



# Episodic Life Stress and the Development of Overgeneral Autobiographical Memory to Positive Cues in Youth

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## Abstract

Overgeneral autobiographical memory (OGM) has been established as a risk factor for depression in both youth and adults, but questions remain as to how OGM develops. Although theorists have proposed that the experience of stressful life events may contribute to the development of OGM, no studies have examined the impact of negative life events on prospective changes in OGM. The goal of the current study was to address this gap in the literature. Participants included 251 mothers and their biological children (aged 8–14 years old at the initial assessment). Using a multi-wave prospective design with assessments every 6 months for 2 years, we found that episodic life stress predicted prospective decreases in youths' autobiographical memory specificity to positive, but not negative, cues. This study supports theories proposing that negative life events may contribute to the development of OGM, but suggest that, in youth, the impact of life stress on OGM may be specific to positive rather than negative memories.

**Keywords** Overgeneral autobiographical memory · Life stress · Depression

According to cognitive theories of depression, the way in which an individual attends to, interprets, and remembers environmental stimuli contributes to the development and maintenance of depression (e.g., Beck 1987; Clark et al. 1999). One form of memory bias, termed overgeneral autobiographical memory (OGM), reflects difficulties in recalling specific autobiographical memories of events that occurred at a particular time and place (Williams et al. 2007a). For example, an OGM would be “I felt sad in school”, whereas a specific memory would be “I felt sad in class last Tuesday when I saw that I failed my math test”. There is growing evidence that depressed individuals have more OGM than healthy controls (see Van Vreeswijk and De Wilde 2004 for a review). Greater OGM also predicts prospective increases in

depressive symptoms (see Sumner et al. 2010 for a review) and maintenance of major depression (Hermans et al. 2008). Furthermore, although research on OGM has focused primarily on adults, recent research has begun to examine OGM as a marker of depression risk in children and adolescents as well. Consistent with what has been observed in adults, there is evidence that depressed youth recall fewer specific memories than never depressed youth (see Hitchcock et al. 2014b for a review). In addition, OGM prospectively predicts increases in depressive symptoms (see Hitchcock et al. 2014b for a review) and risk of recurrence for major depression (Sumner et al. 2011) in youth. It should be noted, however, that there is some evidence that the predictive validity of OGM in youth is stronger among those who also exhibit other risk factors, such as elevated levels of rumination or a family history of depression (Gutenbrunner et al. 2017; Rawal and Rice 2012).

Given evidence that OGM increases risk for depression in youth, it is important to examine how OGM develops. A number of theories have been offered for the development of OGM, many of which focus on the impact of negative life events. For example, according to the functional avoidance theory of OGM (Williams 1996), the experience of trauma in childhood may lead to decreased recall of specific negative memories as youth use avoidance as a coping mechanism. Indeed, there is evidence that youth exposed to trauma displayed heightened levels of OGM (Brennen et al. 2010; Stokes et al. 2004; Valentino

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et al. 2009), and a diagnosis of posttraumatic stress disorder (PTSD) is associated with less specific autobiographical memories (for reviews, see Moore and Zoellner 2007; Ono et al. 2015). Additionally, for individuals with PTSD, greater PTSD symptoms and cognitive avoidance are correlated with greater OGM (Schönfeld and Ehlers 2006; Schönfeld et al. 2007) providing evidence that trauma may lead to the development of OGM through avoidance of the traumatic memory. Although trauma is no longer hypothesized to be the sole predictor of OGM development, more recent models of OGM development still include the experience of negative life events as an important factor in the development of OGM (e.g., Williams et al. 2007b). However, to date, no studies have examined whether stressful life events actually contribute to prospective increases in OGM. Although there is evidence that a history of traumatic events is associated with the presence of OGM (e.g., Sumner 2012; Williams et al. 2007b), these studies have been cross-sectional, and are therefore unable to examine actual changes in OGM. Furthermore, although chronic and episodic stressors have been shown to interact with elevated levels of OGM to predict depression (Anderson et al. 2010; Gibbs and Rude 2004; Sumner et al. 2011), no studies to date have examined whether the experience of stressful life events is associated with changes in OGM.

When examining the development of OGM in youth, it may be important to examine OGM to positive and negative cues separately. Specifically, although confirmatory factor analyses have indicated that, in adults and adolescents, OGM can be represented as a single-factor that encompasses the recall of both positive and negative memories (Griffith et al. 2009; Griffith et al. 2012; Heron et al. 2012) and depressed adults and adolescents have been shown to have greater OGM to both positive and negative cues (Champagne et al. 2016; Park et al. 2002; Van Vreeswijk and De Wilde 2004), the literature on OGM in children is less consistent. For example, whereas one study found that children (ages 9–13) with a history of depression exhibited more OGM to positive and negative cues than never depressed children (Vrielynck et al. 2007), another study found that children and early adolescents (ages 8–14) at risk for depression based on maternal history of major depression exhibited more OGM to negative but not positive cues than children of never depressed mothers (Woody et al. 2015). Similarly, whereas one study found that OGM to negative but not positive cues predicted future depressive symptoms in 10–18 year-old youth (Rawal and Rice 2012), another study found that OGM to positive but not negative cues predicted increases in depressive symptoms for girls (age 11; Hipwell et al. 2011). Finally, in a general community sample, children (ages 7–8) exhibited greater OGM to negative cues compared to positive cues (Drummond et al. 2006) suggesting that OGM to positive and negative cues may not develop at the same time in children. Indeed, in line with the functional avoidance theory, theorists suggest that OGM may develop in children first for

negative memories before generalizing to positive memories (e.g., Drummond et al. 2006; Hitchcock et al. 2014b; Williams et al. 2007b). This is suggested to occur because, although the avoidance of negative memories may help to prevent negative emotion associated with these memories, it is possible that positive cues may also trigger negative memories, and therefore avoidance of all memories is necessary to truly avoid the negative emotion. Given all of this, we examined the impact of negative life events on OGM to positive and negative cues separately in this study.

The primary goal of the current study, therefore, was to examine whether stressful life events were associated with prospective changes in children's OGM over a 24-month follow-up period. We hypothesized that exposure to elevated levels of stress would be associated with decreased levels of autobiographical memory specificity. Additionally, given the mixed literature on whether OGM to both positive and negative cues is observed as a risk factor for depression in youth, coupled with evidence that OGM to negative and positive cues may not develop at the same time, we examined memories for positive and negative cues separately. A precondition for these analyses is that OGM to negative and positive cues are actually separate constructs in this age range. To confirm this, we conducted preliminary analyses to determine whether a two-factor model of OGM did indeed provide a better fit to our data than a one factor model in which OGM to negative and positive cues simply loaded onto a single common factor. Because this is the first study to date to examine the impact of stressful life events on prospective decreases in number of specific autobiographical memories, we did not make specific hypotheses on the impact of life stress on negative versus positive specific memories. A secondary goal of the study was to evaluate the potential role of maternal history depression. Specifically, a number of studies have shown that children of depressed mothers are at increased risk for various forms of cognitive vulnerability including OGM (Woody et al. 2015; see also Garber and Flynn 2001; Garber and Robinson 1997; Jaenicke et al. 1987). In the current study, therefore, we examined whether the impact of stressful life events on children's OGM would be maintained after statistically controlling for the influence of mothers' depression history as well as whether mothers' history of major depression would moderate this relation such that the impact of stressful life events on children's OGM would be stronger among children already at high risk due to a history of depression in their mothers.

## Method

### Participants

Participants included 251 mother-child dyads recruited from the community starting in 2010 as part of a larger study on risk

for the intergenerational transmission of depression (for details, see Burkhouse et al. 2015; Feurer et al. 2016; Woody et al. 2015). To be eligible for the study, mothers had to have either experienced an episode of major depressive disorder (MDD) during their child's lifetime ( $n = 129$ ) or have no lifetime history of any Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (DSM-IV; American Psychiatric Association 1994) mood disorder ( $n = 122$ ). Exclusion criteria for mothers in both groups included symptoms of schizophrenia, alcohol or substance dependence within the last six months, or a history of bipolar disorder. Children were required to be the biological child of the participating mother, be between 8 and 14 years old at the initial assessment, and not have a learning or developmental disability per mothers' report. Only one child per family was included. If there was more than one eligible child, one was chosen at random for participation. The average age of children in this study was 11.40 years, ( $SD = 1.93$ ) and 51.4% were female. In terms of race/ethnicity, 81.3% were Caucasian, 4.8% were African American, 10.8% were biracial, and 3.1% identified as another race. For mothers, the average age was 40.38 ( $SD = 6.80$ ), 87.3% were Caucasian, 4.8% were African American, 4.0% were biracial, and 3.9% identified as another race. The median family income was \$50,001 - \$55,000.

## Measures

Mothers' and children's histories of MDD and other Axis-I disorders were assessed at the initial time point using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al. 1995) and the Schedule for Affective Disorders and Schizophrenia for School Age Children – Present and Lifetime Version (K-SADS-PL; Kaufman et al. 1997), respectively. Within the sample, 129 mothers met for a history of MDD during their child's life, 69 of whom had a history of recurrent MDD and 21 of whom met criteria for current MDD at the initial assessment. Additionally, 18 youth had a lifetime history of MDD at the initial assessment (16 of whom had mothers with a history of MDD). Lifetime rates of anxiety disorders in mothers were: 27 with social phobia (26 from the MDD group), 25 with post-traumatic stress disorder (PTSD; 24 from the MDD group), 22 with panic disorder (21 from the MDD group), 12 with obsessive-compulsive disorder (OCD; 10 from the MDD group), 10 with agoraphobia (8 from the MDD group), and 3 with generalized anxiety disorder (GAD; all from the MDD group). Finally, lifetime rates of anxiety disorders in youth were: 13 with social phobia (11 children of mothers with MDD), 12 with separation anxiety disorder (10 children of mothers with MDD), 7 with GAD (5 children of mothers with MDD), 4 with OCD (4 children of mothers with MDD), 1 with panic disorder (whose mother had MDD), and 1 with PTSD (whose mother had MDD). A subset of 20 SCIDs and 20 K-SAD-PLs was coded by a separate

interviewer to assess inter-rater reliability for diagnoses of MDD and anxiety disorders, yielding excellent kappa coefficients (all  $\kappa$ 's = 1.00).

Children's autobiographical memory was assessed using the Autobiographical Memory Test (AMT; Williams and Broadbent 1986). The AMT task consists of 10 emotional words, 5 positively valenced (happy, surprised, safe, successful, interested) and 5 negatively valenced (sad, lonely, hurt, careless, angry). Words were presented to participants on index cards, alternating between positive and negative words, and participants were asked to retrieve a specific memory for each cue word. To ensure that participants understood the task, all participants completed three practice trials involving neutral words (car, tree, chair) with feedback. During the practice trials, participants were instructed to not give the same memory for more than one cue word. Participants were given 60 s to retrieve a memory. All responses were audiotaped, transcribed, and then coded as specific or overgeneral (categoric or extended). A specific memory is defined as a single event lasting less than one day whereas a categoric memory refers to a generic collection or class of events and extended memory is of a single event lasting more than one day. An example of scoring of a specific response to the cue word "Happy" was, "I was happy when I scored a goal in my game on Saturday". This is a specific memory because the memory is of a particular place and time. As noted, an OGM response to the same cue was, "I am happy when I play with my friends". This was coded as overgeneral because it is a categorical memory. If participants provided a response that included semantic information but no personal memory (i.e., semantic associates), this was coded as a semantic associate. Finally, if participants were unable to retrieve a memory or if they provided the same memory for more than one cue, this was coded as no response. The AMT was administered at each of the assessment points and summary scores were calculated for each participant at each time point, reflecting the number of specific memories provided for each cue valence. All responses were coded by two independent raters and interrater reliability for codes of specific memories was good ( $\kappa = .81$ ). Any discrepancies across raters were discussed until consensus was achieved.

Youth's episodic life stress exposure was assessed at each time point using the UCLA Life Stress Interview for Children (LSI-C; Adrian and Hammen 1993), which is a semi-structured interview modeled after contextual threat interviews (Brown and Harris 1978). At the initial assessment, mother-child dyads were interviewed separately about any episodic stressful life events (e.g., a fight with a friend, failing an exam, a death in the family) across a variety of domains that may have occurred in the 6 months prior to the assessment. For the 6, 12, 18, and 24-month follow-up assessments, participants were asked about any episodic stressful events since the date of their last assessment. If the dyad missed an assessment, the LSI-C focused on stress experienced during

the entire time between assessments instead of just the most recent 6-month interval. In these cases, any events reported before the date of their missed appointment were summed separately from the rest of the events reported at that time period, in order to back-date the events to the appropriate time point. For any reported events, the interviewer probed further to gain contextual information about the timing, duration, and consequences of each event. Each reported event was then presented, devoid of any subjective information, to a team of 4–7 coders who assigned a negative impact threat rating to each event. Any coding discrepancies across team members were discussed until consensus was achieved. These threat ratings ranged from “1”, which implied no stress (e.g., parent changed jobs, but had no impact on the amount of time child saw them or the family income), to “5” for events characterized by severe stress and a significant impact (e.g., primary caregiver passed away unexpectedly). Coders utilized examples of “anchor” events established from previously coded events to help guide coding of the contextual negative impact of stressful life events. Throughout the course of the coding of these life stress events for the current study, examples were added to the list of anchors to ensure that similar life stress events were coded in the same manner, and to prevent coder drift. Each stressor was also coded according to the content of the event, and was classified as either “interpersonal” or “non-interpersonal”. Of the reported life events, 78.01% were classified as “interpersonal”, and 21.99% were classified as “non-interpersonal”. Stress scores for each time point were summed together to calculate the total amount of episodic stress reported at each assessment. Prior to summing the stress scores, the contextual negative impact scale of 1–5 was recoded to 0–4. This was done so that life events with a score of “1” that reflected no stress would not artificially inflate the overall stress score. It is important to note that the stress scores were a summation of the assigned contextual stress scores for each time point, rather than a frequency count of life stressors. Participants reported an average of 3.37 ( $SD = 2.30$ ;  $Range = 0–13$ ) stressful life events at each assessment, and the average overall stress score for each time point was 2.31 ( $SD = 2.27$ ;  $Range = 0–18.50$ ). Finally, to assess inter-rater reliability, a subset of 50 life stress events were coded by an independent team of coders. Inter-rater reliability was excellent for assigned contextual stress scores ( $ICC = .91$ ), as well as for classification of life stressors as interpersonal or non-interpersonal ( $\kappa = .85$ ).

## Procedure

Potential participants were recruited through a variety of means (e.g., newspaper and bus ads, flyers) advertising a study for mothers with and without a history of depression and their biological children. Interested individuals who called in to the laboratory were screened over the telephone to

determine potential eligibility based on the inclusion/exclusion criteria noted above. At the initial assessment, informed consent and assent were obtained from both the mother and the child. Next, the SCID-I was administered to mothers to assess for their lifetime history of MDD and other Axis-I disorders. In addition, youth completed the AMT. Finally, the LSI-C was administered separately to the mother and the child to assess episodic stress in the child’s life. The AMT was administered first to ensure that children’s memory recall was not impacted by the content of the other assessments.

After the initial assessment, mothers and their children came back to the lab for 6, 12, 18, and 24-month follow-up assessments. Of the 251 participants who completed the Time 1 assessment, 211 (84.1%) completed Time 2, 197 (78.5%) completed Time 3, 170 (67.7%) completed Time 4, and 174 (69.3%) completed Time 5. Completion rates did not differ across the two mother MDD groups at any time point. At each follow-up appointment, the AMT was again administered to the child, and youth’s exposure to episodic life stress was assessed using the LSI-C. Participants were paid \$75 for their participation in the initial assessment, and an additional \$50 for the completion of each follow-up assessment. All study procedures were approved by Binghamton University’s Institutional Review Board.

## Analytic Plan

**Preliminary Analyses** To first confirm that OGM to positive and negative cues are distinct constructs in our sample, we conducted a confirmatory factor analysis. Two separate models were compared. In the Separate Constructs model, the number of specific memories recalled for positive cues at each time point were specified to load onto one latent variable (Positive OGM) and the number of specific memories recalled for negative cues at each time point were specified to load onto a different latent variable (Negative OGM). These latent variables were allowed to correlate and the error terms for OGM to positive and negative and at each time point were allowed to correlate (e.g., the error term for Time 1 OGM to positive cues was allowed to correlate with the error term for Time 1 OGM to negative cues). This model was compared to a Single Construct model in which the correlation between the Positive and Negative OGM latent variables was set to equal 1. This allowed us to conduct a nested model comparison to determine whether the Single Constructs Model provided a better fit to the data than the Separate Constructs model.

**Primary Analyses** Next, we used hierarchical linear modeling (HLM; Raudenbush and Bryk 2002; Raudenbush et al. 2004) to test the hypothesis that elevated levels of episodic stress would predict prospective increases in children’s OGM (i.e., decreases in autobiographical memory specificity) and to

determine whether the impact of stress was moderated by mothers’ history of MDD. We ran separate models for number of specific memories to negative and positive cues. The Level 1 model for these HLM analyses was:

$$OGM_{ij} = \pi_{0j} + \pi_{1j}(OGM_{t-1ij}) + \pi_{2j}(\text{Episodic Stress}_{ij}) + e_{ij}$$

where  $OGM_{ij}$  represents the number of specific autobiographical memories (negative or positive) reported by the youth at time  $t$  for assessment  $i$  and participant  $j$ ,  $OGM_{t-1ij}$  represents the number of specific memories (negative or positive) reported by the youth at time  $t-1$  for assessment  $i$  and participant  $j$ , and  $\text{Episodic Stress}_{ij}$  represents the youth’s total level of stress between time  $t-1$  and time  $t$  for assessment  $i$  and participant  $j$ . In addition,  $\pi_{0j}$  is the  $OGM_{ij}$  intercept,  $\pi_{1j}$  is the slope of the relation between the number of youth’s specific memories between time  $t$  and time  $t-1$  at each assessment  $i$  for participant  $j$  (i.e., the autocorrelation),  $\pi_{2j}$  is the slope of the relation between youth’s total stress and number of specific memories at time  $t$ , and  $e_{ij}$  represents the error term.

The Level 2 model was:

$$\begin{aligned} \pi_{0j} &= \beta_{00} + \beta_{01}(\text{MDD}) + r_{0j} \\ \pi_{1j} &= \beta_{10} + \beta_{11}(\text{MDD}) + r_{1j} \\ \pi_{2j} &= \beta_{20} + \beta_{21}(\text{MDD}) + r_{2j} \end{aligned}$$

where  $\beta_{01}$  is the cross-level interaction term representing the effect of maternal MDD history (absent vs. present in youth’s life) on the OGM intercept,  $\beta_{11}$  is the cross-level interaction term representing the effect of maternal MDD history on the slope of the relation between youth’s lagged and current number of specific memories, and  $\beta_{21}$  is the cross-level interaction representing the effect of maternal MDD on the slope of the relation between youth’s episodic stress and number of specific memories. Finally,  $\beta_{00}$ ,  $\beta_{10}$ , and  $\beta_{20}$  are the intercept terms for each of their respective equations, and  $r_{0j}$ ,  $r_{1j}$ , and  $r_{2j}$  are the error terms.

## Results

### Preliminary Analyses

A preliminary inspection of the data revealed significant skew for several variables ( $z > 3.29$ ; cf. Tabachnick and Fidell 2007). These variables were transformed prior to further analysis to satisfy assumptions of normality (square root: T1-T5 Stress; log10: T1-T5 AMT). Next, given the presence of some missing data (T1 AMT: 2.4%, T2 AMT: 17.1%, T3 AMT: 22.3%, T4 AMT: 37.1%, T5 AMT 34.3%, T2 Stress: 10.8%, T3 Stress: 18.7%, T4 Stress: 23.1%, T5 Stress: 30.7%), we examined whether the data were missing at random, thereby justifying the use of data imputation methods for

estimating missing values (cf. Schafer and Graham 2002). Little’s missing completely at random (MCAR) test, for which the null hypothesis is that the data are MCAR, was nonsignificant,  $\chi^2(1154) = 1160.63, p = .44$ . Therefore, maximum likelihood estimates of missing data were created and used for all analyses. Descriptive statistics for all study variables are presented in Table 1. To facilitate comparisons with other research, the means and standard deviations presented in the table are based on untransformed variables. The only demographic difference between the groups was that children of never depressed mothers were more likely to be Caucasian than were children of depressed mothers. Importantly, therefore, we should note that our results were maintained even when limiting our sample to Caucasians.

Prior to conducting our prospective analyses, we first tested whether our OGM data were best represented by a two-factor versus one-factor model using confirmatory factor analysis as described earlier. Fit indices indicated that the Separate Constructs model provided an excellent fit to the data (cf. Hu and Bentler 1999),  $\chi^2(29) = 44.15, p = .04$ , root mean square of approximation (RMSEA) = .05, comparative fit index (CFI) = .97, standardized root mean square residual (SRMR) = .04. Although the Single Construct model also provided excellent fit to the data,  $\chi^2(30) = 54.99, p = .004$ , RMSEA = .06, CFI = .96, SRMR = .04, a nested model comparison revealed that the Single Construct model provided a

**Table 1** Descriptive Statistics for Study Variables

	Never Depressed Mothers ( <i>n</i> = 122)	Depressed Mothers ( <i>n</i> = 129)	<i>r</i> <sub>effect size</sub>
Youth Age	11.40 (1.83)	11.40 (2.02)	.00
Youth Sex (% girls)	54.1%	48.8%	-.05
Youth Race (% Caucasian)	91.0%	72.1%	-.24**
T1 Specific Negative	3.90 (1.15)	3.47 (1.28)	-.18**
T2 Specific Negative	3.80 (1.14)	3.59 (1.16)	-.09
T3 Specific Negative	3.85 (0.93)	3.63 (1.30)	-.06
T4 Specific Negative	4.10 (0.96)	3.78 (1.01)	-.18**
T5 Specific Negative	4.16 (0.91)	4.02 (0.93)	-.10
T1 Specific Positive	3.60 (1.26)	3.54 (1.28)	-.03
T2 Specific Positive	3.51 (1.02)	3.45 (1.24)	.01
T3 Specific Positive	3.72 (1.08)	3.57 (1.21)	-.06
T4 Specific Positive	3.73 (0.99)	3.61 (1.05)	-.06
T5 Specific Positive	4.09 (0.89)	3.90 (0.88)	-.13*
T1 Stress	1.91 (1.66)	3.07 (2.76)	.25**
T2 Stress	1.49 (1.27)	2.79 (2.59)	.27**
T3 Stress	1.75 (1.86)	3.11 (2.43)	.31**
T4 Stress	1.61 (1.38)	2.49 (2.01)	.22**
T5 Stress	1.74 (1.68)	2.92 (2.40)	.27**

\*  $p < .05$ . \*\*  $p < .01$

significantly worse fit to the data than did the Separate Constructs model,  $\chi^2(1) = 10.83$ ,  $p = .001$ . These results support our analysis of OGM to positive versus negative cues separately.

## Primary Analyses

Next, we tested whether elevated levels of life stress prospectively predicted decreases in autobiographical memory specificity using the HLM models previously described. The results of these analyses are presented in Table 2. As can be seen in the table, stress did not significantly predict prospective changes in the number of children's specific memories for negative cues during the follow-up, nor was the mother MDD  $\times$  stress interaction significant. Turning next to memories to positive cues, we found that higher levels of stress prospectively predicted decreases in children's number of specific memories to positive cues during the follow-up. The mother MDD  $\times$  stress interaction was not significant, suggesting that high levels of stress had an equivalent impact on positive memory specificity for children with and without a maternal history of MDD.

Finally, exploratory analyses were conducted to examine whether any of the main effects of stress or interactions between maternal MDD and stress were moderated by child's age or sex. None of these analyses was significant (lowest  $p = .16$ ).

## Discussion

The goal of the current study was to examine whether stressful life events predict prospective changes in youth's

**Table 2** Summary of HLM analyses examining stress  $\times$  maternal MDD interactions predicting changes in youth's number of specific negative and positive memories

	Negative OGM		Positive OGM	
	<i>t</i>	<i>r</i> <sub>effect size</sub>	<i>t</i>	<i>r</i> <sub>effect size</sub>
OGM <sub>t</sub> Intercept ( $\pi_{0j}$ )				
Intercept ( $\beta_{00}$ )	-9.30**	-.51	-8.54**	-.48
MDD ( $\beta_{01}$ )	-0.08	-.005	-0.84	-.05
OGM <sub>t-1</sub> Slope ( $\pi_{1j}$ )				
Intercept ( $\beta_{10}$ )	2.22*	.14	3.19**	.20
MDD ( $\beta_{11}$ )	1.87	.12	0.67	.04
Episodic Stress Slope ( $\pi_{2j}$ )				
Intercept ( $\beta_{20}$ )	0.02	.002	-2.11*	-.13
MDD ( $\beta_{21}$ )	0.31	.02	1.25	.08

OGM Overgeneral Autobiographical Memory, MDD Mother history of major depressive disorder (yes, no)

\*  $p < .05$ . \*\*  $p < .01$

autobiographical memory specificity. Partially supporting our hypotheses, elevated levels of stressful life events predicted prospective decreases in children's autobiographical memory specificity for positive but not negative cues. These results were not moderated by maternal history of MDD or children's age or sex, suggesting that the impact of life stress on prospective changes in children's autobiographical memory specificity was similar across these groups.

This study is the first to examine whether stressful life events predict prospective changes in youth's autobiographical memory specificity. This is important for two reasons. First, although several studies have observed elevated levels of OGM for youth with a history of trauma (e.g., Brennen et al. 2010; Stokes et al. 2004; Valentino et al. 2009), no studies to date have examined whether the experience of episodic stressful life events is associated with changes in autobiographical memory specificity. Second, all previous studies examining the impact of stress on OGM have done so retrospectively, and were not able to establish the temporal precedence necessary to test whether the experience of stress is associated with prospective increases in OGM. Although one study did measure OGM at multiple time points immediately following a traumatic injury (Hitchcock et al. 2014a), OGM was not measured prior to the trauma so the study was not able to account for the impact of trauma on changes in OGM. Therefore, although the results of this earlier research are consistent with theories that the experience of stress may lead to the development of OGM (Williams 1996; Williams et al. 2007b), this is the first study to truly test this theory utilizing a longitudinal design.

A key finding from this study was that stressful life events predicted decreases in autobiographical memory specificity to positive, but not negative, cues. Although this appears to be in contrast with a recent meta-analysis showing that adults with a history of trauma show greater OGM to negative cues (Ono et al. 2015), childhood trauma has been shown to be associated with elevated levels of OGM to both positive and negative cues in youth (Brennen et al. 2010; Stokes et al. 2004; Valentino et al. 2009). It is also important to consider the timing of OGM development in youth. For example, younger children may have greater OGM to negative than positive cues (Drummond et al. 2006), suggesting that OGM to negative cues develops first. Furthermore, within the same sample as the current study, previous findings showed that at the initial assessment, youth at high risk for depression due to maternal history of MDD already demonstrated significantly greater OGM to negative cues than youth of never-depressed mothers (Woody et al. 2015). Therefore, it may be that, in the current sample, the development of OGM to negative cues may have already occurred, and we may only be able to observe the development of OGM to positive cues within the age range of the current study. Similarly, the developmental timing of OGM may explain why we did not observe any moderation by

maternal depression in the current study. Given that children of depressed mothers already demonstrated greater OGM to negative cues at the initial assessment (Woody et al. 2015), our sample may not have included a large enough age range to detect whether maternal history of depression moderates the impact of life stress on changes in autobiographical memory specificity.

It is important to note that, although life stress did predict decreases in autobiographical memory specificity, the effect was small ( $r_{effect\ size} = .13$ ). Although a number of factors may have contributed to this relatively small effect size, one potential influence is the types of stressors examined within the current study. The LSI allows for the examination of stressful life events ranging in severity from mild to severe, and therefore life events assessed in the current study included both normative and traumatic life events. As the functional avoidance hypothesis focuses on the role of *traumatic* life events in the development of OGM (Sumner 2012; Williams et al. 2007b), it stands to reason that more normative stressful life events may have a less pronounced impact on changes in autobiographical memory specificity. Furthermore, in addition to the experience of negative life events, theorists have proposed that rumination and deficits in executive control may also contribute to the development of OGM (Sumner 2012; Williams et al. 2007b). Future prospective studies should seek to include an examination of these other influences. It will also be important for theory on the development of OGM to expand to include the potential role of normative life stressors, and to highlight whether these stressors are hypothesized to contribute to the development of OGM through the same mechanisms as traumatic life events. This may also help to clarify the relation of OGM to risk for depression versus PTSD, in that normative life events may be relevant for OGM models of depression whereas traumatic events may be most relevant for OGM models of PTSD.

Another important point to acknowledge is that, although it is assumed that increased OGM to positive cues is associated with increased risk for depression, it is possible that overgeneral memory for positive cues may actually serve as a protective factor in these youth. Indeed, according to the functional avoidance hypothesis, the overgeneralization of autobiographical memories immediately following a traumatic life event can serve as a short-term protective factor and alleviate distress, as individuals avoid memories which elicit negative emotional reactions (e.g., Williams et al. 2007b). There is support for this hypothesis, as one study found that elevated levels of OGM following a traumatic life event was protective against symptoms of PTSD 6 months after trauma exposure in children (Hitchcock et al. 2014a). Therefore, to establish whether a decrease in autobiographical memory specificity to positive cues following the experience of life stress does increase risk for depression in these youth, it is important that future studies examine whether these changes

in OGM are also associated with increases in depressive symptoms.

This study exhibited several strengths including the use of a multi-wave prospective design to examine changes in youth's autobiographical memory specificity. However, there were also some limitations which should be addressed in future research. First, given considerations of OGM developmental timing (Drummond et al. 2006) and evidence that increased OGM to negative cues may already have been observable in our sample at the initial assessment, (Woody et al. 2015), future studies with younger samples may be required to examine the development of OGM to negative cues. Second, although the multi-wave design of the study was a major strength, it is also important to note that the follow-up assessments were spaced 6 months apart. Given that the only other study to examine OGM at multiple time points following a stressor assessed OGM every 2 months (Hitchcock et al. 2014a), it may be important to assess OGM more frequently to gain a more nuanced picture of the relation between stress and prospective changes in OGM for youth. Third, our sample was recruited based on the presence versus absence of a history of MDD in children's mothers. Although the potential influence of maternal MDD history was statistically controlled for in all analyses and we showed that maternal MDD history did not moderate any of the findings, future research should seek to include more representative samples. Finally, the current study does not address the precise mechanisms through which elevated levels of life stress lead to the development of OGM in youth. Theorists have proposed that the experience of life stress may lead to greater OGM through the avoidance of engaging in painful memories (Williams 1996) and future studies are needed to test whether cognitive avoidance mediates the relation between episodic life stress and the development of OGM in youth.

In conclusion, the current study contributes to the current body of literature on the impact of stress on OGM development, and is the first to prospectively examine whether stressful life events predict decreases in autobiographical memory specificity in youth. Additionally, the current study suggests that, for pre-adolescent and early-adolescent youth, the experience of episodic life stress predicts prospective decreases in memory specificity for positive, but not negative, cues. If replicated, the current findings highlight a specific target for intervention (i.e., elevated levels of negative life events) to reduce risk for the development of overgeneral autobiographical memory in youth. Alternatively, interventions can also target OGM directly to increase autobiographical memory specificity in at-risk youth. Indeed, there is evidence that maternal elaboration during mother-child reminiscing about past events is associated with greater autobiographical memory specificity in children (e.g., Reese and Newcombe 2007; Valentino et al. 2014) and that training mothers in elaborative reminiscing can be an effective intervention for improving offspring

autobiographical memory specificity (for a review, see Corsano and Guidotti 2017). Furthermore, Memory Specificity Training (MEST) has shown to effectively increase autobiographical memory specificity (Raes et al. 2009), and is an effective intervention for youth (Neshat-Doost et al. 2013). Therefore, both elaborative reminiscing training and MEST may help to prevent the development of OGM, which in turn may reduce risk for the development of psychopathology in youth. The current results suggest that these interventions may be particularly important for youth exposed to high levels of negative life stress.

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## Compliance with ethical standards

**Conflict of Interest** The authors declare that they have no conflict of interest.

**Ethical Approval** All study procedures were approved by Binghamton University's Institutional Review Board.

**Informed Consent** Informed consent and assent were obtained from all mothers and children, respectively.

## References

- Adrian, C., & Hammen, C. (1993). Stress exposure and stress generation in children of depressed mothers. *Journal of Consulting and Clinical Psychology, 61*(2), 354–359.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington: Author.
- Anderson, R. J., Goddard, L., & Powell, J. H. (2010). Reduced specificity of autobiographical memory as a moderator of the relationship between daily hassles and depression. *Cognition and Emotion, 24*(4), 702–709. <https://doi.org/10.1080/02699930802598029>.
- Beck, A. T. (1987). Cognitive models of depression. *Journal of Cognitive Psychotherapy, 1*(1), 5–37.
- Brennen, T., Hasanović, M., Zotović, M., Blix, I., Skar, A. M. S., Prelič, N. K., & Gavrilov-Jerković, V. (2010). Trauma exposure in childhood impairs the ability to recall specific autobiographical memories in late adolescence. *Journal of Traumatic Stress, 23*(2), 240–247. <https://doi.org/10.1002/jts.20513>.
- Brown, G. W., & Harris, T. (1978). *Social origins of depression*. London: Free Press.
- Burkhouse, K. L., Siegle, G. J., Woody, M. L., Kudinova, A. Y., & Gibb, B. E. (2015). Pupillary reactivity to sad stimuli as a biomarker of depression risk: Evidence from a prospective study of children. *Journal of Abnormal Psychology, 124*(3), 498–506. <https://doi.org/10.1037/abn0000072>.
- Champagne, K., Burkhouse, K. L., Woody, M. L., Feurer, C., Sosoo, E., & Gibb, B. E. (2016). Overgeneral autobiographical memory in adolescent major depressive disorder: State- or trait-like marker of risk? *Journal of Adolescence, 52*, 72–75.
- Clark, D. A., Beck, A. T., & Alford, B. A. (1999). *Scientific foundations of cognitive theory and therapy of depression*. New York: Wiley.
- Corsano, P., & Guidotti, L. (2017). Parents' reminiscing training in typically developing and "at-risk" children: A review. *Early Child Development and Care, 1–14*. <https://doi.org/10.1080/03004430.2017.1289518>.
- Drummond, L., Dritschel, B., Astell, A., O'Carroll, R. E., & Dalgleish, T. (2006). Effects of age, dysphoria, and emotion-focusing on autobiographical memory specificity in children. *Cognition and Emotion, 20*(October), 488–505. <https://doi.org/10.1080/02699930500341342>.
- Feurer, C., Hammen, C. L., & Gibb, B. E. (2016). Chronic and episodic stress in children of depressed mothers. *Journal of Clinical Child and Adolescent Psychology, 45*(3), 270–278. <https://doi.org/10.1080/15374416.2014.963859>.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured clinical interview for DSM-IV Axis I disorders-patient edition (SCID-I/P)*. New York: Biometrics Research Department, NY State Psychiatric Institute.
- Garber, J., & Flynn, C. (2001). Predictors of depressive cognitions in young adolescents. *Cognitive Therapy and Research, 25*(4), 353–376. <https://doi.org/10.1023/A:1005530402239>.
- Garber, J., & Robinson, N. S. (1997). Cognitive vulnerability in children at risk for depression. *Cognition and Emotion, 11*(5–6), 619–635. <https://doi.org/10.1080/026999397379881b>.
- Gibbs, B. R., & Rude, S. S. (2004). Overgeneral autobiographical memory as depression vulnerability. *Cognitive Therapy and Research, 28*(1), 1–12. <https://doi.org/10.1023/B:COTR.0000045561.72997.7c>.
- Griffith, J. W., Sumner, J. A., Debeer, E., Raes, F., Hermans, D., Mineka, S., et al. (2009). An item response theory/confirmatory factor analysis of the autobiographical memory test. *Memory, 17*(6), 609–723. <https://doi.org/10.1080/09658210902939348>.
- Griffith, J. W., Kleim, B., Sumner, J. A., & Ehlers, A. (2012). The factor structure of the autobiographical memory test in recent trauma survivors. *Psychological Assessment, 24*(3), 640–646. <https://doi.org/10.1037/a0026510>.
- Gutenbrunner, C., Salmon, K., & Jose, P. E. (2017). Do Overgeneral autobiographical memories predict increased psychopathological symptoms in community youth? A 3-year longitudinal investigation. *Journal of Abnormal Child Psychology, pp. 1–12*. <https://doi.org/10.1007/s10802-017-0278-5>.
- Hermans, D., Vandromme, H., Debeer, E., Raes, F., Demyttenaere, K., Brunfaut, E., & Williams, J. M. G. (2008). Overgeneral autobiographical memory predicts diagnostic status in depression. *Behaviour Research and Therapy, 46*(5), 668–677. <https://doi.org/10.1016/j.brat.2008.01.018>.
- Heron, J., Crane, C., Gunnell, D., Lewis, G., Evans, J., & Williams, J. M. G. (2012). 40,000 memories in young teenagers: Psychometric properties of the autobiographical memory test in a UK cohort study. *Memory, 20*(3), 300–320. <https://doi.org/10.1080/09658211.2012.656846>.
- Hipwell, A. E., Sapotichne, B., Klostermann, S., Battista, D., & Keenan, K. (2011). Autobiographical memory as a predictor of depression vulnerability in girls. *Journal of Clinical Child and Adolescent Psychology, 40*(2), 254–265. <https://doi.org/10.1080/15374416.2011.546037>.
- Hitchcock, C., Nixon, R. D. V., & Weber, N. (2014a). A longitudinal examination of overgeneral memory and psychopathology in children following recent trauma exposure. *Applied Cognitive Psychology, 28*(4), 531–538. <https://doi.org/10.1002/acp.3027>.



- Hitchcock, C., Nixon, R. D. V., & Weber, N. (2014b). A review of overgeneral memory in child psychopathology. *The British Journal of Clinical Psychology*. <https://doi.org/10.1111/bjc.12034>.
- Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, 6(1), 1–55. <https://doi.org/10.1080/10705519909540118>.
- Jaenicke, C., Hammen, C. L., Zupan, B., Hiroto, D., Gordon, D., Adrian, C., & Burge, D. (1987). Cognitive vulnerability in children at risk for depression. *Journal of Abnormal Child Psychology*, 15(4), 559–572. <https://doi.org/10.1007/BF00917241>.
- Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., et al. (1997). Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36(7), 980–988. <https://doi.org/10.1097/00004583-199707000-00021>.
- Moore, S. A., & Zoellner, L. A. (2007). Overgeneral autobiographical memory and traumatic events: An evaluative review. *Psychological Bulletin*, 133(3), 419–437. <https://doi.org/10.1037/0033-2909.133.3.419>.
- Neshat-Doost, H. T., Dalgleish, T., Yule, W., Kalantari, M., Ahmadi, S. J., Dyregrov, A., & Jobson, L. (2013). Enhancing autobiographical memory specificity through cognitive training an intervention for depression translated from basic science. *Clinical Psychological Science: A Journal of the Association for Psychological Science*, 1(1), 84–92. <https://doi.org/10.1177/2167702612454613>.
- Ono, M., Devilly, G. J., & Shum, D. H. K. (2015). A meta-analytic review of overgeneral memory: The role of trauma history, mood, and the presence of posttraumatic stress disorder. *Psychological Trauma: Theory, Research, Practice, and Policy*, 8(2), 157–164. <https://doi.org/10.1037/tra0000027>.
- Park, R. J., Goodyer, I. M., & Teasdale, J. D. (2002). Categorical overgeneral autobiographical memory in adolescents with major depressive disorder. *Psychological Medicine*, 32, 267–276. <https://doi.org/10.1017/S0033291701005189>.
- Raes, F., Williams, J. M. G., & Hermans, D. (2009). Reducing cognitive vulnerability to depression: A preliminary investigation of MEMory specificity training (MEST) in inpatients with depressive symptomatology. *Journal of Behavior Therapy and Experimental Psychiatry*, 40(1), 24–38. <https://doi.org/10.1016/j.jbtep.2008.03.001>.
- Raudenbush, S. W., & Bryk, A. S. (2002). *Hierarchical linear models: Applications and data analysis methods* (2nd ed.). Thousand Oaks: Sage.
- Raudenbush, S. W., Bryk, A. S., Cheong, Y. F., & Congdon, R. T. (2004). *HLM 6: Hierarchical linear and nonlinear modeling*. Lincolnwood: Scientific Software International.
- Rawal, A., & Rice, F. (2012). Examining overgeneral autobiographical memory as a risk factor for adolescent depression. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(5), 518–527. <https://doi.org/10.1016/j.jaac.2012.02.025>.
- Reese, E., & Newcombe, R. (2007). Training mothers in elaborative reminiscing enhances children's autobiographical memory and narrative. *Child Development*, 78(4), 1153–1170. <https://doi.org/10.1111/j.1467-8624.2007.01058.x>.
- Schafer, J. L., & Graham, J. W. (2002). Missing data: Our view of the state of the art. *Psychological Methods*, 7(2), 147–177. <https://doi.org/10.1037/1082-989X.7.2.147>.
- Schönfeld, S., & Ehlers, A. (2006). Overgeneral memory extends to pictorial retrieval cues and correlates with cognitive features in post-traumatic stress disorder. *Emotion*, 6(4), 611–621. <https://doi.org/10.1037/1528-3542.6.4.611>.
- Schönfeld, S., Ehlers, A., Böllinghaus, I., & Rief, W. (2007). Overgeneral memory and suppression of trauma memories in post-traumatic stress disorder. *Memory*, 15(3), 339–352. <https://doi.org/10.1080/09658210701256571>.
- Stokes, D. J., Dritschel, B. H., & Bekerian, D. A. (2004). The effect of burn injury on adolescents autobiographical memory. *Behaviour Research and Therapy*, 42(11), 1357–1365. <https://doi.org/10.1016/j.brat.2003.10.003>.
- Sumner, J. A. (2012). The mechanisms underlying overgeneral autobiographical memory: An evaluative review of evidence for the CaR-FA-X model. *Clinical Psychology Review*. <https://doi.org/10.1016/j.cpr.2011.10.003>.
- Sumner, J. A., Griffith, J. W., & Mineka, S. (2010). Overgeneral autobiographical memory as a predictor of the course of depression: A meta-analysis. *Behaviour Research and Therapy*, 48(7), 614–625. <https://doi.org/10.1016/j.brat.2010.03.013>.
- Sumner, J. A., Griffith, J. W., Mineka, S., Rekart, K. N., Zinbarg, R. E., & Craske, M. G. (2011). Overgeneral autobiographical memory and chronic interpersonal stress as predictors of the course of depression in adolescents. *Cognition and Emotion*, 25(1), 183–192. <https://doi.org/10.1080/02699931003741566>.
- Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). New York: Pearson. <https://doi.org/10.1037/022267>.
- Valentino, K., Toth, S. L., & Cicchetti, D. (2009). Autobiographical memory functioning among abused, neglected, and nonmaltreated children: The overgeneral memory effect. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 50(8), 1029–1038. <https://doi.org/10.1111/j.1469-7610.2009.02072.x>.
- Valentino, K., Nuttall, A. K., Comas, M., McDonnell, C. G., Piper, B., Thomas, T. E., & Fanuele, S. (2014). Mother-child reminiscing and autobiographical memory specificity among preschool-age children. *Developmental Psychology*, 50(4), 1197–1207. <https://doi.org/10.1037/a0034912>.
- Van Vreeswijk, 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*. [https://doi.org/10.1016/S0005-7967\(03\)00194-3](https://doi.org/10.1016/S0005-7967(03)00194-3).
- Vrielynck, N., Deplus, S., & Philippot, P. (2007). Overgeneral autobiographical memory and depressive disorder in children. *Journal of Clinical Child and Adolescent Psychology*, 36(1), 95–105. <https://doi.org/10.1080/15374410709336572>.
- Williams, J. M. G. (1996). Depression and the specificity of autobiographical memory. In D. C. Rubing (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 244–267). Cambridge: Cambridge University Press.
- Williams, J. M. G., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology*, 95(2), 144–149. <https://doi.org/10.1037/0021-843X.95.2.144>.
- Williams, H. L., Conway, M. A., & Cohen, G. (2007a). *Autobiographical Memory: (M. A. Conway & G. Cohen, Eds.) Memory in the Real World* (3rd ed.). New York: Psychology Press. <https://doi.org/10.1017/CBO9780511558313>.
- Williams, J. M. G., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., & Dalgleish, T. (2007b). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin*, 133(1), 122–148. <https://doi.org/10.1037/0033-2909.133.1.122>.
- Woody, M. L., Burkhouse, K. L., & Gibb, B. E. (2015). Overgeneral autobiographical memory in children of depressed mothers. *Cognition and Emotion*. <https://doi.org/10.1080/02699931.2014.891972>.