Reduced Autobiographical Memory Specificity Predicts Depression and Posttraumatic Stress Disorder After Recent Trauma

Birgit Kleim and Anke Ehlers
King's College London

In this prospective longitudinal study, the authors examined the relationship between reduced specificity in autobiographical memory retrieval and the development of depression, posttraumatic stress disorder (PTSD), and specific phobia after injury in an assault. Assault survivors ($N = 203$) completed the Autobiographical Memory Test (J. M. G. Williams & K. Broadbent, 1986) at 2 weeks after the trauma as well as structured clinical interviews at 2 weeks and 6 months. Participants with acute stress disorder or major depression at 2 weeks, but not those with phobia, retrieved fewer specific autobiographical memories than those without the respective disorder. Reduced memory specificity at 2 weeks also predicted subsequent PTSD and major depression at 6 months over and above what could be predicted from initial diagnoses and symptom severity. Moderator analyses showed that low memory specificity predicted later depression in participants with prior episodes of major depression but not in those without prior depression. Mediation analyses suggested that rumination partly mediated and perceived permanent change fully mediated the effects of low memory specificity on posttrauma psychopathology at follow-up.

Keywords: Autobiographical Memory Test, overgeneral memory, posttraumatic stress disorder, depression, specific phobia

Supplemental materials: http://dx.doi.org/10.1037/0022-006x.76.2.231.supp

Overgeneral Memory and Depression

People with major depression often show difficulty in retrieving specific autobiographical memories (Williams & Broadbent, 1986; for a review see Williams et al., 2007). When asked to remember a specific event from their lives in response to a cue word (e.g., "happy"), individuals with depression show an overgeneral memory bias (OGM), that is, they tend to reply with descriptions that summarize several different events (e.g., "I am always happy when I visit friends") rather than a specific instance ("I was happy when my school friend rang me last week"). The origins and consequences of OGM in depression have attracted much attention (see Williams et al., 2007, for a review). Depression is often triggered by stressful life events (e.g., Kendler, Karkowski, & Prescott, 1999). This raises the issue of whether stressful or traumatic life events play a role in the development of OGM retrieval. Williams (1996) suggested that an overgeneral retrieval style may develop as a means of regulating affect (*affect-regulation hypothesis*) after

stressful events. He proposed that survivors of trauma or severe stress may learn to avoid the painful emotions that would be provoked if they recalled their negative experiences by halting autobiographical memory retrieval prematurely, that is, before a specific memory is retrieved. Over time, the truncated search may become generalized, leading to an overgeneral level of autobiographical remembering. Such an overgeneral retrieval style is thought to be an index of vulnerability to subsequent depression. OGM has indeed been shown to predict depression and anxiety after a failed in vitro fertilization (van Minnen, Wessel, Verhaak, & Smeenk, 2005) and to interact with the occurrence of stressful life events in predicting subsequent depression in college students (Gibbs & Rude, 2004).

Overgeneral Memory and Trauma

The affect-regulation hypothesis suggests that exposure to stressful events is a precursor of OGM. Empirical research only offers partial support for a relationship between OGM and trauma (Moore & Zoellner, 2007) in that some, but not all, studies found an association. Among depressed samples, a large proportion of individuals with OGM also reported a history of trauma and trauma-related intrusive and avoidance symptoms (e.g., Dalgleish et al., 2003; Hermans et al., 2004; Kuyken & Brewin, 1995). Henderson, Hargreaves, Gregory, and Williams (2002) found in a nonclinical sample that women who reported a history of childhood abuse had difficulties retrieving specific memories in response to cue words. However, some studies failed to find an association between childhood trauma and OGM (e.g., Johnson, Greenhoot, Glisky, & McCloskey, 2005; Wessel, Meeren, Peeters, Arntz, & Merckelbach, 2001).

OGM and Posttraumatic Stress Disorder (PTSD)

The inconsistent findings on the relationship between trauma and OGM raise the possibility that OGM may not be a necessary consequence of trauma (Moore & Zoellner, 2007). However, OGM may influence how well people adapt to such extremely stressful experiences. Problems in autobiographical memory retrieval may prevent trauma survivors from coming to terms with the trauma and from reclaiming their former lives. Researchers and clinicians have observed that people with PTSD appear "frozen" at the time of the traumatic event (e.g., Herman, 1992) and that they have difficulty remembering what they used to be like before the traumatic event (e.g., Ehlers, Maercker, & Boos, 2000). OGM may contribute to such changes in self-perception.

Several studies have found that trauma survivors with PTSD or acute stress disorder (ASD) show less specific autobiographical memory retrieval than those without PTSD or ASD (Harvey, Bryant, & Dang, 1998; Kangas, Henry, & Bryant, 2005; McNally, Lasko, Macklin, & Pitman, 1995; Schönfeld & Ehlers, 2006; Schönfeld, Ehlers, Böllinghaus, & Rief, 2007).

OGM and Other Anxiety Disorders

Interestingly, a series of studies have found that OGM does not appear to be associated with most other anxiety disorders (Burke & Mathews, 1992; Wenzel & Jordan, 2005; Wenzel, Werner, Cochran, & Holt, 2004). Thus, the literature suggests that OGM may be a specific feature of depression and ASD/PTSD. However, there is a lack of studies directly comparing the relationships between OGM and different psychological outcomes after stress.

OGM and the Maintenance of Depression and PTSD

It has been suggested that OGM plays a role in the maintenance of depression (e.g., Gibbs & Rude, 2004; Peeters, Wessel, Merckelbach, & Boon-Vermeeren, 2002; Williams et al., 2006). OGM has been shown to predict poorer long-term clinical outcome in people with depression (e.g., Brittlebank, Scott, Williams, & Ferrier, 1993; Raes, Hermans, Williams, Beyers, et al., 2006; Williams, 1996). It has also been shown to be associated with deficits in social problem solving (Goddard, Dritschel, & Burton, 1996) and low ability to imagine future events (Williams et al., 1996), both of which may contribute to the maintenance of negative affect and hopelessness.

It remains as yet unclear whether OGM is also involved in the maintenance of PTSD. Theories of PTSD suggest that recovery from trauma requires the integration of the traumatic experience with the person's schemas about the self and the world (e.g., Resick & Schnicke, 1993) and with other autobiographical memories that determine the person's sense of self (e.g., Conway & Pleydell-Pearce, 2000; Ehlers & Clark, 2000). An impaired ability to retrieve specific memories from one's past (in particular positive memories) may impede such integration and thus delay recovery (see also Ehlers & Clark, 2000; Harvey et al., 1998; van Minnen et al., 2005; Williams et al., 2006). Prospective longitudinal studies are sparse and have generated mixed evidence as to whether OGM predicts greater long-term posttraumatic stress symptom severity. Harvey et al. (1998) assessed OGM shortly following accidents and found that low specificity in retrieving

memories from the period following the trauma to positive cues predicted subsequent PTSD. However, Kangas et al. (2005) did not find that OGM predicted subsequent PTSD in recently diagnosed cancer patients, although OGM was related to concurrent ASD. In the latter study, memory retrieval was not restricted to the time following the trauma. In the present study, we investigated prospectively whether OGM predicts PTSD and depression in a sample of assault survivors.

Possible Mechanisms of Maintenance

What are the possible mechanisms by which OGM may put people at risk of chronic depression and PTSD after trauma? One possible way in which OGM may impede recovery is through a reciprocal relationship with rumination (Williams, 1996). According to the *capture and rumination hypothesis* (Williams et al., 2006), the cues used in the standard measure of OGM, the Autobiographical Memory Test (AMT; Williams & Broadbent, 1986), may activate rumination and abstract negative self-schemas so that respondents get "captured" in abstract cognitions and have problems progressing to the retrieval of specific memories. The failure to retrieve specific memories may in turn make it more difficult to correct the negative self-schemas and, thus, maintain negative thinking and related negative affect. People with depression or PTSD may be vulnerable to this maintaining process, as these disorders are characterized by negative self-related schemas and rumination (e.g., Foa, Ehlers, Clark, Tolin, & Orsillo, 1999; Ingram, Miranda, & Segal, 1998; Murray, Ehlers, & Mayou, 2002; Nolen-Hoeksema & Morrow, 1991). In contrast, people with other anxiety disorders—such as specific phobias—may be less susceptible, as their anxiety is mainly focused on external situations, and their self-schema may be less affected. In PTSD, failure to retrieve specific memories may be particularly relevant for appraisals of permanent change, that is, the appraisal that one and one's life have irreversibly changed for the worse by the trauma. Schönfeld and Ehlers (2006) found that OGM was associated with rumination and with perceived permanent change in trauma survivors. Perceived permanent change may interfere with people reclaiming their former lives after trauma and delay recovery. It has been shown to predict chronic PTSD (Dunmore, Clark, & Ehlers, 2001; Ehlers et al., 2000).

The above considerations raise the possibility that rumination and negative self-related schemas mediate the relationship between OGM and psychopathology. Preliminary data in support of this hypothesis were presented by Raes, Hermans, Williams, Beyers, et al. (2006), who showed that rumination partly mediated the relationship between OGM and subsequent depression. Data on PTSD and self-related schemas are lacking. In the present study, therefore, we considered rumination and perceived permanent change as potential mediators between OGM and depression and PTSD after trauma.

Aims of the Study

The purpose of the present study was to examine autobiographical memory retrieval in a sample exposed to a recent severe stressor, injury in a violent assault. We studied whether memory specificity is related to major depression, ASD, and specific assault-related phobia at 2 weeks postassault. To our knowledge,

this is the first study to investigate the role of specificity in predicting these three disorders in the same sample of trauma survivors. On the basis of previous findings (Gibbs & Rude, 2004; Harvey et al., 1998; Wenzel, Jackson, & Holt, 2002), we hypothesized that assault survivors with major depression and those with ASD, but not those with specific phobia, would retrieve fewer specific memories than those without these disorders. Furthermore, we expected that memory specificity at 2 weeks would predict major depression and PTSD at 6 months following assault, and we tested whether it predicted over and above initial symptoms. In this study, we also tested whether rumination and perceived permanent change would mediate the influence of OGM on depression and PTSD. Finally, we considered history of childhood abuse, history of major depression, sex, and ethnicity as potential moderators of the relationship between memory specificity and posttrauma psychopathology (Williams et al., 2007).

Method

Participants

Participants were recruited from assault survivors who were treated for their injuries at the emergency department of a large urban teaching hospital during the period between July 2003 and December 2004. To be eligible for the study, participants had to understand and speak English fluently enough to be able to answer interview questions and fill in questionnaires. Participants with current psychosis and substance dependence, as well as those who could not remember the event (e.g., because of a head injury), were excluded.

A total of 1,063 assault survivors who attended the emergency department during the recruitment period were contacted, of whom 389 did not fulfill inclusion criteria, and 197 declined to take part. Another 255 were initially interested but failed to schedule or attend the research session within the designated time period. A total of 222 participants consented to participate in a research session at 2 weeks after the assault, and 203 (91%) of these completed the AMT. Reasons for noncompletion were too little time to complete the test and inability to concentrate for an extended period of time. Table 1 presents the demographic and clinical characteristics of the study sample. Participants had mainly been physically assaulted ($n = 200$, 99%; $n = 3$ sexual assaults). At 6-month postassault, 190 of the participants who had completed the AMT (83%) completed a further diagnostic interview.

To test whether the sample was representative of assault survivors presenting to this emergency department, we compared their demographic characteristics with those of a random sample drawn from all 2,785 assault survivors treated in the department during a period of 1 year. The study group and the random sample did not differ in age, sex, or severity of the injuries (all $ps > .296$). However, comparison with hospital data showed that the study sample may[1] have comprised significantly more Caucasian participants than the random sample, $\chi^2(1, N = 416) = 17.12, p < .001$, namely 57.2% compared with a recorded 36.9% in the hospital sample. This difference may be due to the fact that some non-Caucasians had to be excluded because of language difficulties. We therefore tested whether ethnicity moderated the effects of memory specificity on psychopathology.

AMT

The AMT (Williams & Broadbent, 1986) was administered individually by Birgit Kleim following the procedures outlined in Williams's (2003) manual. Participants saw 12 cue words (6 positive words: cheer, pleased, relieved, lively, glorious, peaceful; 6 negative words: worse, guilty, hopeless, awful, grave, ugly), which occurred in random order on a computer screen. Positive and negative words were matched for word frequency derived from the London–Lund corpus of English conversations (see Brown, 1984). The participants' task was to retrieve, and briefly describe, a specific personal memory in response to each cue word. The experimenter explained that a specific memory is a past experience that happened on a particular day. Participants were told that the event could be important or trivial. The time period from which events could be recalled was not specified. The experimenter gave examples of appropriate specific memories and inappropriate general memories, and gave participants the opportunity to practice the task with practice words. The AMT was not started until the participants had retrieved at least one specific memory to one of the practice words. Participants were given a maximum of 30 s to retrieve a memory for each word. If they did not provide a response in that time, this was scored as an omission. Responses were tape-recorded and later transcribed and scored for the percentage of first memories that were specific memories and for the assault-relatedness of specific memories. A memory was rated as assault-related when it involved the assault or its consequences (e.g., "when I looked into the mirror and saw the scar from the assault this morning"). A trained psychology graduate student who was blind to participants' diagnostic status rated all memories. A second independent rater who was blind to participants' diagnostic status scored a random sample of 50 oral AMT responses; good interrater agreement was obtained for the categorization of specific versus nonspecific responses ($\kappa = .87$) and for the categorization of assault-relatedness ($\kappa = .78$).

Assessment of Diagnoses and Symptom Severities

At 2 weeks, diagnoses of current and past (i.e., prior to the assault) major depression, ASD, and assault-related specific phobia were established with standard structured clinical interviews—the Structured Clinical Interview for *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996) and the Acute Stress Disorder Scale (ASDS, Interview Version; Bryant & Harvey, 2000), respectively. A trained psychologist (Birgit Kleim) conducted all interviews under the supervision of Anke Ehlers. Note that *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) proposes a hierarchical relationship in the diagnosis of some of the psychiatric disorders, stipulating that phobia may not be diagnosed if it is better accounted for by PTSD. To allow the investigation of phobic responses independent of the presence of PTSD symptoms, we omitted this rule in the present study, and participants could thus

---

[1] This figure presents an underestimate of the number of Caucasian hospital attendees. The figures are based on emergency department codes that require the staff to code ethnicity in 80 categories on the basis of country of origin. In practice, the category of "other" was often for non-British participants, regardless of their ethnic background.

Table 1
Sample and Assault Characteristics (N = 203)

Variable	*n*	%
Sex		
Male	137	67.5
Female	66	32.5
Ethnicity		
Caucasian	117	57.6
Black	67	33.3
Other or mixed race	19	9.1
Socioeconomic status[a]		
Very low income (less than £10,000)	86	43.4
Low income (£10,000–£20,000)	47	23.2
Moderate income (£20,000–£40,000)	34	16.7
High income (over £40,000)	23	11.3
Refused information	13	6.4
Marital status		
Single	135	66.5
Married	36	17.7
Divorced/separated	24	11.8
Widowed	2	1.0
Refused information	6	2.9
Employment status		
Employed/studying	137	67.5
Not employed	66	32.5
No. of assailants		
Single assailant	106	52.2
Multiple assailants	96	47.3
Missing information	1	0.5
Weapon usage		
Weapon not used	82	40.4
Weapon used	119	58.6
Missing information	2	1.0
Alcohol or drug use at the time of assault		
Intoxicated	89	43.8
Not intoxicated	113	55.7
Missing information	1	0.5
Diagnoses and comorbidity at 2 weeks		
Major depression (MD)/MD only	38/18	18.7/8.9
Acute stress disorder (ASD)/ASD only	34/10	16.7/4.9
Specific phobia (SP)/SP only	48/22	23.6/10.8
MD and ASD	4	2.0
SP and ASD	10	4.9
MD and SP	6	3.0
MD, ASD, and SP	10	4.9
Neither MD, ASD, nor SP	123	60.6
Diagnoses and comorbidity at 6 months (*N* = 190)		
MD/ MD only	31/8	16.3/4.2
Posttraumatic stress disorder (PTSD)/PTSD only	46/13	24.2/6.8
SP/SP only	38/6	20.0/3.1
MD and PTSD	4	2.1
SP and PTSD	11	5.8
MD and SP	3	1.6
MD, PTSD, and SP	16	8.4
Neither MD, PTSD, nor SP	127	66.8
Age in years, *M* (*SD*)	34.39	(11.17)
Education in years, *M* (*SD*)	14.00	(4.80)
No. of adult traumas, *M* (*SD*)	2.29	(1.41)
Assault duration in minutes, *M* (*SD*)	9.44	(18.52)

[a] Combined household income.

meet diagnoses of both ASD (or PTSD) and phobia if they had phobic symptoms severe enough to cause clinically significant distress.

The ASDS rates the presence of 19 ASD symptoms, each on a scale ranging from 0 (*not at all*) to 4 (*very much*). It determines a diagnosis of ASD as well as a symptom severity score (the sum of the scores for each symptom). In parallel, a depression symptom severity score was calculated as the sum of *DSM–IV* depression symptoms in the SCID (coded as 1 = *absent*, 2 = *subthreshold*, and 3 = *present*). Severity of assault-related phobia was assessed

with the Assault Phobia Questionnaire. This 10-item scale follows *DSM–IV* criteria for specific phobias. It is similar to a scale for accident-related phobia developed by Ehring, Ehlers, and Glucksman (2006) that distinguishes well between people with and without travel phobias. Participants are asked to identify the situation, person, or object that they feared most since the assault. They indicate on a scale ranging from 0 (*not at all*) to 4 (*very strongly*) how afraid they were of this situation, person, or object (e.g., "I am very afraid of . . .") and how much they avoided it (e.g., "I always avoid . . ."). The scale showed excellent internal consistency (α = .94).

At 6 months, the SCID modules for PTSD, major depression, and assault-related phobia were administered over the telephone by the same interviewer. The severity of PTSD symptoms was assessed with the PTSD Symptom Scale—Interview Version (PSS–I; Foa, Riggs, Dancu, & Rothbaum, 1993). The interviewer rated each of the PTSD symptoms on a scale ranging from 0 (*not at all*) to 3 (*5 or more times per week/very much*). The total PSS–I score is the sum of the ratings for the 17 items.

Cases were selected randomly for interrater reliability ratings, which were high (κ = .97 for ASD, κ = 1.00 for depression, κ = .85 for phobia, κ = .82 for PTSD; on the basis of 56 interviews, two raters who were each uninformed as to the other rater's diagnoses).

The interviewer asked participants when their symptoms started to ensure that they were responses to the assault. If participants reported that they had already been depressed or had other symptoms prior to the assault, the interviewer enquired whether the symptoms worsened as a consequence of the assault.

Assessment of Potential Mediators

Response to Intrusions Questionnaire. The Response to Intrusions Questionnaire (Clohessy & Ehlers, 1999; Murray et al., 2002) assesses dysfunctional cognitive strategies in response to intrusive memories. It was developed over a series of studies and shows good internal consistency and validity in predicting chronic PTSD (Clohessy & Ehlers, 1999; Murray et al., 2002). The Rumination subscale (α = .84 in the present study) comprises seven items asking participants to rate what they do when memories of the assault pop into their mind (e.g., "I dwell on how the event could have been prevented," or "I dwell on how I used to be before the event"), each on a scale ranging from 0 (*not at all*) to 3 (*always*).

Perceived permanent change. This was assessed with the four relevant items from the Posttraumatic Cognitions Inventory (Foa et al., 1999)—for example, "I have permanently changed for the worse" (α = .77 in the present sample). The Posttraumatic Cognitions Inventory measures trauma-related thoughts and beliefs that have been shown to discriminate well between trauma survivors with and without PTSD. It has been shown to have good internal consistency and retest reliability.

Verbal Intelligence

Participants completed the National Adult Reading Test (NART; Nelson, 1991), a measure of verbal intelligence that is a widely accepted method of estimating premorbid intelligence levels. The NART requires participants to read out loud a list of 50

irregularly spelled words in order of increasing difficulty. Responses are individually scored as correct or incorrect, according to their pronunciation. The number of words read correctly comprises the final score. The NART has excellent reliability and construct validity (Crawford, Parker, Allan, Jack, & Morrison, 1991). It correlates highly with other measures of intelligence and allows the prediction of full-scale IQ scores (Nelson, 1991). Correlations with the Wechsler Adult Intelligence Scale (Wechsler, 1997) in healthy participants were around $r = .75$ (Bright, Jaldow, & Kopelman, 2002). Performance is characteristically preserved relative to other measures of cognitive function (e.g., Crawford, Steward, Garthwaite, Parker, & Besson, 1988). Recent studies have shown that current NART performance is superior in estimating a premorbid intelligence to scores based on demographic variables, such as education (e.g., Bright et al., 2002).

History of Childhood Abuse and Adult Trauma

Trauma history. Participants were asked whether, during their childhood, they had ever been physically abused or whether they had had any unwanted sexual experience as part of an interview about previous traumas (adapted from Kubany et al., 2000). They also stated the nature of the abuse, their age, and distress at the time. Participants further reported whether they had experienced any from a list of other traumatic events, such as an earlier assault, a sexual assault, a motor vehicle accident, warfare or combat, a robbery, threat of death or serious body harm, or any other life threatening or highly disturbing event, such as a natural disaster. The number of traumatic events experienced was used as an index of adult trauma history.

Objective assault severity. Objective severity of the recent assault was scored as a composite score from the following information: number of assailants (0 = *one assailant*, 1 = *two or more assailants*), assault duration (0 = *5 min or less*, 1 = *more than 5 min*), injury severity (0 = *mild injuries*, 1 = *moderate injuries*, 2 = *severe injuries*), and weapon usage (0 = *no weapon used*, 1 = *weapon used*; see Dunmore et al., 2001).

Procedure

The study was approved by the local ethics committees and undertaken between July 2003 and December 2004. A few days after their admission at the emergency department, assault survivors received information about the study by mail and were invited to participate in a research session at 2 weeks after the assault. The invitation letter was followed by a telephone call in which further information about the study was given. Upon arrival at the session, participants had the opportunity to ask further questions and gave written informed consent. In the session, participants provided some details about their assaults, gave a short trauma narrative, did a picture identification task (results are reported elsewhere), and filled in questionnaires. They then completed the AMT. The diagnostic interviews followed. At 6 months, participants completed the relevant SCID modules over the telephone and filled in questionnaires. Participants were reimbursed £50 ($97) for their time and travel expenses.

Data Analysis

The Statistical Package for the Social Sciences (SPSS 15.0) was used for all analyses. Following common practice in investigations

of OGM (see Williams et al., 2006, 2007), the main dependent variable was the percentage of the first memories that participants retrieved in response to the AMT cue words that were specific (we refer to this variable as *memory specificity*). We tested differences between participants with and without the disorders under investigation at 2 weeks (e.g., ASD vs. no ASD; major depression vs. no depression) with analyses of variance using the general linear modeling procedure, with the between-factors diagnostic group and the within-subject factor AMT cue valence (positive vs. negative). Logistic regression analyses tested whether memory specificity contributed to the prediction of PTSD, depression, and phobia at 6 months over and above what could be predicted from initial symptoms. Multiple regression analyses were conducted for moderator and mediator analyses (Baron & Kenny, 1986). PSS–I scores were square root transformed to normalize distributions. Mediation analyses were conducted in accord with McKinnon and Dwyer (1993; see also McKinnon, Fairchild, & Fritz, 2007). The Sobel test tested the significance of mediation effects.

The significance level was set at $p = .05$, two-tailed. On the basis of the proportion of participants with the respective disorders, the study had 86%, 82%, and 91% power in detecting group differences in the order of $d = 0.55$ (estimated from Schönfeld & Ehlers, 2006; Schönfeld et al., 2007) at 2 weeks for depression, ASD, and specific phobia, respectively, for $\alpha = .05$, two-tailed. At 6 months, the sample size of $N = 190$ had 78% power to detect an odds ratio of 1.7 in the prediction of PTSD or depression diagnosis with OGM, after adjusting for initial symptom severity (estimated from Bryant, Sutherland, & Guthrie, 2007; Schönfeld & Ehlers, 2006).

Results

Diagnoses

As shown in Table 1, 18.7% had major depression at 2 weeks, 16.7% participants had ASD, and 23.6% had an assault-related phobia. Of the 38 participants with major depression, 14 (36.8%) had become depressed after the assault, and 24 (63.2%) had already felt somewhat depressed before the assault. All of the latter participants reported that the depression had significantly worsened as a result of the assault; furthermore, none of these participants had the same degree of depression present before the assault. At 6 months, 16.3% of the participants had major depression, 24.2% had PTSD, and 20.0% had an assault-related specific phobia. There was considerable comorbidity between the three disorders under investigation. At 2 weeks, 41% of the participants who met diagnostic criteria for ASD also had major depression, and 37% of the participants who met diagnostic criteria for major depression also had ASD. At 6 months, 43% of participants with PTSD had comorbid major depression, and 64% of those with major depression also had PTSD.

Group Differences in Memory Specificity at 2 Weeks

On average, participants retrieved $M = 7.63$ ($SD = 2.51$) specific memories across the 12 trials (64%); for positive cues, $M = 3.81$ ($SD = 1.47$); for negative cues, $M = 3.82$ ($SD = 1.46$). Omissions were rare (see Table 2). Table 2 presents the AMT performance at 2 weeks for the ASD, major depression, and phobia groups. For comparison, results for assault survivors who did not have any of the diagnoses under consideration are also reported (see Table 2). As a conservative test of the association between low autobiographical memory specificity and ASD, major depression, and assault-related phobia, we compared participants meeting diagnostic criteria for each of the disorders with those who did not meet the criteria for that disorder[2] (which could include participants meeting criteria for another disorder). These results are illustrated in Figure 1 (a table with means and standard deviations is available online as supplemental material). Significant main effects of diagnostic group indicated that participants with major depression, $F(1, 201) = 6.08$, $p = .014$, $\eta_p^2 = .029$, and participants with ASD, $F(1, 201) = 3.98$, $p = .047$, $\eta_p^2 = .019$, retrieved fewer specific autobiographical memories than participants without these disorders. In contrast, participants diagnosed with assault-related phobias did not show less specific memory retrieval than those without phobia, $F(1, 201) = 0.17$, $p = .681$, $\eta_p^2 = .001$. There were no main effects of cue valence (all $ps > .52$; all $\eta_p^2 < .003$) or interactions between valence and diagnostic group (all $ps > .42$; all $\eta_p^2 < .004$).

There were no significant group differences between any of the diagnostic groups and the respective control groups without that disorder in the assault-relatedness of specific memories (all $ps > .19$; all $\eta_p^2 < .009$). There was a significant main effect of valence: Across diagnostic groups, participants produced more assault-related memories to negative words than to positive words (all $ps < .001$; all $\eta_p^2 > .133$). There was also a significant interaction between valence and ASD, $F(1, 195) = 4.09$, $p = .045$, $\eta_p^2 = .021$, in that the ASD group retrieved more assault-related specific memories to negative cues than participants without ASD ($M_{ASD} = 61.27$, $SD = 38.02$ vs. $M_{NoASD} = 42.13$, $SD = 42.78$, respectively), $F(1, 202) = 5.86$, $p = .016$. Interactions between valence and depression and phobia diagnostic groups were nonsignificant (both $ps > .570$; both $\eta_p^2 < .002$). For omissions, there were no significant group differences (all $ps > .32$; all $\eta_p^2 < .006$) or valence effects (all $ps > .42$; all $\eta_p^2 < .004$). There was a marginally significant interaction between depression and valence, $F(1, 201) = 3.42$, $p = .066$, $\eta_p^2 = .017$. Depressed participants tended to produce more omissions in response to positive cues than nondepressed participants.

Prediction of PTSD, Major Depression, and Specific Phobia at 6 Months

Logistic regression analyses tested whether memory specificity at 2 weeks contributed to the prediction of PTSD, depression, and assault-related specific phobia at 6 months over and above initial symptoms. For PTSD at 6 months, ASD symptom severity at 2 weeks (ASDS) was forced into the regression in the first step and explained 43% of the variance, $\chi^2(1, N = 190) = 64.33$, $p < .001$, Nagelkerke $R^2 = .43$. Low memory specificity significantly improved the prediction, $\chi^2(1, N = 190) = 7.04$, $p = .008$, total Nagelkerke $R^2 = .47$, $\Delta R^2 = .04$. We repeated the analysis to test whether memory specificity contributed to the prediction of PTSD without comorbid depression or phobia. Low memory specificity

---

[2] The results were the same when the disorder groups were compared with participants without depression, ASD, or phobia.

Table 2

Autobiographical Memory Test Performance for Assault Survivors With Acute Stress Disorder (ASD), Major Depression (MD), and Specific Phobia (SP), and for Assault Survivors Without Any of These Disorders at 2 Weeks

Variable	ASD[a] ($n = 34$)		MD[a] ($n = 38$)		SP[a] ($n = 48$)		None of the disorders ($n = 123$)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Total % specific memories	57.11	20.53	56.14	23.47	62.50	21.33	66.01	20.02
Positive cues	55.39	25.53	56.14	31.10	61.81	25.72	66.67	22.18
Negative cues	58.82	24.01	56.14	21.72	63.19	26.62	65.45	24.16
Total % omissions	3.68	6.86	2.78	6.98	4.17	3.90	4.00	7.87
Positive cues	3.92	10.10	1.71	5.12	4.51	10.73	4.88	11.03
Negative cues	3.43	7.98	3.85	10.44	3.82	9.25	3.12	7.80
Total % assault-related specific memories[b]	43.45	30.47	31.74	33.06	37.87	32.59	32.44	31.64
Positive cues	20.83	24.26	13.87	28.08	24.46	34.27	22.91	38.49
Negative cues	58.85	37.89	40.78	42.42	48.70	38.58	43.92	44.02

[a] Disorder groups overlap due to comorbidity. [b] Index of proportion of specific memories that were assault related; data on test performance with regards to other categories (categoric, extended, nonmemories), which were not part of this analysis, can be obtained from the authors.

again improved the prediction over and above initial symptom severity at trend level, $\chi^2(1, N = 139) = 3.18$, $p = .075$, total Nagelkerke $R^2 = .369$, $\Delta R^2 = .04$. For major depression at 6 months, depression severity at 2 weeks (number of symptoms on SCID) was forced into the regression in the first step and explained 13% of the variance, $\chi^2(1, N = 187) = 14.59$, $p < .001$, Nagelkerke $R^2 = .13$. Low memory specificity significantly improved the prediction, $\chi^2(1, N = 187) = 7.45$, $p = .006$, total Nagelkerke $R^2 = .19$, $\Delta R^2 = .06$. For specific phobia at 6 months, phobia severity at 2 weeks (Assault Phobia Questionnaire) was forced into the regression in the first step and explained 27% of the variance, $\chi^2(1, N = 187) = 32.59$, $p < .001$, Nagelkerke $R^2 = .27$.
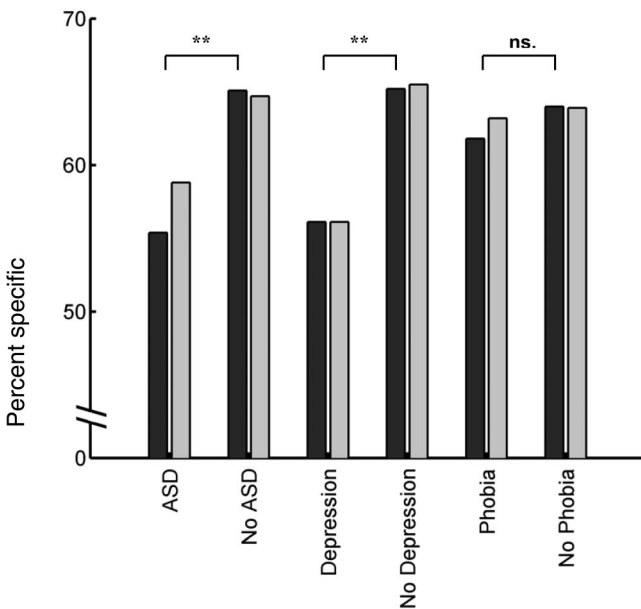

Figure 1. Mean percentages of specific autobiographical memories in assault survivors with and without acute stress disorder (ASD), depression, and assault-related phobia, respectively. Black bars = positive cues; gray bars = negative cues; ns. = nonsignificant. ** $p < .01$.

Low memory specificity did not add significantly to the prediction, $\chi^2(1, N = 187) = 1.45$, $p = .230$, total Nagelkerke $R^2 = .28$, $\Delta R^2 = .01$.

We also tested whether memory specificity predicts PTSD and depression diagnoses at 6 months over and above initial depression and ASD diagnoses. This was the case; memory specificity predicted both PTSD and depression over and above initial ASD and depression diagnoses, $\chi^2(1, N = 190) = 7.29$, $p = .008$, total Nagelkerke $R^2 = .292$, $\Delta R^2 = .047$ (PTSD), and $\chi^2(1, N = 190) = 6.23$, $p = .014$, total Nagelkerke $R^2 = .229$, $\Delta R^2 = .049$ (depression).

Hierarchical linear regression analyses using the continuous PSS–I and depression severity scores at 6 months as outcome measures obtained the same results. In the first step, symptom severity at 2 weeks was forced into the regression function, and OGM in the second step. For both PTSD and depression, OGM predicted over and above initial symptom severity: PTSD prediction, Step 1: $F(1, 188) = 152.90$, $p < .001$, $R^2 = .45$; Step 2: $\Delta F(1, 187) = 6.20$, $p = .014$, $\Delta R^2 = .02$, OGM $\beta = -.14$; depression prediction, Step 1: $F(1, 186) = 27.00$, $p < .001$, $R^2 = .13$; Step 2: $\Delta F(1, 185) = 4.18$, $p = .042$, $\Delta R^2 = .02$, OGM $\beta = -.14$.

OGM in PTSD and Depression: Correlates, Moderation Analyses, and Mediation Analyses

Table 3 shows the correlations between OGM, symptoms of PTSD and depression, trauma variables, intelligence, and the variables considered for moderation and mediation analyses. OGM correlated with the severity of the present assault and low intelligence but not with childhood abuse and past traumas. To test whether memory specificity predicts over and above intelligence and assault severity, we entered NART scores and assault severity in the first step of a hierarchical logistic regression analysis, and we entered memory specificity in the second step. OGM at 2 weeks predicted both PTSD and major depression at 6 months over and above what could be predicted from intelligence and assault severity: for PTSD, $\chi^2(1, N = 181) = 3.68$, $p = .055$, ΔNagelkerke $R^2 = .029$; for depression, $\chi^2(1, N = 181) = 5.76$, $p = .016$, ΔNagelkerke $R^2 = .052$.

Table 3
Pearson Correlations Between Memory Specificity, Acute/Posttraumatic Stress, and Depression Severity at 2 Weeks and at 6 Months With Trauma and Cognitive Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Memory specificity	—													
2. ASDS (2 weeks)	-.16*	—												
3. PSS-I (6 months)	-.24***	.67***	—											
4. Depression severity (2 weeks)	-.15*	.46***	.33***	—										
5. Depression severity (6 months)	-.20**	.34***	.49***	.33***	—									
6. History of child abuse	-.12	.13	.20**	.13	.06	—								
7. No. of prior adult traumas	-.04	.25***	.26***	.23**	.08	.32***	—							
8. Assault severity	-.15*	.11	.13	.13	.07	.07	.03	—						
9. Intelligence	-.30***	-.21**	-.32***	-.01	-.18*	-.10	.05	-.09	—					
10. Rumination	-.16*	.55***	.54***	.27***	.26***	.11	.15*	.20**	-.27***	—				
11. Permanent change	-.25***	.59***	.47***	.36***	.34***	.20**	.19*	.00	-.29***	.41***	—			
12. Sex	.00	.26**	.12	.07	-.02	.15*	.08	-.12	-.05	-.08	.10	—		
13. Ethnicity	.16*	.18**	.22**	.02	.11	.06	-.13	.06	-.53***	.30***	.08	-.06	—	
14. History of major depression	.07	.27***	.19*	.40***	.20**	.10	.24***	.09	.13	.23***	.25***	.06	-.06	—

Note. Memory specificity is indexed as the percentage of first memories that were specific. Negative correlations indicate positive associations with overgenerality; ethnicity is indexed as White (1) versus Other (2). ASDS = Acute Stress Disorder Scale; PSS-I = Posttraumatic Stress Disorder Symptom Scale—Interview Version.
* $p < .05$. ** $p < .01$. *** $p < .001$.

Moderation effects. To test for moderation, we entered memory specificity in the first step of a hierarchical multiple regression analysis, followed by the moderator variable under investigation, followed by the interaction of memory specificity and the moderator (Baron & Kenny, 1986). Sex, childhood abuse, and ethnicity did not moderate the effect of specificity on PTSD or depression; all interaction terms remained nonsignificant in the hierarchical regression equations (all $ps > .11$).

History of major depression prior to the assault moderated the effect of memory specificity on depression severity, as indicated by a significant interaction ($\beta = -.19$, $p = .042$), which contributed significantly to the explained variance in depression symptom severity at 6 months, $\Delta R^2 = .02$, $F(3, 186) = 6.86$, $p < .001$. Further analyses showed that for participants with a history of major depression ($n = 79$), memory specificity significantly predicted depression severity at 6 months ($\beta = -.32$, $p = .004$). Among participants who had never had a major depressive episode before the assault ($n = 106$), however, specificity was not a significant predictor of depression symptom severity at follow-up ($\beta = -.12$, $p = .232$). The interaction between history of major depression and memory specificity was not significant for PTSD symptom severity ($\beta = -.11$, $p = .231$).

Mediation effects. We used mediation analyses to test whether rumination and permanent change mediated the effects of memory specificity on PTSD and depression, using the three-step regression analyses approach suggested by Baron and Kenny (1986). Step 1 requires that the hypothesized mediator correlates with the independent variable (memory specificity). As shown in Table 3, both rumination and permanent change correlated significantly with memory specificity. Step 2 involves predicting the dependent variable with the independent variable alone, and Step 3 involves predicting the dependent variable with both the independent variable and the mediator. Table 4 summarizes the results for Steps 2 and 3. Mediation is demonstrated when the amount of variance explained by the independent variable is smaller when the mediator is included in the regression function compared with when the independent variable is considered alone. This was the case for both hypothesized mediators. For both PTSD and depression severity, the amount of variance explained by memory specificity was reduced when rumination or perceived permanent change was included in the regression, as indicated by significant Sobel tests (all $ps < .042$).

Discussion

In this study, we aimed to investigate the relationship between overgeneral autobiographical memory retrieval at 2-week post-trauma and acute and chronic psychological disorders after a traumatic experience. At 2 weeks, assault survivors with ASD generated fewer specific autobiographical memories in the AMT than participants without these disorders, replicating findings in other trauma populations (Harvey et al., 1998; Kangas et al., 2005). Furthermore, participants who had major depression after the trauma showed lower memory specificity compared with those who did not develop depression. These results are in line with a recent study by van Minnen et al. (2005), who found that low memory specificity predicted the exacerbation of depressive mood after a stressful event, and with previous research showing OGM in depressed people with a history of trauma (Hermans et al., 2004;

Table 4
Results of Regression Analyses Testing Whether Rumination and Permanent Change Mediate the Relationship Between Memory Specificity at 2 Weeks and PTSD and Depression Severity at 6 Months

Predictor in regression	Regression coefficient for memory specificity (independent variable)	p	For regression analyses including mediators		
			% total effect that is mediated	Sobel test	p
Prediction of PTSD severity (PSS–I) at 6 months					
Memory specificity only	−.238	.001	N/A	N/A	N/A
Memory specificity and rumination	−.137	.038	30.78	−2.46	.014
Memory specificity and permanent change	−.133	.053	48.11	−2.73	.006
Prediction of depression severity (SCID) at 6 months					
Memory specificity only	−.195	.001	N/A	N/A	N/A
Memory specificity and rumination	−.151	.047	18.21	−2.02	.042
Memory specificity and permanent change	−.115	.128	36.33	−2.73	.006

Note. Memory specificity is indexed as the percentage of first memories that were specific. Negative regression coefficients indicate positive associations with overgeneral memory. PTSD = posttraumatic stress disorder; PSS–I = PTSD Symptom Scale—Interview Version; N/A = not applicable as there was no mediator entered; SCID = Structured Clinical Interview for *DSM–IV*.

Raes, Hermans, Williams, Beyers, et al., 2006). The lack of group differences in OGM between assault survivors with and without assault-related specific phobia is also consistent with the literature, as previous studies found no relationship between OGM and anxiety disorders other than PTSD (e.g., Wenzel et al., 2002, 2004). Thus, the findings support the specificity of OGM to depression and ASD/PTSD. To our knowledge, this is the first study to demonstrate specificity in the same population.

Low memory specificity at 2 weeks predicted both chronic PTSD and major depression at 6 months, and predicted over and above what could be predicted from initial symptom severity, regardless of whether diagnosis or symptom severity at 6 months was used as outcomes. The current result is in contrast to a recent study of cancer patients (Kangas et al., 2005), which found that deficits in the retrieval of specific memories were not predictive of subsequent PTSD. However, the smaller sample size and attrition during follow-up may have affected these results.

The present study did not find any effects of AMT cue valence. The literature shows inconsistent findings (van Vreeswijk & de Wilde, 2004), with some studies reporting patients to be less specific in response to positive cues (McNally, Litz, Prassas, Shin, & Weathers, 1994; Williams & Scott, 1988), whereas others report OGM effects in response to negative cues, rather than positive and neutral cues (Jones et al., 1999; Mackinger, Pachinger, Leibetseder, & Fartacek, 2000). In line with our findings, McNally et al. (1995), Goddard et al. (1996), Schönfeld and Ehlers (2006), and Schönfeld et al. (2007) reported OGM in patients with PTSD and depression in response to both positive and negative cues.

In the present study, we also explored correlates of OGM, and potential moderator and mediators of the relationship between OGM and posttrauma psychopathology. Sex, ethnicity, and history of childhood abuse did not interact with OGM in predicting PTSD or depression at 6 months in the present sample. There was no relationship between OGM and childhood abuse and number of adult traumas, but OGM correlated with the severity of the recent assault. Together, these results point to a relationship between OGM and trauma, but they also suggest that OGM does not necessarily develop after trauma. The results are in line with a recent review that concluded that trauma exposure per se is unlikely to be the primary mechanism leading to overgenerality (Moore & Zoellner, 2007). Instead, OGM may characterize those survivors of childhood or adult trauma who develop psychopathology. Some previous studies also failed to find a significant association between childhood trauma and OGM (Johnson et al., 2005; Wessel et al., 2001). However, our study only included a limited assessment of childhood abuse, and some participants may not have disclosed abuse or other previous traumas as no treatment was offered. A more detailed assessment of childhood abuse would have allowed us to investigate the relationship more comprehensively and conclusively.

Recent studies by Dalgleish et al. (2007) and Raes, Hermans, Williams, Demyttenaere, et al. (2006) have suggested that the AMT is sensitive to individual differences in executive capacity and depression-related reduction in working memory. In line with their findings, OGM correlated with a measure of verbal intelligence in the current study. However, OGM predicted both PTSD and depression at 6 months over and above what could be predicted from intelligence.

A history of major depression prior to the assault moderated the relationship between OGM and major depression, but not PTSD, at follow-up. These results are in accord with the view that prior depressive episodes constitute a vulnerability factor that may predispose individuals to more general memory retrieval (Gibbs & Rude, 2004). There is evidence that individuals remitted from depression show more overgeneral autobiographical memory retrieval than never-depressed controls (Kuyken & Brewin, 1995; Mackinger et al., 2000). The present results are of interest in the discussion about findings that risk of further depressive episodes increases with every consecutive episode and that successive episodes of major depression require less and less external provocation by stressful life events (e.g., Kendler, Thornton, & Gardner, 2000). It has been hypothesized that in people with repeated episodes of major depression, new episodes become increasingly mediated by internal processes, such as ruminative thinking cycles reactivated by dysphoric mood (e.g., Ma & Teasdale, 2004). OGM

may be an indicator of this cognitive vulnerability to recurrence of depression.

As expected, low memory specificity was related to greater rumination about the trauma and greater perceived permanent change. These variables were also shown to fully or partly mediate the relationship between OGM and both PTSD and depression at follow-up. Previous research has shown that rumination increases the tendency to retrieve overgeneral memories (see Park, Goodyer, & Teasdale, 2004). This is consistent with Williams's (1996) hypothesis that self-focused rumination contributes to a block of specific autobiographical retrieval ("mnemonic interlock"). Previous studies have found associations between OGM and rumination in depression (Raes, Hermans, Williams, Beyers, et al., 2006) and rumination about the trauma in trauma survivors (Schönfeld & Ehlers, 2006). Like the present study, Raes, Hermans, Williams, Beyers, et al. (2006) also found that rumination partly mediated the relationship between OGM and depression 7 months later, although they used a different measure of rumination that was not trauma related. The result that perceived permanent change mediated the effects of OGM on PTSD severity is consistent with Schönfeld and Ehlers's (2006) suggestion that inability to retrieve specific memories from one's life may enhance the sense that one has changed for the worse as a person, which in turn contributes to the maintenance of PTSD (Dunmore et al., 2001; Ehlers et al., 2000).

Although the study has several strengths, including a large sample size, recruitment soon after a trauma, and a prospective longitudinal design, it also has several limitations. First, it remains unclear whether low autobiographical memory specificity is a predisposing factor or a consequence of the trauma. For practical reasons, it was not possible to assess OGM prior to the assault. Hence, future research will need to address whether OGM effects are present prior to the development of psychopathology (see Bryant et al., 2007). Second, there was high comorbidity among the disorders, which is common in survivors of violent assault (e.g., Kilpatrick et al., 2003). This raises the question of whether the outcomes studied represented different disorders. Prior studies suggest that PTSD and depression are best conceptualized as related, but distinct, responses to trauma (Blanchard, Buckley, Hickling, & Taylor, 1998; Breslau, Chase, & Anthony, 2002; O'Donnell, Creamer, Pattison, & Atkin, 2004). High comorbidity makes it difficult to demonstrate specificity. Nevertheless, our findings show that OGM was specific to depression and ASD, whereas participants with phobia did not differ from participants without phobia in autobiographical memory retrieval. It is noteworthy that the study was conservative in testing specificity, as it allowed comorbid ASD and specific phobia. If we had excluded comorbid cases with ASD from the phobia group, we would have biased the results in favor of finding specificity of OGM for ASD as opposed to phobia. However, the high comorbidity may have compromised the ability to detect differences between depression and PTSD. Nevertheless, given the high comorbidity between the disorders, as well as the symptom overlap in diagnostic criteria between PTSD and depression, and between PTSD and phobia, it might be useful for future researchers to adopt a dimensional rather than a categorical approach. This could involve using symptom clusters across disorders (e.g., phobic responses, numbing, and reexperiencing) rather than symptom dimensions that are based on different diagnostic entities as outcomes. Third, diagnoses at 6

months were based on a SCID interview conducted over the phone, rather than a face-to-face diagnostic interview. Such telephone interviews may make participants somewhat less forthcoming in providing information about their symptoms than face-to-face interviews. However, the interviewer had already established a relationship with the participants at the first assessment, which makes it unlikely that participants withheld information from her later. Fourth, the sample comprised nearly exclusively survivors of physical assaults. The results are therefore limited to survivors of physical assaults, and they need to be replicated with other trauma populations.

In the present study, we pointed to an "added value" of OGM in the prediction of chronic PTSD and depression over and above initial symptom severity. However, the observed effect sizes were in the small to medium range. OGM only predicted an additional 4%–6% of the variance of depression and PTSD at follow-up. Nevertheless, the results are of interest, as initial symptom severity is a strong predictor of PTSD (e.g., Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992), and very few variables have been shown to predict PTSD over and above initial symptoms. These effect sizes observed in this study are in the range of the best replicated predictors of PTSD according to a recent meta-analysis by Ozer, Best, Lipsey, and Weiss (2003). Transformed into binominal effect sizes, the results suggest that a trauma survivor high in OGM has an approximately 20% greater chance of having PTSD or depression at 6 months than a survivor low in OGM, an effect size that exceeds that of many standard medical interventions (Rosnow & Rosenthal, 2003).

Interestingly, the two factors that were found to partly mediate the effect of OGM on future depression and PTSD, rumination about the trauma and perceived permanent change, are among the few variables that have been shown to have incremental predictive validity over and above initial symptoms (Dunmore et al., 2001; Ehlers, Mayou, & Bryant, 1998). Overall, given the small incremental predictive power and the results of the mediator analyses, practical applications of the AMT as a tool for identifying traumatized people at risk of chronic PTSD appear limited. Questionnaire measures, such as the rumination or perceived permanent change scales, are easier to administer and score than the AMT and may thus be more useful for screening programs.

Nevertheless, the results point to potential therapeutic strategies. It is possible that training in the accessibility of specific memories would help people break through the capture and rumination cycle (Williams et al., 2006) and help people put the trauma into perspective by integrating it into the context of other experiences. Our data would suggest that such training may be potentially relevant for trauma survivors with episodes of past depression. There is evidence that OGM is modifiable (McBride, Segal, Kennedy, & Gemar, 2007; Watkins, Teasdale, & Williams, 2000). Alternatively, one may argue that the most promising way to reduce OGM and poor posttrauma adaptation would be to target rumination. In a series of studies, Watkins and colleagues (e.g., Watkins & Teasdale, 2001; Watkins et al., 2000) found that reducing rumination leads to less overgeneral memory retrieval in depressed patients. Reducing rumination is a component of some effective treatments for PTSD (e.g., Ehlers, Clark, Hackmann, McManus, & Fennell, 2005). It would also be of interest to study whether treatment procedures used in effective treatments of PTSD to

counteract perceived permanent change, such as "reclaiming your life" assignments (Ehlers & Clark, 2000), change OGM bias.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173–1182.

Blanchard, E. B., Buckley, T. C., Hickling, E. J., & Taylor, A. E. (1998). Posttraumatic stress disorder and comorbid major depression: Is the correlation an illusion? *Journal of Anxiety Disorders, 12,* 21–37.

Breslau, N., Chase, G. A., & Anthony, J. C. (2002). "A second look at comorbidity in victims of trauma: The posttraumatic stress disorder-major depression connection." Reply. *Psychological Medicine, 32,* 1480–1483.

Bright, P., Jaldow, E., & Kopelman, M. D. (2002). The National Adult Reading Test as a measure of premorbid intelligence: A comparison with estimates derived from demographic variables. *Journal of the International Neuropsychological Society, 8,* 847–854.

Brittlebank, A. D., Scott, J., Williams, J. M., & Ferrier, I. N. (1993). Autobiographical memory in depression: State or trait marker? *British Journal of Psychiatry, 162,* 118–121.

Brown, G. D. (1984). A frequency count of 190,000 words in the London–Lund Corpus of English Conversation. *Behavior Research Methods, Instruments, & Computers, 16,* 502–532.

Bryant, R. A., & Harvey, A. G. (2000). *Acute stress disorder: A handbook of theory, assessment, and treatment.* Washington, DC: American Psychological Association.

Bryant, R. A., Sutherland, K., & Guthrie, R. M. (2007). Impaired specific autobiographical memory as a risk factor for posttraumatic stress after trauma. *Journal of Abnormal Psychology, 116,* 837–841.

Burke, M., & Mathews, A. (1992). Autobiographical memory and clinical anxiety. *Cognition and Emotion,* 23–35.

Clohessy, S., & Ehlers, A. (1999). PTSD symptoms, response to intrusive memories and coping in ambulance service workers. *British Journal of Clinical Psychology, 38,* 251–265.

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

Crawford, J. R., Parker, D. M., Allan, K. M., Jack, A. M., & Morrison, F. M. (1991). The Short NART: Cross-validation, relationship to IQ and some practical considerations. *British Journal of Clinical Psychology, 30,* 223–229.

Crawford, J. R., Steward, L. E., Garthwaite, P. E., Parker, D. M., & Besson, J. A. O. (1988). The relationship between demographic variables and NART performances in normal subjects. *British Journal of Clinical Psychology, 27,* 181–182.

Dalgleish, T., Perkins, N., Williams, J. M. G., Golden, A.-M. J., Barret, L. F., Barnard, P. J., et al. (2007). Reduced specificity of autobiographical memory and depression: The role of executive processes. *Journal of Experimental Psychology: General, 136,* 23–42.

Dalgleish, T., Tchanturia, K., Serpell, L., Hems, S., Yiend, J., de Silva, P., et al. (2003). Self-reported parental abuse relates to autobiographical memory style in patients with eating disorders. *Emotion, 3,* 211–222.

Dunmore, E., Clark, D. M., & Ehlers, A. (2001). A prospective investigation of the role of cognitive factors in persistent posttraumatic stress disorder (PTSD) after physical or sexual assault. *Behaviour Research and Therapy, 39,* 1063–1084.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38,* 319–345.

Ehlers, A., Clark, D. M., Hackmann, A., McManus, F., & Fennell, M.

(2005). Cognitive therapy for PTSD: Development and evaluation. *Behaviour Research and Therapy, 43,* 413–431.

Ehlers, A., Maercker, A., & Boos, A. (2000). Posttraumatic stress disorder following political imprisonment: The role of mental defeat, alienation, and perceived permanent change. *Journal of Abnormal Psychology, 109,* 45–55.

Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology, 107,* 508–519.

Ehring, T., Ehlers, A. & Glucksman, E. (2006). Contribution of cognitive factors to the prediction of posttraumatic stress disorder, phobia and depression after road traffic accidents. *Behaviour Research and Therapy, 44,* 1699–1716.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured Clinical Interview for DSM–IV Axis I Disorders.* Washington, DC: American Psychiatric Press.

Foa, E. B., Ehlers, A., Clark, D. M., Tolin, D. F., & Orsillo, S. M. (1999). The Posttraumatic Cognitions Inventory (PTCI): Development and validation. *Psychological Assessment, 11,* 303–314.

Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing posttraumatic stress disorder. *Journal of Traumatic Stress, 6,* 459–473.

Gibbs, B. R., & Rude, S. S. (2004). Overgeneral autobiographical memory as depression vulnerability. *Cognitive Therapy and Research, 28,* 511–526.

Goddard, L., Dritschel, B., & Burton, A. (1996). Role of autobiographical memory in social problem solving and depression. *Journal of Abnormal Psychology, 105,* 609–616.

Harvey, A. G., Bryant, R. A., & Dang, S. T. (1998). Autobiographical memory in acute stress disorder. *Journal of Consulting and Clinical Psychology, 66,* 500–506.

Henderson, D., Hargreaves, I., Gregory, S., & Williams, J. M. (2002). Autobiographical memory and emotion in a nonclinical sample of women with and without a reported history of childhood sexual abuse. *British Journal of Clinical Psychology, 41,* 129–141.

Herman, J. L. (1992). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress, 5,* 377–391.

Hermans, D., van den Broeck, K., Belis, G., Raes, F., Pieters, G., & Eelen, P. (2004). Trauma and autobiographical memory specificity in depressed inpatients. *Behaviour Research and Therapy, 42,* 775–789.

Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). *Cognitive vulnerability to depression.* New York: Guilford Press.

Johnson, R. J., Greenhoot, A. F., Glisky, E., & McCloskey, L. A. (2005). The relations among abuse, depression, and adolescents' autobiographical memory. *Journal of Clinical Child and Adolescent Psychology, 34,* 235–247.

Jones, B., Heard, H., Startup, M., Swales, M., Williams, J. M. G., & Jones, R. S. P. (1999). Autobiographical memory and dissociation in borderline personality disorder. *Psychological Medicine, 29,* 1397–1404.

Kangas, M., Henry, J. L., & Bryant, R. A. (2005). A prospective study of autobiographical memory and posttraumatic stress disorder following cancer. *Journal of Consulting and Clinical Psychology, 73,* 293–299.

Kendler, K. S., Karkowski, L. M., & Prescott, C. A. (1999). Causal relationship between stressful life events and the onset of major depression. *American Journal of Psychiatry, 156,* 837–841.

Kendler, K. S., Thornton, L. M., & Gardner, C. O. (2000). Stressful life events and previous episodes in the etiology of major depression in women: An evaluation of the "kindling" hypothesis. *American Journal of Psychiatry, 157,* 1243–1251.

Kilpatrick, D. G., Ruggiero, K. J., Acierno, R., Saunders, B. E., Resnick, H. S., & Best, C. L. (2003). Violence and risk of PTSD, major depression, substance abuse/dependence, and comorbidity: Results from the National Survey of Adolescents. *Journal of Consulting and Clinical Psychology, 71,* 692–700.

Kubany, E. S., Haynes, S. N., Leisen, M. B., Owens, J. A., Kaplan, A. S.,

Watson, S. B., et al. (2000). Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: The Traumatic Life Events Questionnaire. *Psychological Assessment, 12,* 210–224.

Kuyken, W., & Brewin, C. R. (1995). Autobiographical memory functioning in depression and reports of early abuse. *Journal of Abnormal Psychology, 104,* 585–591.

Ma, S. H., & Teasdale, J. D. (2004). Mindfulness-based cognitive therapy for depression: Replication and exploration of differential relapse prevention effects. *Journal of Consulting and Clinical Psychology, 72,* 31–40.

Mackinger, H. F., Pachinger, M. M., Leibetseder, M. M., & Fartacek, R. R. (2000). Autobiographical memories in women remitted from major depression. *Journal of Abnormal Psychology, 109,* 331–334.

McBride, C., Segal, Z., Kennedy, S., & Gemar, M. (2007). Changes in autobiographical memory specificity following cognitive behaviour therapy and pharmacotherapy for major depression. *Psychopathology, 40,* 147–152.

McKinnon, D. P., & Dwyer, J. H. (1993). Estimating mediated effects in prevention studies. *Evaluation Review, 17,* 144–158.

McKinnon, D. P., Fairchild, A. J., & Fritz, M. S. (2007). Mediation analysis. *Annual Review of Psychology, 58,* 593–614.

McNally, R. J., Lasko, N. B., Macklin, M. L., & Pitman, R. K. (1995). Autobiographical memory disturbance in combat-related posttraumatic stress disorder. *Behaviour Research and Therapy, 33,* 619–630.

McNally, R. J., Litz, B. T., Prassas, A., Shin, L. M., & Weathers, F. W. (1994). Emotional priming of autobiographical memory in posttraumatic stress disorder. *Cognition and Emotion, 8,* 351–367.

Moore, S. A., & Zoellner, L. A. (2007). Overgeneral autobiographical memory and traumatic events: An evaluative review. *Psychological Bulletin, 133,* 419–437.

Murray, J., Ehlers, A., & Mayou, R. A. (2002). Dissociation and posttraumatic stress disorder: Two prospective studies of motor vehicle accident survivors. *British Journal of Psychiatry, 180,* 363–368.

Nelson, H. (1991). *National Adult Reading Test (NART) test manual.* Windsor, United Kingdom: NFER-Nelson.

Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: The 1989 Loma Prieta earthquake. *Journal of Personality and Social Psychology, 61,* 115–121.

O'Donnell, M. L., Creamer, M., Pattison, P., & Atkin, C. (2004). Psychiatric morbidity following injury. *American Journal of Psychiatry, 161,* 507–514.

Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin, 129,* 52–73.

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

Peeters, F., Wessel, I., Merckelbach, H., & Boon-Vermeeren, M. (2002). Autobiographical memory specificity and the course of major depressive disorder. *Comprehensive Psychiatry, 43,* 344–350.

Raes, F., Hermans, D., Williams, J. M., Beyers, W., Eelen, P., & Brunfaut, E. (2006). Reduced autobiographical memory specificity and rumination in predicting the course of depression. *Journal of Abnormal Psychology, 115,* 699–704.

Raes, F., Hermans, D., Williams, J. M., Demyttenaere, K., Sabbe, B., Pieters, G., et al. (2006). Is overgeneral autobiographical memory an isolated memory phenomenon in major depression? *Memory, 14,* 584–594.

Resick, P. A., & Schnicke, M. K. (1993). *Cognitive processing therapy for rape victims.* Newbury Park, CA: Sage.

Rosnow, R. L., & Rosenthal, R. (2003). Effect sizes for experimenting psychologists. *Canadian Journal of Experimental Psychology, 57,* 221–237.

Rothbaum, B. O., Foa, E. B., Riggs, D. S., Murdock, T. B., & Walsh, W. (1992). A prospective examination of posttraumatic stress disorder in rape victims. *Journal of Traumatic Stress, 5,* 455–475.

Schönfeld, S., & Ehlers, A. (2006). Overgeneral memory in posttraumatic stress disorder extends to pictorial retrieval cues and correlates with self-perception. *Emotion, 6,* 611–622.

Schönfeld, S., Ehlers., A., Böllinghaus, I., & Rief, W. (2007). Overgeneral memory and suppression of trauma memories in posttraumatic stress disorder. *Memory, 15,* 339–352.

van Minnen, A., Wessel, I., Verhaak, C., & Smeenk, J. (2005). The relationship between autobiographical memory specificity and depressed mood following a stressful life event: A prospective study. *British Journal of Clinical Psychology, 44,* 405–415.

van Vreeswjik, M. F., & de Wilde, E. J. (2004). Autobiographical memory specificity, psychopathology, depressed mood, and the use of the Autobiographical Memory Test: A meta-analysis. *Behaviour Research and Therapy, 42,* 731–743.

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

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

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale* (3rd ed.). San Antonio, TX: The Psychological Corporation.

Wenzel, A., Jackson, L. C., & Holt, C. S. (2002). Social phobia and the recall of autobiographical memories. *Depression and Anxiety, 15,* 186–189.

Wenzel, A., & Jordan, J. (2005). Autobiographical memory in angry and anxious individuals. *Behaviour Research and Therapy, 43,* 1099–1109.

Wenzel, A., Werner, M. M., Cochran, C. K., & Holt, C. S. (2004). A differential pattern of autobiographical memory retrieval in social phobic and nonanxious individuals. *Behavioural and Cognitive Psychotherapy, 32,* 1–13.

Wessel, I., Meeren, M., Peeters, F., Arntz, A., & Merckelbach, H. (2001). Correlates of autobiographical memory specificity: The role of depression, anxiety and childhood trauma. *Behaviour Research and Therapy, 39,* 409–421.

Williams, J. M. G. (1996). Depression and the specificity of autobiographical memory. In D. C. Rubin (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 244–267). Cambridge, England: Cambridge University Press.

Williams, J. M. G. (2003). *Autobiographical Memory Test scoring manual.* Unpublished manuscript.

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

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

Williams, J. M. G., Chan, S., Crane, C., Barnhofer, T., Eade, J., & Healy, H. (2006). Retrieval of autobiographical memories: The mechanisms and consequences of truncated search. *Cognition and Emotion, 20,* 351–382.

Williams, J. M. G., Ellis, N. C., Tyers, C., Healey, H., Rose, G., & MacLeod, A. K. (1996). The specificity of autobiographical memory and imageability of the future. *Memory & Cognition, 24,* 116–125.

Williams, J. M. G., & Scott, J. (1988). Autobiographical memory in depression. *Psychological Medicine, 18,* 689–695.