	0–1
AMT Trial 6 Polite	0.76 (0.43)	0.78 (0.41)	0–1
AMT Trial 7 Hope	0.69 (0.46)	0.66 (0.48)	0–1
AMT Trial 8 Frustrated	0.70 (0.46)	0.73 (0.44)	0–1
AMT Trial 9 Brave	0.64 (0.48)	0.71 (0.46)	0–1
AMT Trial 10 Tense	0.70 (0.46)	0.73 (0.45)	0–1
AMT Trial 11 Interested	0.78 (0.42)	0.77 (0.42)	0–1
AMT Trial 12 Regret	0.60 (0.49)	0.61 (0.49)	0–1

Note. Means and standard deviations (in parentheses) are presented. CaR capture and rumination; FA functional avoidance; X impaired executive control; AMS Autobiographical Memory Specificity; MDDmajor depressive disorder; SLE stressful life event; COWAT Controlled Oral Word Association Test; AMT Autobiographical Memory Test.

a SLEs stressful life events of severity ratings 2.5 on the Life Stress Interview. b Nonspecific memories were coded as 0 and specific memories were coded as 1. Significant difference between those with and without a history of MDD, p ——— .001 (Bonferroni correction).

Results

Descriptive Statistics

Descriptive statistics for indicators of the brooding, avoidant coping, childhood/early adolescent adversity, late adolescent/early adulthood adversity, avoidant coping, verbal fluency, and AMS latent variables are presented in Table 2. On average, participants reported engaging only infrequently in most brooding and avoidant coping items. Most participants also reported relatively few major adversities during childhood/early adolescence and late adolescence/early adulthood. In addition, participants were generally more likely to retrieve specific than nonspecific memories on the AMT. Compared to those without a history of MDD, individuals with a history of MDD endorsed significantly higher levels of brooding and adversity.

Latent variable correlations among the CaR–FA–X mechanism and AMS variables (with some exceptions) are displayed in Table 3. Identification of causal indicator latent variables based on causal indicators alone is not possible because causal indicators influence the latent variable rather than vice versa (Bollen & Bauldry, 2011); these latent variables were not examined until structural models were tested. Therefore, for the adversity variables, the total number of major adverse experiences in childhood/early adolescence and total severity score for moderate-to-severe adverse experiences in late adolescence/early adulthood were used as proxies for the latent variables for these correlations. In all participants, childhood/early adolescent and late adolescent/early adulthood adversity were significantly positively correlated, as were brooding and avoidant coping. In addition, AMS was significantly positively correlated with verbal fluency. The following correlational patterns were different for those with and without a history of MDD. For those without a history of MDD, brooding was significantly positively correlated with the adversity measures, and AMS was significantly negatively correlated with late adolescent/early adulthood adversity. For those with a history of MDD, AMS was significantly positively correlated with brooding, and childhood/early adolescent adversity was significantly positively correlated with avoidant coping.

Table 3. Correlations Between the CaR–FA–X Mechanism and AMS Variables in Participants With and Without a History of MDD at the AMT

Variable	1	2	3	4	5	6
1. Brooding	—	.17*	.13*	.37***	.03	-.10
2. Childhood/early adolescent adversity	-.002	—	.31***	.07	-.09	-.09
3. Late adolescent/early adulthood adversity	-.09	.48***	—	-.003	-.10	-.19**
4. Avoidant coping	.53***	.18*	.03	—	-.11	-.05
5. Verbal fluency	-.08	-.04	-.05	-.15	—	.27***
6. AMS	.30**	-.04	-.10	-.02	.18*	—

Note. History of MDD: n = 164; no history of MDD: n = 275. Correlations below the diagonal correspond to those with a history of MDD and correlations above the diagonal correspond to those without a history of MDD. CaR capture and rumination FA functional avoidance; X impaired executive control; AMS Autobiographical Memory Specificity; MDD major depressive disorder. AMT Autobiographical Memory Test.

*p < .05. **p < .01. *** p < .001.

Table 4. Fit Statistics for Measurement Models for Latent Variables and for Testing Measurement Invariance in Participants With and Without a History of MDD at the AMT

	History of MDD					No history of MDD				
Latent variable	x^2 *(df)*	CFI	RMSEA	SRMR[a]	WRMR	x^2 *(df)*	CFI	RMSEA	SRMR[a]	WRMR
Brooding	6.93(4)	.99	08 [.00, .17]	.03	—	10.27(5)	.99	.07 [.00, .14]	.03	—
Avoidant coping	77.20(39)	.93	.09 [.06, .12]	.05	—	68.37(39)	.97	.06 [.04, .09]	.04	—
Verbal fluency	0.00(0)	1.00	.00 [.00, .00]	.00	—	0.00(0)	1.00	.00 [.00, .00]	.00	—
AMS	55.36(54)	.99	.01 [.00, .05]	—	0.73	52.43(54)	1.00	.00 [.00, .04]	—	0.70
	Configural invariance model					Metric invariance model				
Latent variable	x^2 *(df)*	CFI	RMSEA	SRMR[a]	WRMR	x^2 *(df)*	CFI	RMSEA	SRMR[a]	WRMR
Brooding	17.19(9)	.99	.07 [.01,.13]	.03	—	22.31(14)	.99	.06 [.00, .10]	.08	—
Avoidant coping	145.57(78)	.95	.07 [.05, .09]	.05	—	166.39(89)	.95	.07 [.06, .09]	.06	—
Verbal fluency	0.00(0)	1.00	.00 [.00, .00]	.00	—	5.73(3)	1.00	.06 [.00, .14]	.08	—
AMS	107.90(108)	1.00	.00 [.00, .04]	—	1.01	122.90(120)	1.00	.01 [.00, .04]	—	1.13

Note. MDD major depressive disorder; AMT Autobiographical Memory Test; df degrees of freedom; CFI comparative fit index; RMSEA root mean square error of approximation; SRMR standardized root mean square residual; WRMR weighted root-mean-square residual; AMS Autobiographical Memory Specificity.
a The SRMR statistic was used for the brooding, avoidant coping, and verbal fluency models with continuous indicators and the WRMR statistic was used for the AMS models with dichotomous indicators.

Final measurement models included correlated residuals suggested by modification indexes if there was a strong substantive or empirical rationale for allowing these residual variance terms to covary. In those with a history of MDD, residuals were correlated for (1) brooding: RRS5 ("Think 'What am I doing to deserve this?'") and RRS15 ("Think 'Why do I have problems other people don't have?'"); (2) avoidant coping: C38 ("I reduce the amount of effort I'm putting into dealing with it") and C39 ("I turn to work or other activities to take my mind off things"); and (3) avoidant coping: C26 ("I refuse to believe that it has happened") and C33 ("I give up the attempt to cope"). In those without a history of MDD, residuals were correlated for (1) avoidant coping: C38 ("I reduce the amount of effort I'm putting into dealing with it") and C39 ("I turn to work or other activities to take my mind off things"); and (2) avoidant coping: C31 ("I do something to think about it less") and C39 ("I turn to work or other activities to take my mind off things").

There was evidence for metric invariance in the measurement of brooding, verbal fluency, and AMS across participants with and without a history of MDD (see Table 4). The chi-square difference tests comparing the configural and metric invariance models for these latent variables were nonsignificant, ps > .40. Thus, constraining the factor loadings to be equal across groups did not significantly deteriorate model fit. There was evidence for partial metric invariance in the measurement of avoidant coping in these two groups. The metric invariance model for avoidant coping had a significantly worse fit than the configural invariance model, $\chi 2(11, N = 329) = 20.82$, $p = .04$. However, only one parameter estimate in the configural invariance model differed significantly between participants with and without a history of MDD (specifically, the loading of item C31 on the mental disengagement factor). A model constraining all factor loadings to be equal across groups except for this loading had, overall, a good fit: $\chi 2(88, N = 329) = 162.32$, $p < .001$, CFI = .95, RMSEA = .07, 90% CI [.05, .09], and SRMR = .06. This partial metric invariance model was not associated with a significantly worse model fit compared to the configural invariance model, $\chi 2(10, N = 329) = 17.65$, $p = .06$. Given that 13 of 14 factor loadings could be constrained to be equal across groups, there was substantial evidence that the measurement of avoidant coping was highly similar in participants with and without a history of MDD. In sum, for each latent variable of interest, the different constructs were being measured similarly in participants with and without a history of MDD.

Structural Models of the CaR–FA–X Mechanisms and AMS

As described earlier, models of main effects of, and interactions among, the CaR–FA–X mechanisms on AMS were examined using SEM separately in participants with and without a history of MDD at AMS assessment. A structural model with direct associations between the AMS latent variable and (1) brooding, (2) avoidant coping, and (3) verbal fluency latent variables was tested first (see Figure 1). Consistent with Williams et al.'s (2007) general approach, the brooding, avoidant coping, and verbal fluency latent variables were allowed to correlate. Associations also were modeled between the childhood/early adolescent adversity latent variable (defined using a causal indicator model) and the avoidant coping, brooding, and verbal fluency latent variables. Paths between eight late adolescent/early adulthood adversity indicators (the total severity scores for the baseline and follow-up LSIs) and the avoidant coping, brooding, and verbal fluency latent variables were included as well. By modeling indicator variables—rather than a latent variable—for late adolescent/early adulthood adversity, a partially reduced form model for causal indicators was employed (Bollen & Davis, 2009). We adopted this approach because the model was not empirically identified when a late adolescent/early adulthood adversity latent variable was

specified. After examining main effects, we tested the three hypothesized interactions between the mechanism latent variables within this overall model.

Figure 1. Model with main effects of the CaR–FA–X mechanisms on autobiographical memory specificity. Ovals represent latent variables. A single rectangle represents the eight indicators of late adolescent/early adulthood adversity to simplify model presentation. Indicators of latent variables are omitted to simplify model presentation as well. Adol = adolescent.

Mechanisms Underlying AMS in Participants With a History of MDD

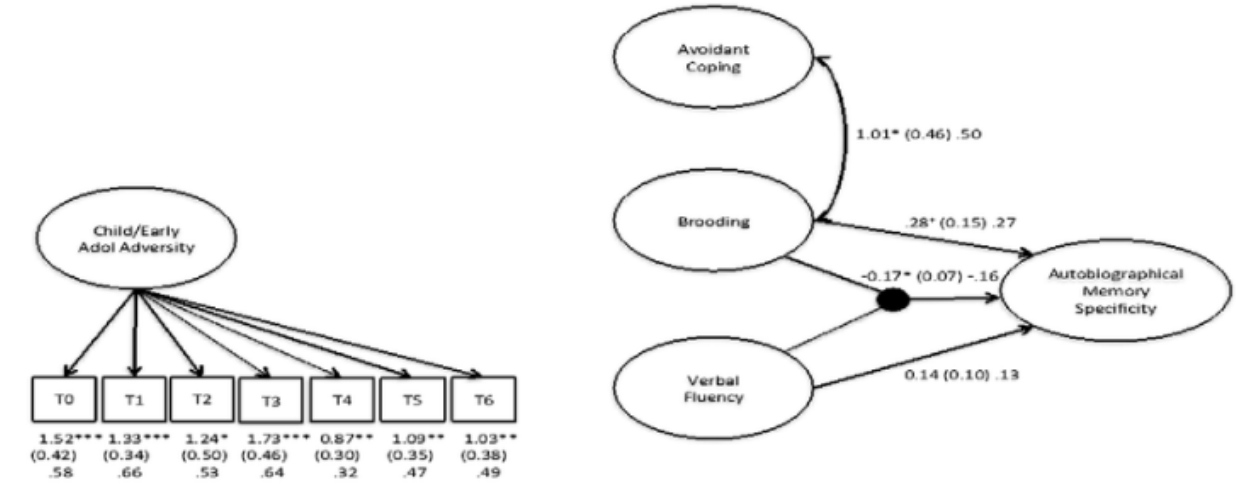
The main effects model had an excellent fit in participants with a history of MDD at AMS assessment: $\chi 2(740, N = 164) = 763.14$, $p = .27$, CFI = .96, RMSEA = .01, 90% CI [.00, .03], and WRMR = .82. Childhood/early adolescent and late adolescent/early adulthood adversity were significantly positively associated (bs > 0.70, ps < .001; only the path to the indicator for the seventh follow-up interview was nonsignificant, $b = -0.03$, $p = .85$). Childhood/early adolescent adversity also was significantly positively associated with avoidant coping ($b = 0.33$, $p = .03$). Verbal fluency ($b = 0.20$, $p = .03$) and brooding ($b = 0.49$, $p = .004$) were significantly associated with AMS, with lower verbal fluency and lower brooding both being associated with reduced AMS. Consistent with the notion that brooding is an avoidance strategy (e.g., Moulds, Kandris, Starr, & Wong, 2007), avoidant coping and brooding were significantly positively correlated ($r = .56$, $p < .001$). There was no regular pattern of significant associations between the late adolescent/early adulthood adversity indicators and the avoidant coping, brooding, and verbal fluency latent variables. Furthermore, constraining these paths to zero was not associated with a significant decrement in model fit, ps > .20. Neither the Avoidant Coping × Verbal Fluency nor Childhood/Early Adolescent Adversity × Avoidant Coping latent variable interaction was significant, ps > .43. However, the Brooding × Verbal Fluency interaction ($b = -0.19$) was associated with a significant improvement in model fit, $\chi 2(1, N = 164) = 4.71$, $p = .03$ (model with the latent variable Brooding × Verbal Fluency interaction: AIC = 15,970.68, SABIC = 15,961.50; model without this interaction: AIC = 15,971.34, SABIC = 15,962.22). This finding suggested the importance of modeling an interaction between these two CaR–FA–X mechanisms.

The final model of relationships between the CaR–FA–X mechanisms and AMS in participants with a history of MDD was determined by deleting nonsignificant paths from the model with the Brooding × Verbal Fluency latent variable interaction (see Figure 2a). Deleting nonsignificant paths was not associated with a significant decrement in model fit, $\chi 2(6, N = 164) = 10.14$, $p = .12$. In this model, childhood/early adolescent and late adolescent/early adulthood adversity were significantly positively related, as were brooding and avoidant coping. The adversity measures were not significantly associated with any of the CaR–FA–X mechanism latent variables, which thus did not support the notion that aspects of the CaR–FA–X model mediated an association between adversity and reduced AMS. Furthermore, the Brooding × Verbal Fluency interaction was significantly related to AMS. At the mean of brooding, a 1 standard deviation increase in verbal fluency was associated with a 0.13 standard deviation increase in AMS. At 1 standard deviation below the mean of brooding, a 1 standard deviation increase in verbal fluency was associated with a 0.29 standard deviation increase in AMS. At 1 standard deviation above the mean of brooding, a 1 standard deviation increase in verbal fluency was associated with a 0.03 standard deviation decrease in AMS. In other words, for individuals with a history of MDD, verbal fluency was most strongly related to AMS at low levels of brooding, with greater verbal fluency associated with greater AMS. However, at high levels of brooding, the relationship between verbal fluency and AMS was negligible (see Figure 3a).

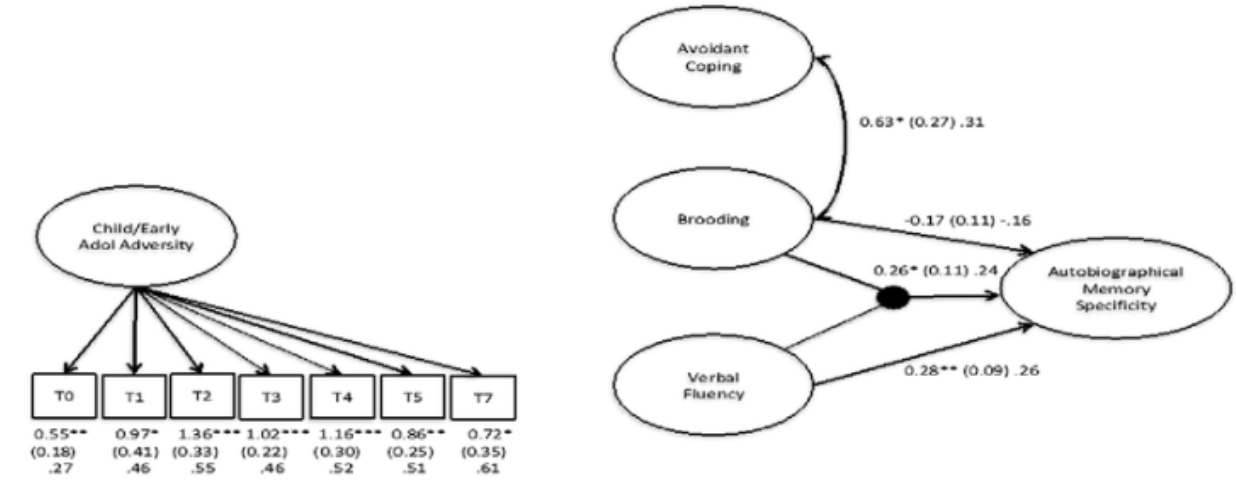
Mechanisms Underlying AMS in Participants Without a History of MDD

Overall, the main effects model had a good fit in those without a history of MDD at AMS assessment: $\chi 2(741, N = 275) = 849.06$, $p = .004$, CFI = .90, RMSEA = .02, 90% CI [.01, .03], and WRMR = .91. Childhood/early adolescent and late adolescent/early adulthood adversity were significantly positively associated (bs > 0.62, ps < .02). In addition, greater childhood/early adolescent adversity was significantly associated with greater brooding ($b = 0.23$, $p = .01$) and lower verbal fluency ($b = −0.20$, $p = .01$). Verbal fluency ($b = 0.34$, $p < .001$) and brooding ($b = −0.20$, $p = .05$) were significantly associated with AMS, with lower verbal fluency and greater brooding associated with reduced AMS. In addition, avoidant coping and brooding were significantly positively correlated ($r = .36$, $p < .001$). As in those with a history of MDD, no consistent pattern of significant associations emerged between the late adolescent/early adulthood adversity indicators and avoidant coping, brooding, and verbal fluency latent variables. Constraining these paths to zero was not associated with a significant decrement in model fit, ps > .20. Furthermore, the Brooding × Verbal Fluency interaction in participants without a history of MDD ($b = 0.27$) was associated with a significant improvement in model fit, $\chi 2(1, N = 275) = 10.20$, $p = .001$ (model with this latent variable interaction: AIC = 24,186.30, SABIC = 24,248.28; model without the interaction: AIC = 24,190.62, SABIC = 24,252.16). Again, this finding suggested the importance of modeling an interaction between these two CaR–FA–X mechanisms. None of the other interactions was significant (ps > .37)

We deleted nonsignificant paths from the model with the Brooding × Verbal Fluency latent variable interaction to obtain the final model for participants without a history of MDD (see Figure 2b). Deleting nonsignificant paths was not associated with a significant decrement in model fit, $\chi 2(6, N = 275) = 9.25$, $p = .16$. In this model, childhood/early adolescent and late adolescent/early adulthood adversity were significantly positively associated, as were brooding and avoidant coping. The adversity measures were not significantly associated with any of the CaR–FA–X mechanism latent variables, which thus did not support the notion that aspects of the CaR–FA–X



a) Participants with a history of MDD



b) Participants without a history of MDD

Figure 2. Final models of relationships between the CaR–FA–X mechanisms and autobiographical memory specificity, with nonsignificant relationships removed. Unstandardized parameter estimates are presented first, followed by their standard errors in parentheses, followed by standardized parameter estimates. The Brooding × Verbal Fluency latent variable interaction is represented by the small black circle. Asterisks denoting statistical significance are based on unstandardized parameter estimates. T0 to T7 represent the total severity of late adolescent/early adulthood adversity from the Life Stress Interviews administered at baseline through the seventh annual follow-up assessment. (a) Participants with a history of major depressive disorder (MDD). (b) Participants without a history of MDD. CaR = capture and rumination FA = functional avoidance; X = impaired executive control; Adol = adolescent; T = time (Time 0 (T0) = baseline assessment, T1 = first annual follow-up assessment, etc).
+p < .10. * p < .05. ** p < .01. *** p < .001.

model mediated an association between adversity and reduced AMS. Lower verbal fluency and the Brooding × Verbal Fluency interaction were significantly associated with reduced AMS. At the mean of brooding, a 1 standard deviation increase in verbal fluency was associated with a 0.26 standard deviation increase in AMS. At 1 standard deviation below the mean of brooding, a 1 standard deviation increase in verbal fluency was associated with a 0.02 standard deviation increase in AMS. At 1 standard deviation above the mean of brooding, a 1 standard deviation increase in verbal fluency was associated with a 0.50 standard deviation increase in AMS. In sum, for individuals without a history of MDD, verbal fluency was most strongly related to AMS at high levels of brooding, with greater verbal fluency being associated with greater AMS. However, at low levels of brooding, the relationship between verbal fluency and AMS was negligible (see Figure 3b).

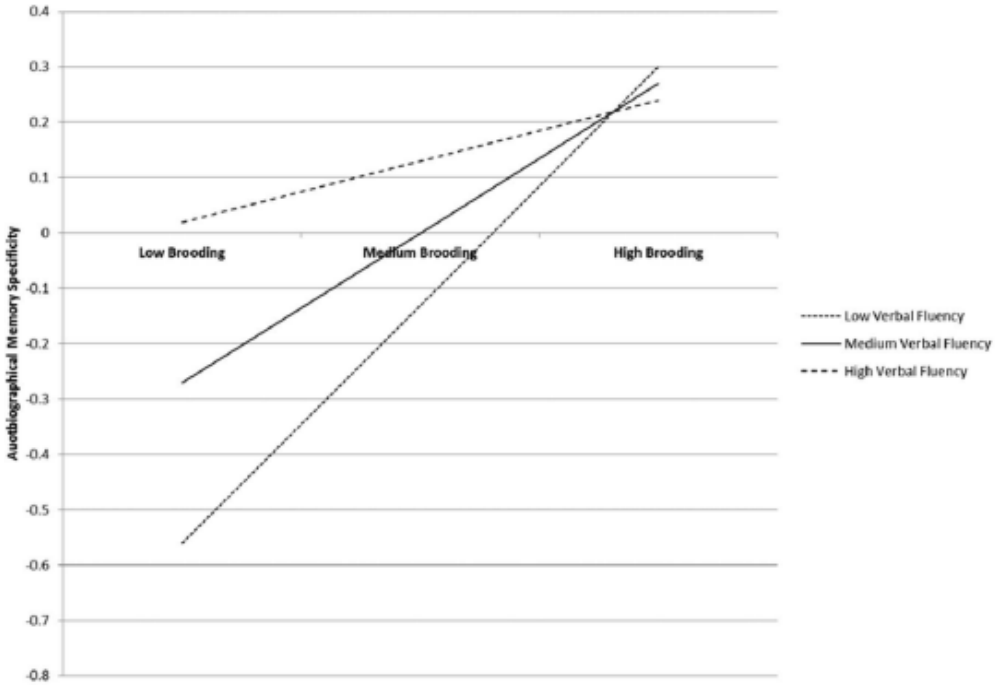
Furthermore, the Brooding × Verbal Fluency–AMS relationships differed significantly in participants with and without a history of MDD; the 95% CI for the difference between the two parameter estimates [0.17, 0.69] did not contain zero. The brooding–AMS relationship estimates also differed significantly between the two groups, 95% CI [−0.94, −0.18], although the verbal fluency-AMS relationship estimates did not, 95% CI [−0.19, 0.31]. Thus, although the same mechanism variables related to AMS in participants with and without a history of MDD, there were some differences in the nature of those relationships. Attempts to use a multiple group comparison to test for differences between the models for those with and without a history of MDD did not converge due to high complexity and low covariance coverage.

Discussion

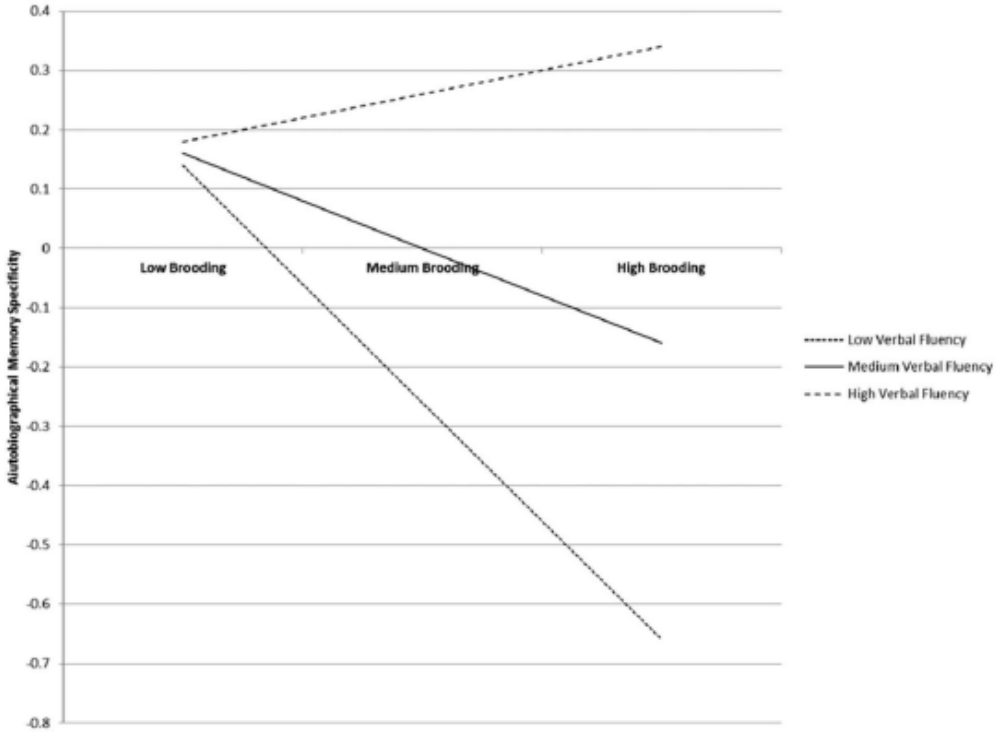
The present study tested Williams et al.'s (2007) CaR–FA–X model of mechanisms underlying reduced AMS. In both individuals with and without a history of MDD, we found greatest support for associations between reduced AMS and the capture and rumination, and impaired executive control mechanisms. In these groups, brooding and verbal fluency contributed to reduced AMS in interaction. However, consistent with evidence suggesting differences in the nature of the influences on AMS in different populations (e.g., Dalgleish et al., 2008; Williams et al., 2007), the pattern of this interaction differed for these groups. Among participants without a history of MDD, lower verbal fluency was associated with reduced AMS at high levels of brooding. However, in those with a history of MDD, lower verbal fluency was associated with reduced AMS at low levels of brooding. The former finding was in line with predictions from the CaR–FA–X model whereas the latter was not.

The pattern of the Brooding × Verbal Fluency interaction in participants without a history of MDD is consistent with the notion that those without a history of MDD but high on brooding may be especially likely to activate overgeneral self-relevant information during retrieval. This information is thought to be highly elaborated and interconnected due to being processed repeatedly in an analytical and evaluative way during brooding. However, these individuals may only become "captured" and provide an overgeneral response on the AMT if they lack sufficient executive control to reject these inappropriate responses.

In contrast, individuals with a history of MDD and high levels of brooding exhibited higher levels of AMS, irrespective of verbal fluency. This pattern of results was counter to our predictions based on the CaR–FA–X model. Indeed, both correlational and experimental research on individuals with current MDD has suggested that processing information in a repetitive, evaluative, and analytical way is associated with reduced AMS (e.g., Watkins & Teasdale, 2004;



a) Participants with a history of MDD



b) Participants without a history of MDD

Figure 3. Autobiographical memory specificity (scored on a standardized latent variable metric) as a function of brooding and verbal fluency. (a) Participants with a history of major depressive disorder (MDD). (b) Participants without a history of MDD.

Williams et al., 2007). Our unpredicted findings may be due to our having examined associations between reduced AMS and the CaR–FA–X mechanisms in individuals with a history of MDD rather than with current MDD. Most research on associations between analytical, evaluative ruminative processing and reduced AMS has been conducted in dysphoric or depressed individuals; very little research has examined this topic in individuals who only have a history of MDD. As summarized by Raes, Schoofs, Griffith, and Hermans (2012), a few unpublished studies have failed to find significant relationships between analytical, evaluative ruminative processing and reduced AMS in participants in remission from MDD. Those in remission from MDD are likely to be in a euthymic state, and this may interfere with observing an association between ruminative processing and reduced AMS. It is unfortunate that we did not assess depressive symptoms at the time of the AMT, and thus we were unable to examine directly whether the degree of current depressive symptoms impacted the association between brooding and reduced AMS. However, Raes et al. (2012) proposed and provided some initial support for the notion that relationships between trait measures of analytical, evaluative ruminative processing and reduced AMS may only be observed in individuals remitted from MDD when a state of ruminative processing is activated (e.g., by a challenging context). Future research should compare relationships between the CaR–FA–X mechanisms and AMS that is assessed under both neutral conditions and conditions that should activate depressogenic processing in individuals with a history of MDD.

An alternative explanation for our finding that individuals with a history of MDD and high levels of brooding exhibited high, rather than low, levels of AMS is that these individuals may have been exerting extra effort on the AMT as a way to compensate for prior patterns of negative thinking. Research has suggested that individuals in remission from depression may employ effortful control strategies as a way to counter underlying tendencies toward negative thinking, thereby masking these depressotypic thinking patterns (e.g., Wenzlaff & Bates, 1998). However, disrupting these effortful mental strategies (e.g., with the addition of a cognitive load) in those with a history of depression has been found to reveal cognitive biases that are characteristic of depression (e.g., Watkins & Moulds, 2007). It is possible that the nature of the Brooding × Verbal Fluency interaction in those with a history of MDD may reflect that participants with a history of MDD and a tendency toward brooding worked very hard to overcome thinking patterns that are characteristic of a depressed state. Although we did not find that reduced AMS was "revealed" under lower levels of verbal fluency in these participants, it is possible that a more disruptive and taxing condition, such as imposing a cognitive load, might be needed to sufficiently interfere with the effortful control strategies that serve to mask underlying cognitive biases. It would be of interest for further research to directly test this possibility to better understand the processes underlying reduced AMS in those with a history of MDD.

Recommendations for the CaR–FA–X Model

In sum, we found greatest support for relationships between AMS and the capture and rumination, and impaired executive control mechanisms of the CaR–FA–X model. Overall, our results are consistent with findings of an interaction between capture and rumination, and impaired executive control in contributing to reduced AMS in individuals in remission from MDD (Barnhofer et al., 2007) and with the conclusions from our recent review of the CaR–FA–X model literature (Sumner, 2012). These findings suggest that considering analytical, evaluative ruminative processing and impaired executive control and, more important, how they interact, may be critical

for understanding reduced AMS in individuals with and without a history of MDD. Furthermore, we found that brooding and verbal fluency differed in their relationships with AMS in those with and without a history of MDD. As noted by Williams et al. (2007), the presence of psychopathology (e.g., MDD) can impact the different CaR–FA–X mechanisms. Moreover, there is no one path for the development of reduced AMS, and the influences on AMS may vary for different populations. Consistent with the notion that executive control is associated with AMS irrespective of the presence of psychopathology (Williams et al., 2007), greater verbal fluency was associated with greater AMS in both groups but whether this relationship emerged under low or high brooding differed based on MDD history. Thus, an analytical, evaluative processing style may impact the accessibility of memories differently in those with versus without MDD.

Although we found minimal support for the functional avoidance mechanism, it is premature to conclude that functional avoidance does not contribute to reduced AMS. For one, our measures of the functional avoidance mechanism assessed a history of adversity and avoidant coping behavioral tendencies rather than whether avoiding the retrieval of specific memories served as a means of affect regulation (a key tenet of the functional avoidance mechanism). This limitation may have contributed to our failure to detect significant associations between our particular measure of functional avoidance and reduced AMS. Furthermore, a growing body of research has suggested that relationships between functional avoidance and reduced AMS may emerge in particular contexts. For example, as noted earlier, initial evidence suggests that functional avoidance may underlie reduced AMS in trauma-exposed individuals with PTSD symptoms (Dalgleish et al., 2008). Associations between avoidant coping and reduced AMS also may emerge when AMS is assessed under threatening conditions that signal a potential need for affect regulation. In an undergraduate sample, Debeer et al. (2011) found that avoidant coping was negatively correlated with AMS when participants were told that the AMT could elicit emotionally painful memories, but not when the AMT was administered under neutral conditions. Avoidant tendencies may therefore manifest as reduced AMS when individuals expect to be distressed by their memories. This is a topic of interest for future research.

Limitations

The present study has several strengths, including a large, diverse young adult sample (one of the largest studies of AMS to date), interview measures of Axis I psychopathology and adversity, tests of theory-driven hypotheses, and the use of SEM. Nevertheless, there are limitations. This study provides a cross-sectional assessment of relationships between the CaR–FA–X mechanisms and reduced AMS. Moreover, some measures of the mechanism variables were conducted after AMS assessment (e.g., the brooding and avoidant coping questionnaires could be completed before or after the AMT). Due to this, our study cannot address whether the CaR–FA–X mechanisms cause reduced AMS. Another limitation is that we were unable to test directly whether the different patterns of the Brooding × Verbal Fluency interactions were a function of MDD history because attempts to conduct a multiple group comparison did not converge due to high complexity and low covariance coverage. Even though the ranges of scores on the brooding measure were equal for those with and without a history of MDD, individuals with a history of MDD had higher mean scores and larger standard deviations compared to individuals without a history of MDD. We were thus unable to definitively ascertain whether our pattern of findings was due to a range effect on the brooding measure as opposed to a genuine group difference, and this is a limitation of the study.

Furthermore, it is possible that the comparability of our participants without a history of MDD to other nondepression control groups in the literature may be limited due to the behavioral high-risk design of the YEP in which we oversampled individuals scoring in the upper tertile on the EPQ–R–N scale at screening. There is some evidence that higher neuroticism is associated with reduced AMS (e.g., Kuyken & Dalgleish, 2011). In our study, there was no significant association between the EPQ–R–N score and the proportion of specific memories retrieved on the AMT, and there was no significant interaction between the EPQ–R–N score and the history of MDD in predicting reduced AMS. Nevertheless, this issue may limit the generalizability of our findings, and additional research on the mechanisms underlying reduced AMS in individuals not selected on neuroticism is needed. In addition, given that we examined participants with a history of MDD, it is not possible to determine what may be a scar, rather than a cause or correlate, of depression. Another limitation is that we did not measure depressive symptoms at the AMT. Even though SCID data were used to determine participants' MDD status at the AMT, we were thus unable to take into consideration how current depressed mood at the AMT may have impacted AMS and its associations with the CaR–FA–X mechanisms. This is an important topic for future research to examine.

Finally, some measures did not fully assess the CaR–FA–X mechanisms they represented. For example, the "capture" component of capture and rumination was not addressed. Furthermore, limiting the measure of impaired executive control to verbal fluency—a broad measure of executive control—prevented us from examining whether certain aspects of executive control (e.g., inhibition) were related to AMS. Additional research with more comprehensive measures of the CaR–FA–X mechanisms is needed.

Summary and Future Directions

The present study is the first to test relationships between AMS and the three CaR–FA–X mechanisms in individuals with and without a history of MDD. We found greatest support for associations between reduced AMS and the capture and rumination, and impaired executive control mechanisms, although these results warrant replication. Furthermore, several questions remain regarding the mechanisms underlying reduced AMS. One such question is the extent to which the CaR–FA–X mechanisms contribute to reduced AMS over time. Longitudinal investigations with multiple assessments of AMS and the CaR–FA–X mechanisms are needed to test whether these mechanisms lead to the development of reduced AMS. In addition, our findings highlight the importance of examining relationships between AMS and the CaR–FA–X mechanisms as a function of MDD history. Future research should continue to investigate factors that contribute to reduced AMS in different populations (e.g., individuals with current MDD, trauma victims with PTSD). Such work will further understanding of the processes underlying this important memory phenomenon.

Footnotes

1 OGM has been associated with trauma-related psychopathology (e.g., Williams et al., 2007), and we excluded the small number of individuals who met criteria for PTSD and ASD. We also excluded those with a history of bipolar disorder, major depression due to a general medical condition, substance-induced mood disorder, and psychotic symptoms to have a relatively homogenous sample of those with a history of unipolar depression.

2 In our study, there was no significant association between the neuroticism score based on the EPQ–R–N administered at screening and the proportion of specific memories retrieved on the AMT, $r = .001$, $p = .49$. In addition, there was no significant interaction between the EPQ–R–N score and history of MDD in predicting reduced AMS, $F(1, 435) = 0.04$, $p = .85$.

3 Reliability of categorical SCID diagnoses was assessed with Cohen's kappa and adjusted kappa due to uneven prevalence of counts in the compared categories (specifically, low base rates of cases with disorders relative to no disorders). This imbalance can attenuate Cohen's kappa. Some researchers have recommended calculating an adjusted kappa that adjusts cell frequencies to evenly distribute the prevalence of cases across categories (Sim & Wright, 2005).

4 See Vrshek-Schallhorn et al. (2014) for results demonstrating that early adversity predicted onset of mood disorders in this sample.

5 The observed relationships between avoidant coping and other constructs in the models are based on modeling relationships between the higher order avoidance latent variable and the other variables in the model. However, the overall pattern of results was the same when paths were included between not only a general avoidant coping factor and the other variables in the model but also between lower order factors representing denial, behavioral disengagement, and mental disengagement and the other variables in the model (cf. Lewis, Zinbarg, Mineka, Craske, & Griffith, 2010).

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