Overgeneral Memory, Trauma, and Psychopathology in Children

Caitlin Hitchcock,

BPsyc (Hons.)

School of Psychology

Social and Behavioural Sciences

Flinders University

Adelaide, Australia

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Summary

Overgeneral memory (OGM) occurs when an individual demonstrates impaired retrieval of specific, single incident events from autobiographical memory. OGM theorists have argued that childhood trauma will increase OGM as this retrieval style will be adaptive for emotion regulation in the short term following trauma. However, OGM is thought to become associated with psychopathology if maintained over the longer term (Williams, 1996). This proposition has not been directly tested, although OGM is consistently associated with persistent depression in adolescents (see Hitchcock, Nixon, & Weber, 2013, for recent review of childhood OGM). Further, it is unclear how OGM relates to paediatric posttraumatic stress disorder (PTSD). Better understanding of whether the relationship between OGM and psychopathology may change over time will indicate at what point certain clinical interventions may be optimally timed following childhood trauma (especially those that target memory retrieval processes). This thesis comprised the first examination of OGM and the course of psychological symptoms in young people exposed to trauma. This investigation was driven by two research questions. First, how does OGM relate to psychopathology following childhood trauma exposure? In particular, I explored how OGM related to symptoms of PTSD and depression, and whether time since trauma moderated these relationships. Second, does the CaR-FA-X model (Williams et al., 2007) adequately explain childhood OGM? Specifically, key components of the model, namely the capture and rumination, functional avoidance, and executive control mechanisms, were assessed.

Study 1 longitudinally examined OGM and the development of psychological symptoms over six months following childhood trauma exposure. The capture and rumination, and executive control mechanisms of the CaR-FA-X model were also assessed. Results indicated that the relationship between OGM and PTSD symptoms

changed over time. Interestingly, OGM was negatively related to PTSD symptoms at six months post-trauma. No evidence was found for a relationship between OGM and depression, or for the assessed CaR-FA-X mechanisms to explain OGM. Study 2 built upon these findings by providing preliminary, cross-sectional evidence that the relationship between OGM and PTSD symptoms may continue to change years after trauma exposure. All three mechanisms of the CaR-FA-X model were assessed to thoroughly test the theory. No evidence was found for the CaR-FA-X model as a whole, although some results were consistent with the proposed role of functional avoidance. Based on these results, Study 3 and Study 4 experimentally assessed the key assumptions of functional avoidance. Results indicated that overgeneral retrieval of memories of an adverse event regulated affect. This provided further support for the hypothesised role of the functional avoidance mechanism.

Three main conclusions were drawn from results. First, findings indicated that OGM may initially be adaptive for mood regulation, and also PTSD symptoms, following childhood trauma. At this initial stage, functional avoidance appears to explain OGM. Second, OGM may change to become maladaptive if used in the longer term. This change may occur years after exposure, and at this point OGM may become associated with depression. Once maladaptive, the capture and rumination, and executive control mechanisms of the CaR-FA-X model may become operational. Finally, the mechanism underlying OGM's change from adaptive to maladaptive is unclear. Theory and previous research suggest this may occur due to OGM generalising from a trauma memory to retrieval of other autobiographical memories. Further research on the trajectory of OGM following childhood trauma, and the cognitive processes underlying the retrieval style will improve the efficacy of emerging clinical interventions. Ultimately, further understanding of OGM may help to reduce the impact and severity of psychopathology following childhood trauma.

Declaration

I certify that this thesis does not incorporate without acknowledgement any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

Caitlin Hitchcock, BPsyc (Hons.)

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CHAPTER 1

Introduction[1]

Twenty five years of research has implicated overgeneral memory (OGM), or reduced autobiographical specificity, in the development and maintenance of depression, and emphasised OGM as a potential intervention point for treatment. OGM occurs when an individual demonstrates impaired retrieval of specific, single incident events (e.g., my football game last Saturday) from autobiographical memory, instead retrieving categories of events (e.g., when I play football) or events that last for an extended period of time (e.g., I played well last football season). In adult populations, OGM has been consistently related to poorer prognosis for depression and posttraumatic stress (for review see Moore & Zoellner, 2007; Williams et al., 2007). Focus has therefore shifted to developing theoretical understanding of OGM (for review of research in adult samples see Sumner, 2012). The dominant model of OGM in adult clinical samples (that of Williams, 1996; and Williams et al., 2007) originally proposed that OGM may develop in childhood, with adverse life experiences being one of several factors that accounted for increased OGM in psychopathology (Williams & Broadbent, 1986). Recently, extensions and variations of this basic proposition have been proposed (e.g., Dalgleish et al., 2007; Valentino, 2011), however, despite the model being supported in adult populations (Sumner, 2012), the assumption of childhood onset has not been thoroughly assessed. Surprisingly, research has only recently begun to examine the phenomenon in children, despite evidence that children also experience difficulty retrieving specific memories following the experience of trauma (e.g., de Decker, Hermans, Raes, & Eelen, 2003; Stokes, Dritschel, & Bekerian, 2004).

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[1] A large portion of this chapter has been published in Hitchcock, C., Nixon, R.D.V., & Weber, N. (2013). A review of overgeneral memory in child psychopathology. *British Journal of Clinical Psychology.* doi: 10.1111/bjc.12034

This thesis aims to advance theoretical understanding by examining the trajectory of OGM following childhood trauma. In doing so, I explore the cognitive underpinnings of the retrieval style, and also aim to expand clinical understanding by determining how OGM relates to paediatric psychopathology. This literature review will outline the current theoretical understanding of OGM, and existing evidence on how OGM relates to childhood trauma and subsequent psychopathology. Previous reviews of OGM have been informative however they have either consisted almost exclusively of data from adult samples (e.g., Sumner, 2012), have not given an indication of the size of the effect in children and youth (e.g., Valentino, 2011), or have not thoroughly examined whether Williams et al.'s (2007) model actually explains OGM in young people. A reasonable body of research in the child field now exists that allows several key questions to be examined. First, does OGM form in childhood? Second, how does OGM relate to child psychopathology? Third, how well do models account for OGM in children? The need to study OGM in children from a developmental framework has been emphasised (see Valentino, 2011). Valentino (2011) outlined numerous factors that may be pertinent to OGM in children that could be added to current models. I agree that childhood processes and disorders should not be assumed to be simpler versions of adult processes. I also believe that parsimonious explanations serve the field best, and a detailed examination of how well current models explain OGM in childhood psychopathology, including a methodological analysis of the discrepant findings, is required, and this constitutes a third aim of the review. I do not aim to evaluate support for each proposed model of OGM; rather, I seek to assess the proposition that an overgeneral style will begin in childhood and evaluate the evidence for the mechanisms proposed by Williams et al. (2007). Accordingly I (a) outline Williams et al.'s (2007) model of OGM, (b) demonstrate there is evidence that OGM occurs in children, although this is largely in the context of depression and trauma, and

(c) highlight that the mechanisms require further empirical confirmation. Finally, I outline how this thesis aims to close the identified gaps in the literature.

Theories of OGM

Conway and Pleydell-Pearce's (2000) model of autobiographical memory has formed the basis of dominant OGM theories (e.g., Williams et al, 2007). The model proposes autobiographical information is arranged into a series of knowledge structures. These structures hierarchically link three nested indices of memory specificity. At the broadest level are lifetime events, which are broad periods of an individual's life (e.g., when I was at university). More detailed are general events, which represent repeated events (e.g., attending neuropsychology lectures). Finally, event specific knowledge represents the specific temporal details of an event (e.g., learning about traumatic brain injury in my lecture last Thursday). [2] In generative retrieval, a cue is used to guide a controlled, top-down search of the memory structure. It is argued that OGM results from an interruption to this process (Dalgleish, Rolfe, Golden, Dunn, & Barnard, 2008; Williams et al., 2007), whereby the search does not reach the event specific level. Importantly, one does not either have OGM, or not have OGM. That is, there is no clinical cut-off for OGM. Rather, those who are described as overgeneral simply retrieve overgeneral memories more often than others.

Initial theory of OGM in clinical disorders (Williams, 1996) stated generative retrieval may be impaired if a child was exposed to trauma, due to the child avoiding the event specific index to avoid negative affect generated by such information. This was termed functional avoidance. As such, adverse childhood experiences are thought to

[2] Developmental literature debates whether children first develop the ability to form general memories, and progress to distinguishing specific events, or whether the development of specific memories precedes the ability to group events into general memories (e.g., Howe et al., 2006; Valentino, 2011). The reporting of autobiographical memories in a general or specific manner may therefore be representative of the child's developmental stage. However, the ability to recall both specific and general memories is fully developed by the age of seven years (e.g., Gathercole, 1998; Nelson & Gruendel, 1981). As child participants in OGM research are consistently aged seven or above, this thesis does not contribute to this debate.

increase OGM through initiating or increasing use of functional avoidance. This explanation was later expanded in the CaR-FA-X model (Williams et al., 2007). Williams et al.'s (2007) expanded model does not reduce the centrality of functional avoidance. Rather, two alternate mechanisms were added to account for OGM in those without a childhood trauma history.

CaR-FA-X Model

Capture and rumination (CaR). The cue used to guide generative retrieval is often a self-representation (e.g., self-beliefs; Williams et al., 2007). Abstract information related to a self-representation may capture attention during the search, particularly for individuals with strong negative self-beliefs, as in depression. The self-belief will then be ruminated on, stopping the search from progressing to event specific information. For example, when presented with the cue word *hopeless*, activation of the self-belief 'I'm hopeless, I never do well at anything' will lead to rumination on information that supports this self-belief, stopping the individual from retrieving a specific memory of a time when they were hopeless. Conceptual or generalised information is then volunteered as the retrieved memory. This capture of attention is more likely to occur when the individual is prone to rumination.

Functional avoidance (FA). The model proposed that individuals would abandon a search when specific details of a negative event were recalled to reduce negative affect. This process becomes negatively reinforced by avoidance of emotional pain. Importantly, the generic retrieval style will not only be used when a negative cue is activated, rather the retrieval style will generalise to all memory searches as specific memories of any kind may lead to other negative information. Hence, overgeneral memories will be produced for all cued recall, regardless of cue valence.

Executive control (X). The model stated that emotional disturbance would impair the control processes governing a search. These control processes include

inhibiting irrelevant information to avoid capture errors and holding the retrieval model and retrieved information in working memory. Reduced executive capacity may increase OGM by impairing these processes.

There are a number of definitions of these executive control processes. Williams et al. (2007) do not prescribe to any one description of working memory, or inhibition. Considering working memory, the model states that specific retrieval will rely on the updating on working memory, and the maintenance of a retrieval model. Storage and processing roles of working memory is consistent with a dual component explanation of working memory. A dual component model outlines a primary component of working memory responsible for directing attention and processing information, and a secondary component used purely for short-term storage (Unsworth & Engle, 2007). This thesis therefore uses assessments of working memory which measure both processing and storage aspects. In terms of inhibition, Friedman and Miyake (2004) outline two subtypes; resistance to proactive interference and response-distractor inhibition. The response-distractor subtype is defined as the ability to actively maintain a goal in the face of the interference of prepotent responses or distracting stimuli. This type of inhibition is closely related to effortful control, task switching and everyday cognitive failures. Thus, inhibiting irrelevant information during memory retrieval is consistent with response-distractor inhibition. This thesis therefore considered response-distractor inhibition when assessing the inhibition aspect of the CaR-FA-X model.

Summary. Functional avoidance is thought to be an important mechanism through which OGM occurs. As functional avoidance is proposed to increase as a result of childhood trauma, it is important to determine that children do experience OGM, along with examining if functional avoidance, and the other mechanisms, are able to account for childhood OGM. I now review OGM literature in child and adolescent samples to address three key questions; (1) Do children demonstrate OGM? (2) How

does the experience of OGM relate to childhood trauma and associated psychopathology? And (3) Is the CaR-FA-X model able to explain OGM in child psychopathology once developmental issues have been considered?

Method

Articles were identified in PsycINFO and PubMed searches completed from inception until July 2013 using the terms *overgeneral memory AND children*, *autobiographical memory specificity AND children*, and *autobiographical memory AND children.* The database searches identified a total of 442 articles. I examined the title of each, and then the abstract of 35 articles to determine inclusion. The reference lists of identified papers were also examined to source further articles. Inclusion criteria were that the article was in English, published in a peer-reviewed journal and examined either OGM or reduced memory specificity in those under the age of eighteen. Thesis dissertations and psychometric evaluations of measures were excluded. Twenty four articles were identified to examine OGM or reduced specificity. Two were excluded as they were not in English, and one was excluded as it was a psychometric evaluation of a measure. One paper (Brennen et al., 2010) included participants above the age criterion, however it was included as it examined OGM in adolescents who had experienced trauma as a child. Hence, a total of 21 articles were reviewed (see Table 1).

To make results comparable between cross-sectional studies, I calculated Cohen's *d* for the difference between groups using pooled standard deviations (Cohen, 1988). I also converted *r* statistics into Cohen's *d*. To provide a more informative indication of each study's estimate of the true effect size, I focus on 95% confidence intervals around Cohen's *d*. In this review, I separated studies based on trauma and depression samples. This was dictated by the selection of sample in each article. Given the interrelation between trauma and depression, and the difficulty in separating their effects (Williams et al., 2007), it is worth noting that the model appears not to

distinguish between the two in the development of OGM, despite studies attempting to separate the sample types for methodological reasons.

Do Children Demonstrate OGM?

Childhood trauma. Ten articles examined the relationship between OGM and trauma exposure in children or adolescents. Eight studies supported the relationship. Some studies examined OGM in trauma-exposed samples but did not report the effect of trauma exposure on OGM, instead focussing on effects attributable to depression symptoms (e.g., Orbach, Lamb, Sternberg, Williams, & Dawud-Noursi, 2001). As such, those studies were not included in this section of the review. Trauma was operationalized as a Criterion A event required for diagnosis of posttraumatic stress disorder (PTSD) in the Diagnostic and Statistical Manual of Mental Disorders (DSM-V; American Psychiatric Association, 2013). Identified events varied between single incident and chronic traumas, such as abuse. Findings were consistent between event types. Interestingly, articles predominately focussed on the effect of trauma exposure on OGM, rather than posttraumatic stress. Importantly, all studies accounted for depression symptoms in analyses.

Table 1

Summaries of Reviewed Studies

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Meesters et al. (2000)	14-19 year olds 10 (7) with trauma history 17 (12) psychiatric control Cross-sectional, unmatched control	Mixed	Author compiled questionnaire	None	Semantic AMT (SAMT)	Total SAMT score (total correct/ total - irrelevant items)	Trauma < control No correlation depression symptoms and SAMT	*d* = 0.79* *r* = -.19, *d* = 0.39	[0.03, 1.74] [-0.53, 0.21] [-0.43, 1.25]
Orbach et al. (2001)	8-12 year olds 34 family violence [a] 16 control [a] Cross-sectional, unmatched control	Family violence	CDI	None	Coding of responses on Family Disagreements Questionnaire	Number of overgeneral memories (categoric)	Correlation between depression and OGM	*r* = .32*, *d* = .68*	[0.05, 0.55] [0.01, 1.32]
Swales et al. (2001)	Mean age 14 years 26 (13) mixed diagnoses 24 (14) control Cross-sectional, unmatched control	Not measured	BDI	None	AMT	Number of specific memories	Correlation between specificity and depression Control > Clinical	*r* = .40*, *d* = .87* *d* = 0.99*	[0.01, 0.69] [0.02, 1.91] [0.38, 1.68]

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Park et al. (2002)	12-17 year olds 49 (34) MDD 26 (16) clinical control 33 (21) community control Cross-sectional, unmatched control	Not measured	HDRS	None	AMT	Number of overgeneral memories (categoric)	MDD > community control for pos cues and neg cues Correlation HDRS and OGM to neg cues	d = 0.91** d = 0.88** r = .27*, d = 0.56*	[0.44, 1.42] [0.42, 1.39] [0.07, 0.45] [0.14, 1.01]
de Decker et al. (2003)	14-20 year olds 27 (10) inpatients without formal diagnoses Correlational	Mixed	BDI	Dutch IES	Dutch AMT	Number of specific memories	Trauma history correlated with specificity No correlation for BDI and specificity	r = - .60*, d = 1.50* r = .07, d = 0.14	[-0.23,- 0.82] [0.47, 2.87] [-0.32, 0.44] [-0.67, 0.98]
Stokes et al. (2004)	11-16 year olds 12 (12) burns victims 12 (12) orthodontic control Cross-sectional, unmatched control	Burns	Birleson depression scale (Birleson, 1981)	IES	Cued recall task	Number of overgeneral memories (extended)	Burns > Control	d =1.19 *	[0.27 , 2.32]

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Park et al. (2004)	12-17 year olds 44 (31) MDD 31 (23) remitted MDD 26 (16) clinical control 33 (21) community control Cross-sectional, unmatched control	Not measured	Diagnosis of MDD	None	AMT	Proportion of overgeneral memories	MDD > community control	$d = 0.89$[b]	[0.41, 1.42]
Johnson et al. (2005)	12-18 year olds 134 (72) violence exposed Correlational	Family violence	CESDS	None	Specific memories to cue words	Number of overgeneral memories	Depression predicted OGM	$\beta = 0.22$*	-
Drummond et al. (2006)	7-11 year olds 14 dysphoric [a] 56 nondysphoric [a] Cross-sectional, unmatched control	No experience of trauma	CDI	None	AMT	Number of specific memories	Dysphoric < nondysphoric	$d = 0.45$*	[0.03, 0.96]

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Kuyken et al. (2006)	12-18 year olds 28 (21) healthy control 12 (9) MDD-no trauma 22 (20) MDD-trauma Cross-sectional, unmatched control	Mixed	Diagnosis of MDD	Children's IES	AMT	Number of overgeneral memories	MDD-trauma = control MDD-trauma < MDD- no trauma MDD-no trauma > control Probable PTSD < non PTSD	*d* = 0.45 *d* = 0.62* *d* = 1.26* *d* = 1.27*	[0.20, 1.15] [0.09, 1.41] [0.97, 3.25] -
Vrielynck et al. (2007)	9-13 year olds 15 (7) depressed 25 (6) clinical control 20 (7) community control Cross-sectional, unmatched control	Mixed	Lifetime diagnosis of depressive disorder other than MDD	Number of post traumatic reactions	AMT	Number of overgeneral memories (generic)	Depressed = clinical control Clinical control > community control Depressed > community control Trauma exposure and OGM correlated	*d* = 0.60 *d* = 0.88* *d* = 1.63* *r* = .31*, *d* = 0.65*	[0.05, 1.32] [0.25, 1.59] [0.82, 2.64] [0.06, 0.52] [0.12, 1.22]

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Valentino et al. (2009)	7-13 year olds 36 (12) abused 34 (15) neglected 115 (61) community control Cross-sectional, unmatched control	Abuse or neglect	CDI	None	AMT	Number of overgeneral memories	Abused > neglected Abused > control Depression predicted OGM Abuse predicted OGM	d = 0.64* d = 0.51* β = .22* β = .26*	[0.15, 1.17] [0.18, 0.85] - -
Brennen et al. (2010)	17-19 year olds 40 (20) trauma-exposed 49 (22) control Cross-sectional, culturally different control	War exposure	BDI-II	IES-R	Norwegian and Bosnian AMT	Proportion of overgeneral memories (categoric)	Trauma-exposed > control No correlation OGM and depression symptoms	d = 1.76** not reported	[1.23, 2.36] -
Study 1			BDI-II						
Study 2	18-22 year olds 50 (41) high exposure 90 (80) low exposure Cross-sectional	Exposed to bombing as 9-13 year olds		IES-R	AMT	Proportion of overgeneral memories (categoric)	High exposure > low exposure Depression not correlated with OGM	d = 0.64** not reported	[0.30, 1.00] -

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Raes et al. (2010)	9-13 year olds 135 (72) primary school students Correlational	Not measured	CDI	None	AMT (written format)	Number of overgeneral memories (categoric)	Overgeneral memories correlated with CDI score	*r* = .22*, *d* = 0.45*	[0.05, 0.37] [0.10, 0.80]
Kuyken & Dalgleish (2011) Study 1	14-18 year olds 179 (113) healthy community sample Cross-sectional High risk cohort	Not measured	BDI-II	None	AMT (written format)	Proportion of overgeneral memories (categoric)	BDI score predicted proportion of overgeneral memories	β = .14* pos cues β = .28** neg cues	- -
Study 2	14-18 year olds 15 (13) formerly depressed 15 (10) community control Cross-sectional Remitted cohort, age, gender and ID matched control	Not measured	SCID	None	AMT	Proportion of overgeneral memories (categoric)	Formerly depressed > control on negative cues	*U* = 66.00*	-

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Sumner et al. (2011)	16-18 year olds 55 (41) past history of MDD Longitudinal (16m f/up) Remitted cohort	Not measured	SCID	None	AMT	Proportion of specific memories	Specific memories significant predictor of MDE 16 months later	OR = 0.45*	-
Hipwell et al. (2011)	11-12 year olds 195 (195) high symptoms of depression Longitudinal (12m f/up) High risk cohort	Not measured	K-SADS-PL	None	AMT	Proportion of overgeneral memories	Correlation between current depression symptoms and OGM OGM predicted depression symptoms one year later	Spearman *rho* =.16* *B* = .02**	[0.02, 0.30] -
Valentino et al. (2012)	7-17 year olds 30 (18) abused inpatients 19 (14) psychiatric control Cross-sectional, unmatched control	Physical or sexual abuse	CDI	None	AMT	Number of overgeneral memories	No effect of abuse Abuse and depression interacted; depression only predicted OGM in no abuse group	β = -.06 β = .17*	- -

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Rawal & Rice (2012)	10-18 year olds 187 (111) controls 25 (20) depressive disorder Prospective (12m f/up) High risk cohort	Not measured	Diagnosis of MDD, or other depressive disorder	None	AMT	Number of overgeneral memories	OGM on neg cues predicted depression symptoms 12 months later for females only	β = .19*	-
Stange et al. (2013)	12-13 year olds 174 (100) experiencing emotional maltreatment Longitudinal (8m f/up) High risk cohort	Emotional abuse (EA) or neglect	CDI	None	AMT	Number of overgeneral memories	EA predicted depression in Caucasians with high but not low OGM No effect of OGM on depression	β = -.25* β = .18	- -
Nixon, Ball, et al. (2013) Study 1	7-17 year olds 24 (7) low acute stress 11 (7) high acute stress 32 (11) hospital control Longitudinal (2m f/up)	Accidental injury	CDI	CPSS CAPS	AMT	Number of specific memories	High acute stress > low acute stress[c]	*d* = 0.85*	[0.13, 1.67]

Study	Sample (no. female) and Study Design	Trauma	Depression Measure	PTS measure	AM measure	Variable	Findings	Effect Size	95% CI
Nixon, Ball, et al. (2013) Study 2	7-16 year olds 37 (15) non-PTSD trauma exposed 33 (14) PTSD Longitudinal (2m f/up)	Accident or inter-personal	CDI	CPSS CAPS	AMT	Number of specific memories	PTSD < non-PTSD	d = 0.52*	[0.28, 0.82]

Note. Effect sizes are absolute values. d = Cohen's *d;* CI = 95% confidence interval for the effect size, m = months; f/up = follow up; SES = social economic status; PTS Measure = Posttraumatic stress measure; AM measure = Autobiographical Memory measure; CAPS = Clinician Administered PTSD Scale for Children (Nader et al., 1996); CPSS = Child Posttraumatic Stress Scale (Foa, Johnson, Feeny, & Treadwell, 2001); IES = Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979); IES-R = IES-Revised (Weiss & Marmar, 1997); Children's IES = Children's Impact of Events Scale (Smith, Perrin, Dyregrov, & Yule, 2003); K-SADS= Schedule for Affective Disorders for School Age Children-Present and Lifetime Version (Kaufman et al., 1997); MDD = Major Depressive Disorder; CDI = Child Depression Inventory (Kovacs, 1992); HDRS = Hamilton Depression Rating Scale (Hamilton, 1967); SCID = Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 1995); BDI = Beck Depression Inventory (Beck & Steer, 1993); BDI-II = Beck Depression Inventory-II (Beck, Steer, & Brown, 1996); CESDS = Centre for Epidemiologic Studies Depression Scale (Radloff, 1977); β = Standardised regression coefficient; OR = Odds ratio; U = Mann-Whitney U; Pos cues = effect size for positive cues; Neg cues = effect size for negative cues

[a] Gender characteristics not reported. [b] Analysis of significance not reported. [c] Effect size calculated using total mean for both constrained and traditional AMT conditions.

- confidence interval unable to be calculated.

* $p < .05$ ** $p < .001$

OGM was typically assessed using the Autobiographical Memory Test (AMT; Williams and Broadbent, 1986). The AMT is a cued recall task using positive and negative cue words. Participants are instructed to report a specific event in response to the cue. Instructions define a specific event as lasting for less than a day, at a particular time and place. Participants are typically allowed 30-60 seconds to retrieve a memory. Responses are coded into several categories, including specific (i.e., memory contains a specific episode in which a particular time or place have been reported), or overgeneral when a specific episode is not reported. Overgeneral memories are often separated into categoric (i.e., memory summarises a number of repeated events, such as, when I go to school), or extended (i.e., memory lasted for more than one day, such as a reference to a holiday), however, for ease of the reader, this review will simply refer to specific or overgeneral memories.

Of the ten studies that considered the effect of trauma exposure, seven used the AMT. Griffith and colleagues (2012) highlighted inconsistency in AMT administration between researchers, and the limitations this places on the comparison of results. In particular, Griffith et al. (2012) demonstrated that effect sizes based on different scoring methods (e.g., the inclusion or exclusion of omissions in total number of recalled memories when calculating proportions) cannot be compared. However, all studies either examined frequencies or included omissions in their calculations (i.e., proportion overgeneral = number overgeneral / number of cue words), minimising this issue. A greater issue was variation in specific or overgeneral memories as the dependent variable. Although number of overgeneral and specific memories are inversely related (Griffith et al., 2011), effect sizes are not necessarily equivocal. Hence, effect sizes are only directly comparable between those studies using the same dependent variable.

Conceptually, however, effect sizes were similar for number of specific and overgeneral memories on the AMT. For specific memories, de Decker et al. (2003) and

Nixon and colleagues (Study 2, 2013) both reported large effect sizes relative to controls. Of the five studies that examined number of overgeneral memories on the AMT, three reported moderate to large effects of trauma exposure relative to controls (Cohen's *d*s of 0.51 to 1.76). Only one study did not support an effect of trauma exposure. Kuyken, Howell and Dalgleish (2006) demonstrated that adolescents diagnosed with major depressive disorder (MDD) who had experienced trauma were less overgeneral than an MDD group who had never experienced trauma. Further, the trauma-exposed group did not differ from the control group in number of overgeneral memories. However, repeating analyses with number of specific memories showed the trauma group retrieved fewer specific memories than control. Despite this anomaly, the majority of OGM studies using the AMT in trauma populations report a moderate effect relative to controls. Large effect sizes were also observed relative to controls for two of three studies utilising autobiographical memory measures other than the AMT, such as other cued recall tasks (e.g., Stokes et al., 2004), and coding of written questionnaires (Meesters, Merckelbach, Muris, & Wessel, 2000). Only two of the ten studies demonstrated a lack of effect on both specific and overgeneral memories (Johnson, Follmer Greenhoot, Glisky, & McCloskey, 2005; Valentino, Bridgett, Hayden, & Nuttall, 2012). Interestingly, both of these findings were in samples with elevated depression symptoms. The relationship between trauma, depression and OGM is likely to be complex, and will be later discussed, along with other potential moderators of the relationship between trauma and OGM.

Reviewed findings reveal that trauma-exposed children demonstrate OGM relative to controls across a range of trauma types. Effects sizes ranged from moderate to large. A moderate effect size was included in the confidence intervals around Cohen's *d* for all studies. This indicates that a moderate effect is the best estimate of the true effect size. This is further reflected in the larger effect sizes being reported in

studies with smaller sample sizes (e.g., de Decker et al., 2003, N = 27; Meesters et al., 2000, N = 27; Swales, Williams, & Wood, 2001, N = 51) while studies with larger sample sizes and greater power reported moderate effect sizes (e.g., Brennen et al., 2010, *N* = 140; Valentino et al., 2009, *N* = 185). Unsurprisingly, confidence intervals were wider for large effects. Further, moderate effect sizes were more frequently reported than large effect sizes. As such, the true size of the effect of trauma on OGM is likely to be moderate.

OGM and symptoms of PTSD. Only 8 studies examined the relationship between OGM and PTSD symptoms. Measures of PTSD were predominately self-report questionnaires (see Table 1). Findings were inconsistent. Four studies reported no significant relationship between OGM and symptoms of PTSD (Study 1 & Study 2, Brennen et al., 2010; de Decker et al., 2003; Vrielynck, Delpus, & Philippot, 2007). Similarly, the only study to examine the predictive ability of OGM (Study 2, Nixon, Ball, et al., 2013) demonstrated that OGM did not predict PTSD symptoms three months later. Two studies reported that participants with low posttraumatic stress demonstrated more OGM than those with clinically significant PTSD symptoms (Kuyken et al., 2006) or Acute Stress Disorder diagnoses (Study 1, Nixon, Ball, et al., 2013). That is, OGM was associated with fewer PTSD symptoms. However, one study demonstrated that OGM was positively associated with current avoidance symptoms (Stokes et al., 2004). The only study to examine OGM in those with a PTSD diagnosis (Study 2, Nixon, Ball, et al., 2013) reported that those with PTSD demonstrated more OGM than a non-PTSD, trauma-exposed comparison group. As such, the relationship between OGM and PTSD symptoms is currently unclear.

There are a number of factors that may account for the inconsistent findings. These studies variously examined a combination of total PTSD symptoms, intrusion symptoms, or avoidance symptoms as dependent variables of interest. The relationship

may be different for different symptom subscales. For example, OGM is proposed to be a type of avoidance. As such, OGM may be more strongly related to avoidance symptoms compared to intrusive symptoms. Samples also varied in terms of symptom severity (ranging from minimal to severe levels), and whether the trauma occurred months previously, or years previously. Each of these factors may contribute to discrepant findings. Most importantly, none of these studies examined the influence of time since trauma exposure. Initial theory (Williams, 1996) proposed that OGM would be adaptive for psychological symptoms in the short term following trauma as OGM would regulate negative affect. The functional avoidance mechanism was proposed to underlie this adaptive relationship. However, Williams stated that OGM would become associated with psychopathology if used in the longer term. As such, time since trauma exposure may account for why OGM is not significantly related, or negatively related to PTSD symptoms in some studies, and positively associated with PTSD symptoms in other studies. This prediction has not been previously assessed. Only one of the reviewed articles (Nixon, Ball, et al., 2013) did examine the relationship between OGM and PTSD in the months following trauma exposure, however, all other studies examined the relationship years after exposure. There is therefore a lack of evidence of whether the relationship between OGM and PTSD may be different depending on time since trauma. In sum, how OGM relates to paediatric PTSD is currently unclear. This thesis therefore aims to build upon previous findings by determining how OGM relates to psychopathology, and in particular PTSD, following childhood trauma. In doing so, this thesis provides the first assessment of whether time since trauma exposure influences these relationships.

OGM and symptoms of depression. Seventeen studies examined OGM in samples with elevated depression symptoms. The studies included both subclinical samples, examining the correlation between depressive symptoms and OGM using self-

report measures of depression in normative samples, and clinical groups, which compared number of overgeneral memories between children with lifetime diagnosis of depression and community or clinical controls. Fourteen studies supported a relationship between OGM and depressive symptoms.

Overall, moderate to large effect sizes were demonstrated (with Cohen's *d*s ranging from 0.45 to 1.10), regardless of whether reduced specificity or OGM on the AMT was examined. Effect sizes for studies using measures other than the AMT were comparable (e.g., Orbach et al., 2001). A moderate effect size was most frequently reported and was included in the confidence interval for all Cohen's *d*'s. Again, the larger effects were reported in studies with smaller sample sizes, and the confidence intervals for these effects were large. The reviewed research has therefore established that OGM is related to depressive symptoms in young people, and that the true effect is likely to be moderate in size.

While the findings indicated that MDD samples are more overgeneral than healthy controls (Park et al., 2004; Rawal & Rice, 2012), I was most interested in how OGM related to prognosis for depression. The findings offered insight into a) OGM as a vulnerability factor to predict future symptoms, and b) how OGM relates to the maintenance of depression. First, only one of the reviewed studies was prospective in design and examined the ability of OGM to predict depression in those without existing symptoms (Rawal & Rice, 2012). Rawal and Rice reported that OGM to negative cues at baseline predicted onset of depression symptoms one year later in 10-18 year old girls who were at familial risk for depression. While this is consistent with OGM as a vulnerability factor for depression, research with non-depressed samples is lacking in young people. Consistent relationships with current symptoms do however implicate OGM in depression maintenance. Cross-sectional studies indicated that depression symptoms were a unique predictor of OGM (Kuyken & Dalgleish, 2011; Valentino,

Toth, & Cicchetti, 2009). Similarly, Sumner et al. (2011) demonstrated that OGM predicted recurrence of symptoms sixteen months later in adolescents with MDD. Hipwell and colleagues (2011) also demonstrated that baseline OGM predicted depression symptoms a year later in 11-12 year olds girls with elevated symptoms at baseline. Interestingly, Rawal and Rice (2012) reported that adolescents who no longer met criteria for MDD at one-year follow-up demonstrated the same number of overgeneral memories as those participants who maintained diagnoses. Hence, reduction of depression symptoms does not necessarily relate to reduction of OGM, or vice versa. Thus OGM appears associated with the maintenance and relapse of depression symptoms in adolescence. These findings suggest OGM is a potential intervention point for adolescent depression.

Of all studies reviewed, those that reported no significant relationship between OGM and depression (de Decker et al., 2003; Meesters et al., 2000; Stange, Hamlat, Hamilton, Abramson, & Alloy, 2013; Valentino et al., 2012) utilised trauma-exposed samples. Further, Kuyken et al. (2006) reported that their MDD group without a trauma history demonstrated greater OGM than community controls; however, there was no significant difference between the MDD group with a trauma history and controls. It appears the effects of trauma, OGM, and depression interact. Initial theory (Williams, 1996) stated that OGM would regulate negative affect produced by specific memories of adverse childhood events. That is, OGM would be beneficial in the early stages following trauma, and would only become associated with psychopathology if maintained in the long-term. These findings are consistent with this proposal. As participants were trauma-exposed, OGM may have been affect regulating and adaptive. Thus, OGM would not be related to depression. This is still consistent with the observed relationship between OGM, childhood trauma and adult onset depression, as OGM will become maladaptive if maintained until adulthood. I was unable to thoroughly assess

this proposition due to a lack of studies that simultaneously examine trauma, OGM and depression in younger samples. As such, this thesis simultaneously examined trauma exposure, depression and OGM to more thoroughly examine interactive relationships.

Whether OGM constitutes a vulnerability for depression, or is caused by depression has been much debated (see Sumner, Griffith, & Mineka, 2010). However, at this time there is insufficient research to conclude whether OGM is a vulnerability or maintenance factor for adolescent depression. The reviewed research has indicated that it is likely to be a combination of the two. Importantly, current research exploring a vulnerability or maintenance role of OGM has only examined samples at increased risk (e.g., previous major depressive episode, currently elevated symptoms or familial risk). In order to establish that OGM is a vulnerability factor, future research will need to longitudinally examine OGM in normative samples. This thesis began to address this issue. Despite a lack of clarity about the direction of the relationship, a moderate relationship between depressive symptoms and OGM is consistently observed.

Previous Evidence for the CaR-FA-X Model in Childhood OGM

The reviewed studies also provide insight to the application of the CaR-FA-X model to children. Seven studies determined whether OGM was significantly related to the cognitive processes outlined by the CaR-FA-X model. The CaR-FA-X model was developed for OGM in adults, however, given strong evidence of OGM in childhood psychopathology, and adult models of PTSD and depression have been applied to children (McKinnon, Nixon, & Brewer, 2008; Meiser-Stedman, 2002), it is important to assess if the model accounts for OGM in childhood. Here I review current support for each mechanism. Valentino (2011) emphasised our limited understanding of the childhood experience of OGM, and promoted consideration of developmental stages when theorising OGM in children. Accordingly, I examine developmental aspects that may affect the mechanisms and how this indicates that separate components of the

model may play a larger role in OGM dependent on the developmental stage of the child.

Capture and Rumination

In adults, research has examined relationships between negative self-concepts/beliefs or rumination and OGM (e.g., Watkins & Teasdale, 2001). In children and adolescents, examination of this mechanism is limited. Valentino et al. (2009) supported the capture aspect by demonstrating that negative self-beliefs uniquely predicted OGM, accounting for 28% of variance. Park et al. (2004) explored rumination by manipulation of either rumination or distraction prior to administering the AMT. The authors recruited teenagers with MDD, alternate psychiatric diagnoses or no diagnoses. Within the MDD group, change from pre- to post-manipulation indicated those instructed to ruminate on the self (e.g., emotions, attributes) experienced an increase in proportion of overgeneral memories to negative cues compared to the distraction condition. No significant difference was seen between pre- and post-measures in the distraction condition. These findings are consistent with the mechanism, particularly as the participants were instructed to ruminate on the self. Furthermore, that change was only observed in response to negative cues supports the notion that those with strong negative self-beliefs will be more sensitive to capture errors. Thus, two studies (Park et al., 2004; Valentino et al., 2009) have demonstrated that negative self-beliefs and rumination are associated with greater OGM. This mechanism offers a promising area for future research.

Reduced Executive Control

To date OGM research in children has focussed on inhibition and working memory capacity, as outlined by Williams et al. (2007). Current findings are mixed.

Inhibition. Williams et al. (2007) stated that for a specific memory to be retrieved, irrelevant information needs to be inhibited during the retrieval process.

Although norms for inhibitory control are available for children (e.g., Manly et al., 2001), and inhibition has been studied extensively in childhood disorders (e.g., attention deficit hyperactivity disorder; Brocki, Eninger, Thorell, & Bohlin, 2010; Geurts, van der Oord, & Crone, 2006), only two studies have examined inhibition in childhood OGM. Raes and colleagues (2010) demonstrated that scores on the inhibitory control subscale of the Revised Early Adolescent Temperament Questionnaire (Ellis & Rothbart, 2001) partially mediated the relationship between depression symptoms and number of overgeneral memories on the AMT in primary school students. Interestingly, the authors utilised a personality measure of inhibition, rather than a behavioural or cognitive measure. When Valentino et al. (2012) utilised a cognitive measure of inhibition (i.e., Colour-Word Interference Task), no correlation was found. Further research is clearly required, especially that using more traditional and objective measures of inhibition.

Working memory. The maintenance of a retrieval model and retention of the recalled memory relies on working memory capacity. Only two of the reviewed studies included a measure of working memory capacity. Although Nixon, Ball, et al. (Study 1, 2013) reported a positive correlation between working memory capacity and specific memories, de Decker et al. (2003) reported no correlation. Verbal fluency (i.e., ability to retrieve verbal information from memory) may also tap impairments in the retrieval process as it too loads onto the updating component of executive control, however Kuyken et al. (2006) reported no relationship between verbal fluency and autobiographical memory. Valentino et al. (2012) separated verbal fluency further, measuring letter fluency (i.e., ability to quickly produce words beginning with a certain letter) and category fluency (i.e., ability to quickly produce words that fit into a certain category). Although they reported no effect of letter fluency, category fluency (which requires retrieval of semantically as opposed to phonetically related information) did

uniquely account for variance in number of overgeneral memories. Further research separating unique aspects of executive control will more clearly assess this mechanism.

Functional Avoidance

In adults, the functional avoidance mechanism has been assessed by examining avoidant coping or whether a specific retrieval style increases stress following a negative event (e.g., Raes, Hermans, de Decker, Eelen, & Williams, 2003; Williams, Eelen, Raes, & Hermans, 2006). Surprisingly, the mechanism has yet to be examined in children, despite the proposal that functional avoidance will occur in childhood as a result of trauma. If we consider that functional avoidance is a reflection of more general avoidance, two studies may offer some insight. Stokes et al. (2004) reported a positive relationship between avoidance and OGM in a sample of adolescents, while Kuyken et al. (2006) demonstrated a negative relationship. Direct examination of functional avoidance is clearly needed, particularly as functional avoidance forms the basis of current theory.

Developmental Aspects of the Mechanisms

Developmental literature suggests that the impact of the individual mechanisms may vary depending on the child's age. Research on rumination has indicated that although no age differences are present in children younger than 12 years (Abela, Vanderbilt, & Rochon, 2004; Broderick, 1998; Jose & Brown, 2008), preadolescents demonstrate less rumination than early adolescents (Hampel & Petermann, 2005). Furthermore, rumination increases with age during teenage years, particularly for females (Jose & Brown, 2008). In applying this literature to the model, age may moderate the relationship between rumination and OGM. Similarly, separate components of executive control are seen to reach adult-like abilities at different points. Research indicates that inhibition develops continuously until approximately 12 years of age, with no further improvement from this point (e.g., Brocki et al., 2010; Levin et al.,

1991; Welsh, Pennington, & Groisser, 1991). Further, working memory capacity increases in an approximately linear fashion during childhood and adolescence (e.g., Crone, Wendelken, Donohue, van Leijenhorst, & Bunge, 2006; Gathercole, 2004; Hale, Bronik, & Fry, 1997). Together, this suggests that executive control errors may be more common in children compared to adolescents.

Finally, functional avoidance may be engaged more in younger children due to their possessing fewer emotion regulation strategies (Cole, Michel, & Teti, 1994). Again, avoidant coping may tap this mechanism (Hermans, Defranc, Raes, Williams, & Eelen, 2005). Research on avoidant coping has demonstrated a negative relationship with age (Phipps, Fairclough, & Mulhern, 1995), consistent with increased functional avoidance in young relative to older children. In sum, developmental literature suggests the functional avoidance and executive control mechanisms may be more prominent in childhood, while capture and rumination may play a larger role in older children and adolescents. However, the moderating effect of age has yet to be considered in relationships between OGM and the mechanisms. This thesis examines developmental effects to advance theory of OGM.

Summary and Aims of this Thesis

The overarching goals of this thesis are to clarify how OGM relates to psychopathology following childhood trauma and to assess the cognitive processes underlying childhood OGM. Exploring the first goal has important clinical implications. This review has highlighted that although OGM has been consistently related to both childhood trauma and depression, a greater understanding of the direction and nature of these relationships is needed before OGM can be addressed in clinical practice. Although OGM is implicated in the maintenance of adolescent depression, the relationship between OGM and paediatric PTSD has yet to be determined. OGM predicts poor prognosis for PTSD in adulthood (Harvey, Bryant, & Dang, 1998),

however theoretical models were developed with the view that OGM may be an affect regulating strategy, and therefore could be adaptive immediately following childhood trauma (Williams, 1996). This proposal has not been adequately assessed. As this review highlighted, a finding that OGM can change from being adaptive to maladaptive could explain discrepant findings of how OGM relates to trauma and psychopathology. Prior to extending emerging clinical interventions to children (e.g., Neshat-Doost et al., 2013), we must first determine if there is a time that OGM may be adaptive following childhood trauma. Failure to do so may see that clinical intervention is completed when OGM may in fact be adaptive for children. Similarly, greater understanding of how OGM relates to the development of depression and PTSD will improve understanding of the aetiology of these disorders. This thesis therefore provides the first exploration of the trajectory of OGM and psychological symptoms following childhood trauma.

Assessing the cognitive mechanisms underlying OGM also has important theoretical implications. This review indicated that the cognitive processes underlying childhood OGM have not been thoroughly tested. Current examination of the dominant OGM theory (i.e., the CaR-FA-X model; Williams et al., 2007) in young people is very limited. The capture and rumination, and executive control mechanisms have each received a small amount of support, and warrant further investigation. More importantly, the functional avoidance mechanism has yet to be adequately examined. As such, further investigation of all three of the mechanisms is justified. The reviewed literature has also demonstrated a gap in our knowledge regarding the developmental effects on these mechanisms. Direct examination of these mechanisms would provide firm evidence as to whether the model applies to children. Hence, this thesis provides the first simultaneous assessment of all three mechanisms of the CaR-FA-X model, and the most comprehensive theoretical examination of childhood OGM to date.

In closing these gaps in the literature, I address two key research questions. First, how does OGM relate to psychopathology following childhood trauma exposure? Specifically, this thesis aims to determine a) how OGM relates to symptoms of PTSD, and b) whether time since trauma exposure moderates the relationship between OGM and symptoms, such that OGM may be adaptive immediately following trauma exposure. Second, do the CaR-FA-X mechanisms account for childhood OGM? Examination of these questions comprises of four studies. Study 1 and Study 2 aim to determine if the relationship between OGM and psychopathology changes over months or years following childhood trauma exposure. These studies simultaneously assess whether the CaR-FA-X mechanisms predict OGM. Based on the findings from these studies, Studies 3 and 4 shift focus towards the functional avoidance mechanism, which is proposed to underlie the adaptive role of OGM. I experimentally manipulate retrieval specificity in primary school children to assess key assumptions of the functional avoidance mechanism. As will be documented, this thesis will demonstrate that time since trauma exposure is an important influence on the relationship between OGM and psychopathology. Further, evidence was found that indicates the functional avoidance mechanism alone, as opposed to the entire CaR-FA-X model, best explains OGM following childhood trauma.

CHAPTER 2

Data Analysis Approach

Before detailing the first study of the thesis, it is important to explain the unique data analysis approach used in this thesis. I employed a linear mixed-effects modelling approach using the lme4 package (Bates, Maechler, & Bolker, 2011) in the R environment for statistical computing (R Development Core Team, 2011). Linear mixed-effects modelling is an extension of regression. Ordinary regression examines the relationship between scores for standard, fixed effects and scores on the outcome. When fitting a regression model, it is assumed that the scores for each participant are independent from one another. This makes regression unsuitable when scores from some participants may be more strongly related to one other than to scores from other participants. For example, when we sample from different schools, participants from the same school are likely to have more closely related scores due to demographic factors and shared experiences, compared to scores from participants from two different schools. Mixed-effects regression modelling allows us to take these similarities into account by specifically modelling these associations as random effects. When examining our standard, fixed effects, we can account for the variability in intercept (overall outcome score) and slope (the relationship between the fixed effects and the outcome) due to the random effects. That is, mixed-effects regression modelling allows us to consider both the standard fixed effects, and also random effects.

Some of my research questions would typically be considered ANOVA questions. ANOVA is simply a specific presentation of the results of regression (Field, Miles, & Field, 2012). That is, when testing the difference between means, we are fitting a regression model, but the regression model includes only categorical predictors (i.e., a grouping variable; Field et al., 2012). Therefore, questions that are typically answered using ANOVA can be answered equally effectively with regression analyses.

The use of mixed-effects regression models in particular (i.e., linear mixed-effects modelling), offers a number of benefits over ANOVA.

First, in essence, linear mixed-effects modelling allowed me to consider the effects of fixed factors, after variation in the outcome due to random effects have been accounted for. If differences in the outcome within such random effects are meaningful (e.g., one school demonstrates more symptoms of psychopathology than the other schools), an ANOVA model's approximation of the effect of the fixed factor may mask a real effect, or even falsely suggest an effect that is unlikely to have occurred. Linear mixed-effects models allow us to take account of variation in the effects of these other variables (i.e., school, age) as random effects. That is, we can account for variation in the outcome due to the random effects, when evaluating the fixed effect of interest. ANOVA models assume that observations of the fixed effect are independent. However, my data violates this assumption as data from several studies are grouped by schools. Historically, ANOVA has been used even when this assumption is violated, as there have not been alternative analyses. Linear mixed-effects modelling presents an alternative, more appropriate statistical approach in this situation.

Additionally, use of linear mixed-effects modelling allowed me to collapse data over multiple assessments while still modelling variance by including assessment date as a random effect. Finally, I was able to include participants who were missing data due to attrition. That is, the model was fit for those values that were available for the variable (e.g., for the assessments that the individual did complete), rather than excluding all data on that variable, as occurs in ANOVA models. Overall, the use of this data analysis approach was more powerful than ANOVA, and allowed me to include variability due to random effects in my models, rather than simply treating this variability as noise, as it is in ANOVA models. This was particularly important as data

were collected from multiple sites, over multiple observations, and across a wide age range (i.e., 7-17 year olds).

The creation of a linear mixed-effects model, and the interpretation of results, is essentially equivalent to conducting a regression analysis. First, I fitted a baseline model to the data that included only the intercept. I then added predictors to the model in hierarchical steps, and examined improvement in fit. The improvement in fit of the new model over the previous model was evaluated by examining the likelihood ratio. The likelihood ratio assesses the reduction in deviance, that is, the difference between the model predictions and the observed data, from the previous step to the added step. The model of best fit was the most complex model (i.e., the most predictors) that significantly improved the fit of the model from the previous step.

Once the model of best fit has been determined, the *b* coefficients indicate the effect size associated with a factor. Specifically, the coefficient indicates how much change would be predicted in the outcome variable for a one unit change in the predictor. As in regression, entering a categorical predictor (e.g., group), into the model requires use of a reference group. That is, when comparing groups, I examined how much change was observed in one group, relative to a reference group. For example, when comparing trauma-exposed and non-exposed children, I used the non-exposed group as the reference group. I then examined the *b* coefficient for the trauma-exposed group to determine the size of the effect. A *b* coefficient of -1.02 would indicate that the trauma-exposed group was predicted to be, on average, 1.02 units lower on the outcome variable, relative to the non-exposed group. I calculated significance (*p*) values for fixed effect coefficient estimates on the basis of Markov chain Monte Carlo samples (Baayen, Davidson, & Bates, 2008). To aid interpretation, I have also presented more familiar aggregate descriptive statistics based on the observed data.

I also reported estimated standard deviations for random effects from the model of best fit. The standard deviation associated with a random effect reflects the predicted variance in model intercept associated with a change in the random effect. That is, variation in outcome, independent of the fixed effect. For example, the standard deviation for the random factor of participant reflects the variability that would be predicted between participants in the outcome variable (e.g., difference in the number of memories produced due to individual differences).

To conclude, use of linear mixed-effects modelling allowed me to model variability in the outcome due to random effects when examining the relationship between fixed effects and the outcome. This resulted in more powerful analysis of my data than a traditional ANOVA approach. For each analysis, I have described how the model was created. This has included outlining the random effects included in each model, and the order the fixed effects were entered into the model. I then evaluate the *b* coefficients for the model of best fit to interpret the size and significance of the effect.

CHAPTER 3

Study 1[3]

This study explored the mechanisms underlying OGM, and how the style related to psychopathology in the first six months following childhood trauma exposure. Chapter 1 indicated that OGM may form a cognitive vulnerability for recurrent depression in adolescence (e.g., Hipwell et al., 2011; Kuyken & Dalgleish, 2011; Rawal & Rice, 2012). Indeed, the phenomenon has been found to predict depression symptoms up to and over a year after assessment (Rawal & Rice, 2012; Sumner, Griffith, Mineka, et al., 2011). However, how OGM relates to paediatric PTSD is unclear. Although research with adult samples has established that OGM is associated with poorer prognosis for PTSD (Moore & Zoellner, 2007), theory suggests that OGM may be adaptive initially following childhood trauma (Williams, 1996). That is, theory states that OGM may be associated with emotional stability immediately after trauma exposure, and only become associated with psychopathology if maintained over the long term (Williams, 1996). No previous work has longitudinally examined OGM immediately after childhood trauma exposure to assess this proposal. This study sought to close this important gap in the literature.

Theory of OGM began with the idea that adverse childhood experiences may cause interruptions to the generative retrieval process through which specific memories are retrieved (Williams, 1996). More specifically, Williams proposed that a child would begin to actively avoid specific memories in order to avoid negative affect produced by recollection of their adverse experience. This functional avoidance hypothesis stated that avoidance of specific memories would regulate negative affect and, as such, help the child manage emotional disturbance. However, over time, the adaptive function of

[3] A large proportion of this chapter has been taken from Hitchcock, C., Nixon, R.D.V., & Weber, N. (in press). A longitudinal examination of overgeneral memory and psychopathology in children following recent trauma exposure. *Applied Cognitive Psychology.*

OGM would decline, and much like other forms of avoidance in clinical disorders, OGM would become associated with psychopathology (e.g., by impairing problem solving; Williams et al., 2007). Hence, time since trauma exposure is proposed to moderate the relationship between OGM and psychopathology.

No study has longitudinally examined OGM development after childhood trauma exposure, and as such, whether OGM may be initially adaptive has yet to be assessed. Although support has been found for increased OGM following childhood trauma (e.g., Brennen et al., 2010; de Decker et al., 2003; Stokes et al., 2004), how trauma relates to subsequent psychopathology is less clear. Currently, three studies with young people have reported no relationship between OGM and symptoms of PTSD (Brennen et al., 2010; de Decker et al., 2003; Vrielynck et al., 2007), two studies have reported that those with elevated avoidance symptoms (Stokes et al., 2004) or diagnosed PTSD (Study 2, Nixon, Ball, et al., 2013) show increased OGM, and two have reported that those with elevated avoidance (Kuyken et al., 2006) or acute stress symptoms (Study 1; Nixon, Ball, et al., 2013) show less OGM. Previous research has not indexed time since trauma exposure in examining the relationship between OGM and psychological symptoms, despite trauma characteristics being implicated as potential moderators of the relationship, particularly for depression (Valentino et al., 2012). However, differences in time since trauma exposure may contribute to discrepant findings. That is, if Williams' (1996) theory is correct, no, or a negative, relationship would be expected between OGM and symptoms immediately following trauma, and a positive relationship would be expected to emerge in the longer term. Examining how OGM relates to psychopathology immediately post-trauma will inform theory, and also expand understanding of cognitive risk factors for emotional disturbance. It is important to determine if there is a time that OGM is adaptive as clinical interventions that target OGM to reduce depression (e.g., Neshat-Doost et al., 2013) may have a detrimental

impact on symptoms if administered shortly following trauma. Therefore, the primary aim of this study was to determine how OGM related to symptoms of depression and PTSD in the first six months following childhood trauma exposure, and in particular, whether time since exposure moderated this relationship.

The second aim of this study was to assess the mechanisms through which childhood trauma may interrupt the retrieval of specific memories. Williams and colleagues (2007) expanded the functional avoidance hypothesis to include other mechanisms through which generative retrieval may be interrupted. The CaR-FA-X model (Williams et al., 2007) outlines three processes through which OGM can occur: capture and rumination (CaR), functional avoidance (FA), and reduced executive control (X). The capture and rumination mechanism interrupts the search by diverting attention towards irrelevant information that is then ruminated upon. For example, when searching for a memory of a party, information on feeling ignored at parties may capture attention, which may lead the individual to ruminate on why no one likes them. Hence, rumination is a key component of the model. As described above, the functional avoidance mechanism avoids specific information in order to reduce negative affect. Finally, reduced executive control will mean impaired guidance and maintenance of a controlled search. For example, impaired executive control may reduce the ability to update information held in working memory or limit inhibition of irrelevant information during the memory search. Williams et al. outlined inhibition and working memory as the main executive processes that may influence retrieval of specific memories.

A modest amount of research has examined these processes in children, with prior work focussing on rumination and executive control processes. Mixed support has been provided for inhibition, with Raes and colleagues (2010) demonstrating that inhibition partially mediated the relationship between depression symptoms and OGM in a non-clinical sample of primary school students. In contrast, Valentino et al. (2012)

found no evidence for a significant relationship between inhibition and OGM in a psychiatric sample. Other executive functions that have been assessed include verbal fluency and working memory capacity. No firm support has been provided for these processes (see de Decker et al., 2003; Kuyken et al., 2006; Valentino et al., 2012). Only one study demonstrated that category fluency was related to OGM (Valentino et al., 2012). In terms of rumination, Park et al. (2004) demonstrated that experimentally induced rumination was related to increased retrieval of overgeneral memories in adolescents experiencing Major Depressive Disorder (MDD). It is interesting that support for rumination and executive control has been found in samples with disordered mood, whereas the original model (Williams, 1996) focussed on describing OGM following trauma exposure. Importantly, while trauma exposure seems important in increasing functional avoidance, the rumination and executive control mechanisms were introduced to account for OGM in samples without a trauma history (Williams et al., 2007). As trauma exposure may increase activation of these mechanisms (e.g., rumination on blame for the adverse event; Williams et al., 2007), I was interested in whether the rumination and executive control mechanisms may also operate in a trauma-exposed sample.

To summarise, this study addressed two key questions: (1) are the rumination and executive control mechanisms applicable to a trauma-exposed sample of children, and (2) does the relationship between OGM and psychopathology change over time? To examine these questions, I completed three assessments of psychological symptoms, OGM, rumination, and executive control over six months following trauma exposure. Although six months may not be long enough to see changes in the relationship between OGM and symptoms, it does offer insight as to whether OGM is initially adaptive following trauma. I hypothesised that rumination, inhibition, and working memory measures would significantly predict OGM. I also predicted that time since trauma

exposure would moderate the relationship between OGM and symptoms of PTSD and depression. As the CaR-FA-X model is unclear on the exact timeline of when OGM may be adaptive and maladaptive, I did not make specific predictions about the direction of the relationship at each time point. However, in line with the model I did expect that a negative relationship would be seen initially (i.e., more OGM would be associated with fewer symptoms).

Method

Participants

Fifty seven children who had experienced a single-incident accidental injury were recruited in person while attending a metropolitan public hospital.[4] Experienced events included motor vehicle accidents, extreme sport accidents (e.g., Motocross or BMX), incidents with dangerous equipment (e.g., power tools, spear fishing equipment) and falls (e.g., off a roof or cliff, or out of a tree). All of these events have been associated with acute stress in a paediatric sample (Daviss et al., 2000). Exclusion criteria included injury resulting from physical or sexual abuse, and traumatic brain injury/loss of consciousness as a result of the trauma. Ages ranged from 7-17 years (M = 11.90 years, SD = 3.31 years). All participants were Caucasian. No participants were receiving psychological treatment nor taking psychotropic mediation.[5] No participants had previous diagnoses of depression, PTSD, or anxiety.

Six children withdrew from the study prior to the first data collection point, and one participant was excluded due to ongoing treatment for an eating disorder. Hence, I analysed data for 50 (10 female) participants. I conducted three assessments: one month (T1), three months (T2), and six months (T3) post-trauma. Four participants were lost to attrition over the six months (one moved interstate, two declined further assessments

[4] I also collected data from a healthy control group to examine differences between trauma-exposed and non-exposed children. However, the control group did not match well for age and gender. As such, they did not form an appropriate comparison group and will not be further discussed.

due to time commitments, and one declined due to feeling uncomfortable talking about her trauma). Participants received a $10 gift voucher as compensation at each assessment (i.e., total of $30 over six months).

Estimation of power for within-subjects designs requires knowledge of the correlation between observations (in this case, T1, T2 and T3). As this is difficult to estimate a priori, I was conservative and assumed no correlation between observations. That is, I treated my analyses as between-subjects comparisons. To detect a medium effect at power of 0.80, Cohen's conventions (1988) recommend 33 participants per cell. Thus, as I only had one cell, recruiting a minimum of 33 participants should have ensured adequate power. Following completion of data collection, I completed post hoc analysis of effective power to ensure that my initial estimation of sufficient sample size was accurate. I completed Pearson's correlations between the number of overgeneral memories (the key variable) at T1 and T2 (r = .56, n = 46, p < .001), T2 and T3 (r = .53 n = 45, p < .001), and T1 and T3 (r = .47, n = 45, p < .001). Following the guidelines of Rosenthal and Rosnow (2008), the smallest correlation observed (r = .47) with this sample size produces power of .85. As such, this study possessed adequate statistical power.

Measures

Autobiographical memory test (AMT; Williams & Broadbent, 1986). The AMT possesses adequate psychometric properties with young people (Heron et al., 2012) and is commonly used to assess OGM (Griffith et al., 2012). The AMT is a cued-recall task with five positive and five negative cue words. I used a different word list at each of the three time points, and all words were taken from previous studies of OGM (e.g., Kuyken et al., 2006; Valentino et al., 2009). The lists were balanced for word frequency and imagery (all three lists are presented in Appendix A). Different word lists

[5] Three children were referred to psychologists at the conclusion of the study.

between assessments are commonly used to reduce practice effects (e.g., Raes et al., 2006; Rawal & Rice, 2012). Factor analysis has demonstrated the consistency of OGM between different cues, suggesting that different cues will produce a similar rate of OGM (Heron et al., 2012). Participants were instructed to provide a specific memory (i.e., one particular time that something happened) for each cue word. Examples of correct and incorrect memories were given in instructions. Instructions and all cue words were simultaneously presented in written and oral form. Participants were allowed sixty seconds to orally report a memory. Two practice trials with neutral words (*egg* and *beach)* preceded the scored cues to ensure the child understood the task. Responses were audio-recorded and coded as specific, overgeneral (categoric or extended), or omission by the first author. Interrater reliability with an independent rater was adequate. Raters agreed on 90.3% of responses ($\kappa = .84$), which is comparable to previous studies (e.g., Kuyken et al., 2006). Number of overgeneral memories was used as the dependent variable in all analyses. Repeating analyses with number of specific memories did not alter the results, except where noted.

Trauma history. An adaptation of the Cambridge Life Development Measure (Goodyer, Herbert, Tamplin, Secher, & Pearson, 1997) indexed trauma history. Children indicated which of 13 adverse life events, such as the death of a family member, had ever occurred to them. They also rated the emotional nature of the event on a five-point scale, from 1 (pleasant/really happy) to 5 (unpleasant/sad/painful). Events rated as a five were counted as potentially traumatic as some events (e.g., someone leaving your family) may be considered positively by the child. In addition to listed events, an open-ended question asked about any previous trauma exposure. The rating scale was completed for any events reported. Trauma history was operationalized as the total number of events which were rated as five at T1. I administered this measure at all assessments to control for exposure to additional events over the duration of the

study. Thirty eight percent of participants (*n* = 19) had experienced a potentially traumatic event prior to the event indexed in the study.

Depression. The Child Depression Inventory – Short Form (CDI-S; Kovacs, 1992) measured symptoms of depression. Adequate alternate-form reliability exists between the short and full forms (*r* = .89; Kovacs, 1992). Normed scores were used to identify those who placed in the clinical range for depression (i.e., a T score of 65 or greater).

Posttraumatic stress. The Child Posttraumatic Stress Scale (CPSS; Foa, Johnson, Feeny, & Treadwell, 2001) contains one item for each of the seventeen PTSD symptoms listed in the Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition (DSM-IV; American Psychiatric Association, 2000), along with a functional impairment scale. I calculated a total symptom severity score and scores for the symptom clusters of re-experiencing, avoidance, and arousal. The symptom severity scale has been found to have adequate internal consistency (α = .89), convergent validity (*r* = .80) with the Child Posttraumatic Stress Reaction Index (Pynoos et al., 1987), and test-retest reliability (*r* = .84; Foa et al., 2001). I used the revised clinical cut-off of 16 (Nixon, Meiser-Stedman, et al., 2013).

Rumination. The Children's Response Style Scale (CRSS; Ziegert & Kistner, 2002) is a self-report questionnaire consisting of rumination and distraction subscales. Research has demonstrated that the rumination and distractive response style scales load onto different factors (Ziegert & Kistner, 2002), validating the use of the rumination subscale individually. The rumination subscale has demonstrated good internal consistency (α = .81), test-retest reliability (*r* = .69), and convergent validity (*r* = .39) with the rumination factor of the Children's Responses to Hypothetical Events Questionnaire (Broderick, 1998; Ziegert & Kitsner, 2002).

Working memory. I assessed working memory capacity using the Digit Span subtest of the Wechsler Intelligence Scale for Children- Fourth Edition (WISC-IV; Wechsler, 2003). I also assessed working memory updating using a computerised n-back task. The task is suitable for use with school-age children (Vuontela et al., 2003). A series of letters is presented one at a time on the computer screen and the child is required to press the spacebar when the current letter is the same as a target letter that came, depending on condition, one, two, or three letters before it. No response was needed if the letter was different from the target letter. Sequences of forty letters were presented for 2 seconds each with a 1 second inter-stimulus interval. A 0-back (i.e., press the space bar when the letter 'H' is on screen) practice, 1-back, 2-back, and 3-back condition were completed. In each condition, twelve target and twenty eight filler letters were presented. For the 1-, 2-, and 3-back conditions, ten practice trials were completed. During these, corrective feedback was given. N-back score was calculated as the proportion of targets that were correctly identified (i.e., the proportion of targets for which the spacebar was pressed).

Verbal fluency. Verbal fluency requires monitoring of verbal retrieval, and as such is proposed to measure executive abilities needed for retrieval of specific memories (Valentino et al., 2012). The Verbal Fluency Task (VFT; Tombaugh, Kozak, & Rees, 1999) was administered to assess the ability to retrieve verbal information. Participants were given a minute to recall as many words as possible that start with a specified letter ('F' at T1 and 'S' at T3). Instructions stated that names and word variations (e.g., fast, faster) were not allowed. The version of the test using the letters 'F', 'A', and 'S' has adequate internal consistency ($\alpha = .83$) and test-retest reliability ($r = .74$; Tombaugh et al., 1999). The number of words recalled was used as the VFT score.

Inhibition. The Walk, Don't Walk subtest of the Test of Everyday Attention for Children (Manly, Robertson, Anderson, & Nimmo-Smith, 1999) requires the child to maintain attention and inhibit an automatic response over a six minute period. The task possesses adequate test-retest reliability (Manly et al., 1999), and has norms up until the age of 15 years. Although all participants completed the measure, inhibition data were only used for the 86% for whom norms were available. This measure was chosen as it is an objective, standardised measure of inhibition errors, and theory states errors in inhibition will produce OGM.

Procedure

Data collection was approved by the Clinical Research Ethics Committee. Informed consent was gained from the child's legal guardian prior to participation. Verbal assent was also received from the child prior to each session. All sessions were one-on-one and occurred in a quiet room at the young person's home or the university. The AMT was administered first in order to prevent priming effects that may have arisen due to completing the symptom measures first. This was followed by trauma history, CPSS, CDI-S, and CRSS. All questionnaires were read aloud by the researcher. The VFT and Digit Span subtest were then completed, and finally the Walk, Don't Walk and n-back tasks. Participants completed all measures at T1 and T3. Alternate versions of the Walk, Don't Walk and Verbal Fluency tasks were administered at T1 and T3 to reduce practice effects. At T2, the executive control measures were excluded to reduce participant burden.

Results

Throughout this thesis, an alpha level of .05 was used in all analyses. All reported confidence intervals are 95% confidence intervals. To address the key research questions, I used a Gaussian linear mixed-effects modelling approach using the lme4 package (Bates et al., 2011) in the R environment for statistical computing (R

Development Core Team, 2011). Participant was entered as a random effect in all models. As in regression, the *b* coefficient indicates the size of the effect. Specifically, the coefficient demonstrates how much change I would expect in the dependent variable for a one unit change in the predictor. I report estimated standard deviations for random effects from the model of best fit for all analyses. This value reflects the estimated variance in model intercept between different levels of the random factor (i.e., between participants).

At T1, 24% (*n* = 12) of participants reached the clinical cut-off on the CPSS (*M* = 9.74, *SD* = 7.75, [8.65, 10.83]). Only 6% (*n* = 3) reached the clinical cut-off on the CDI-S (*M* = 46.84, *SD* = 8.72, [45.61, 48.07]). Trauma history was not uniquely related to OGM (*b* = 0.39, SE_b = 0.22, *p* = .49), participant *SD* = 1.61, $\chi^2(1) = 22.84$, $p < .001$. Considering that my sample was predominately male, it is important to note that gender was not significantly related to OGM, (*b* = -0.44, SE_b = 0.68, *p* = .52), participant *SD* = 1.36, $\chi^2(1) = 0.43$, *p* = .51. I included age in all analyses as it significantly predicted OGM (*b* = -0.02, SE_b = 0.01, *p* = .003), participant *SD* = 1.34, $\chi^2(1) = 8.83$, *p* = .003.

Do the CaR-FA-X Mechanisms Apply to This Sample?

Rumination. A mixed-effects model was created to assess whether rumination predicted number of overgeneral memories uniquely or in interaction with time or age. The initial model included participant as a random effect (*SD* = 1.37) along with age and time as fixed effects. The next step added rumination. Although adding rumination improved the fit of the model, $\chi^2(1) = 4.31$, *p* = .04, rumination itself was not significantly related to OGM (*b* = 0.11, SE_b = 0.11, *p* = .30).[6] The fit of the model was

[6] Adding a predictor to a mixed-effects model can allow better estimates of other coefficients (particularly random effects) and, can therefore improve the fit of the model, even when the coefficient of the added effect does not significantly differ from zero (i.e., the additional predictor is not significantly related to the outcome).

Table 2

Summary of Means and Standard Deviations for Psychological Symptoms, Cognitive Measures and AMT Performance by Assessment

Variable	Possible Scores	Time 1			Time 2			Time 3		
		n	*M* (*SD*)	95% *CI*	*n*	*M* (*SD*)	95% *CI*	*n*	*M* (*SD*)	95% *CI*
PTSD symptoms (CPSS)	0-51	50	9.74 (7.75)	[7.54, 11.94]	46	9.87 (7.06)	[7.77, 11.97]	45	7.87 (7.69)	[5.56, 10.18]
Depression symptoms (CDI-S)	39-100	50	46.84 (8.72)	[44.36, 49.31]	46	46.07 (7.21)	[43.93, 48.21]	44	44.43 (5.35)	[42.80, 46.06]
Rumination	0-10	49	4.09 (2.15)	[3.47, 4.70]	45	3.80 (2.11)	[3.17, 4.43]	45	3.63 (2.40)	[2.64, 4.08]
Inhibition	1-19	44	6.86 (4.62)	[5.46, 8.27]	-	-	-	38	7.11 (3.65)	[5.91, 8.31]
Verbal Fluency	0 – no upper limit	50	8.32 (3.62)	[7.29, 9.35]	-	-	-	45	10.40 (4.23)	[9.13, 11.67]
WM capacity	0-19	49	9.45 (2.07)	[8.86, 10.05]	-	-	-	43	9.28 (2.75)	[8.43, 10.13]
WM updating	0-1	40	.73 (.17)	[0.68, 0.78]	-	-	-	45	.79 (0.11)	[0.76, 0.82]
Number of Specific memories	0-10	50	4.34 (2.65)	[3.59, 5.09]	46	3.28 (2.66)	[2.49, 4.07]	45	4.49 (2.31)	[3.76, 5.18]
Number of Overgeneral memories	0-10	50	3.90 (2.51)	[3.19, 4.61]	46	4.74 (2.65)	[3.95, 5.53]	45	2.13 (2.06)	[1.51, 2.75]

Note. WM = working memory; WM updating is the proportion correct.

not improved by adding an interaction with age, $\chi^2(1) = 0.14$, $p = .71$, or time, $\chi^2(1) = 0.13$, $p = .72$. Thus, I had no evidence that rumination was significantly related to OGM.

Executive control. I started from the same initial model as the above analysis and inhibition was added in the second step. Inhibition, operationalized as score on the Walk, Don't Walk task, significantly improved model fit, participant $SD = 1.33$, $\chi^2(1) = 50.18$, $p < .001$, however it was not uniquely related to OGM ($b = 0.01$, $SE_b = 0.06$, $p = .87$). The model was not significantly improved by adding an interaction between inhibition and age, $\chi^2(1) = 1.14$, $p = .29$, or time, $\chi^2(1) = 0.10$, $p = .75$. Similarly, in a separate model, verbal fluency ($b = -0.11$, $SE_b = 0.07$, $p = .12$) did not significantly contribute to the model, participant $SD = 1.33$, $\chi^2(1) = 2.51$, $p = .11$, nor did either of its interactions, χ^2s$(1) < 0.19$, ps $> .66$. In terms of working memory, updating ($b = -0.51$, $SE_b = 2.44$, $p = .83$) did not improve the fit of the model, participant $SD = 1.80$, $\chi^2(1) = 0.05$, $p = .83$, nor did either of its interactions, χ^2s$(1) < 2.47$, ps $> .12$.

For working memory capacity, the best fitting model included a main effect ($b = 1.12$, $SE_b = 0.46$, $p = .02$) and an interaction with age ($b = -0.01$, $SE_b = 0.01$, $p = .03$), participant $SD = 1.37$, $\chi^2(1) = 5.13$, $p = .02$. Age moderated the relationship between working memory capacity and OGM. When plotted, the model illustrated that the direction of the relationship was different for younger (>1 *SD* below *M*) compared to older (>1 *SD* above *M*) children. In older children, a negative relationship was observed between working memory capacity and OGM. However, in younger children, a positive relationship was observed. In order to assess if greater working memory capacity in younger children simply led to increased retrieval of all types of memories, the analyses were rerun on number of specific memories. Although adding working memory capacity did improve the fit of the model, $\chi^2(1) = 14.00$, $p < .001$, it was not related to specific memories ($b = -0.17$, $SE_b = 0.11$, $p = .13$). Adding an interaction with age did not improve the fit of the model, $\chi^2(1) = 1.37$, $p = .24$. This indicates that greater

working memory capacity was not simply related to all retrieval, but specifically to OGM.

In addition, I assessed interactions between the subcomponents of executive control. When added to separate models each containing the baseline of random effects, age, and time, with main effects in the second step, the interactions between inhibition and updating, and working memory capacity and updating did not significantly contribute to the fit of the models (*b*s < 0.17, SE_b*s* = 0.03 to 0.11, *p*s > .05). The interaction between inhibition and working memory capacity did, however, contribute uniquely to the fit of the model (*b* = 0.06, SE_b = 0.03, *p* = .02), participant *SD* = 1.45, $\chi^2(1) = 5.90$, *p* = .02. When plotted, the model illustrated that at low levels of inhibition (>1 *SD* below *M*), working memory capacity did not influence OGM. At high levels of inhibition (>1 *SD* above *M*), a positive relationship was observed between working memory capacity and OGM.[7]

In summary, rumination, verbal fluency, updating, and inhibition were not significantly associated with OGM. Working memory capacity was related to OGM, an effect which was moderated by age. Working memory capacity also interacted with inhibition on OGM.

Interactions between the separate mechanisms. I also assessed the interaction between components of the separate mechanisms in their relationship with OGM, as suggested by Sumner (2012). I created a series of models predicting OGM. Each initial model was formed by a random effect of participant with age and time as fixed effects. Main effects were added in the next step, followed by interactions. No evidence of significant interactions was found for rumination and working memory capacity (*b* = 0.06, SE_b = 0.05, *p* = .20), or rumination and updating (*b* = 0.15, SE_b = 0.75, *p* = .84), (χ^2s (1) < 2.0, *p*s > .20). Again, small coefficients (particularly in

comparison to the relevant error) indicate that the effects are of negligible size. The interaction between inhibition and rumination (b = 0.06, SE_b = 0.03, p = .06) was approaching significance, $\chi^2(1) = 3.70$, p = .05. To summarise, I found no strong evidence for significant interactions between the mechanisms in their effect on OGM.

Does Time Moderate the Relationship Between OGM and Psychopathology?

I next examined the moderating role of time since exposure on psychological symptoms. I hypothesised that time since trauma exposure would moderate the relationship between number of overgeneral memories and symptoms of both PTSD and depression.

PTSD symptoms. I created a mixed-effects model with PTSD symptoms (as measured by the CPSS) as the outcome. The initial model was formed by participant (as a random effect) and age as a fixed effect. Next I added time and OGM, which did not significantly improve the fit of the model, $\chi^2(3) = 7.05$, p = .07. That is, I had no evidence that time and OGM significantly contributed to symptoms. However, adding the interaction term did significantly improve the fit of the model, participant SD = 1.11, $\chi^2(2) = 9.07$, p = .01. The coefficients demonstrate that although the OGM-PTSD relationship at T2 did not significantly differ from that at T1 (b = 0.02, SE_b = 0.98, p = .98), the relationship at T3 was significantly different from T1 (b = -2.59, SE_b = 1.10, p = .02). The interaction is depicted in Figure 1. It shows a positive, though non significant relationship was observed between OGM and PTSD symptoms at both T1 and T2, whereas a significant negative relationship was observed at T3. At T3, the reporting of an additional overgeneral memory was related to a reduction of 2.59 points on the CPSS. As such, time since trauma exposure moderated the relationship between PTSD symptoms and OGM.[8]

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[7] This interaction did not significantly improve the model of best fit for number of specific memories, $\chi^2(1) = 0.01$, p = .93.

[8] This interaction was not found to be significant for number of specific memories, $\chi^2(1) = 1.82$, p = .18.

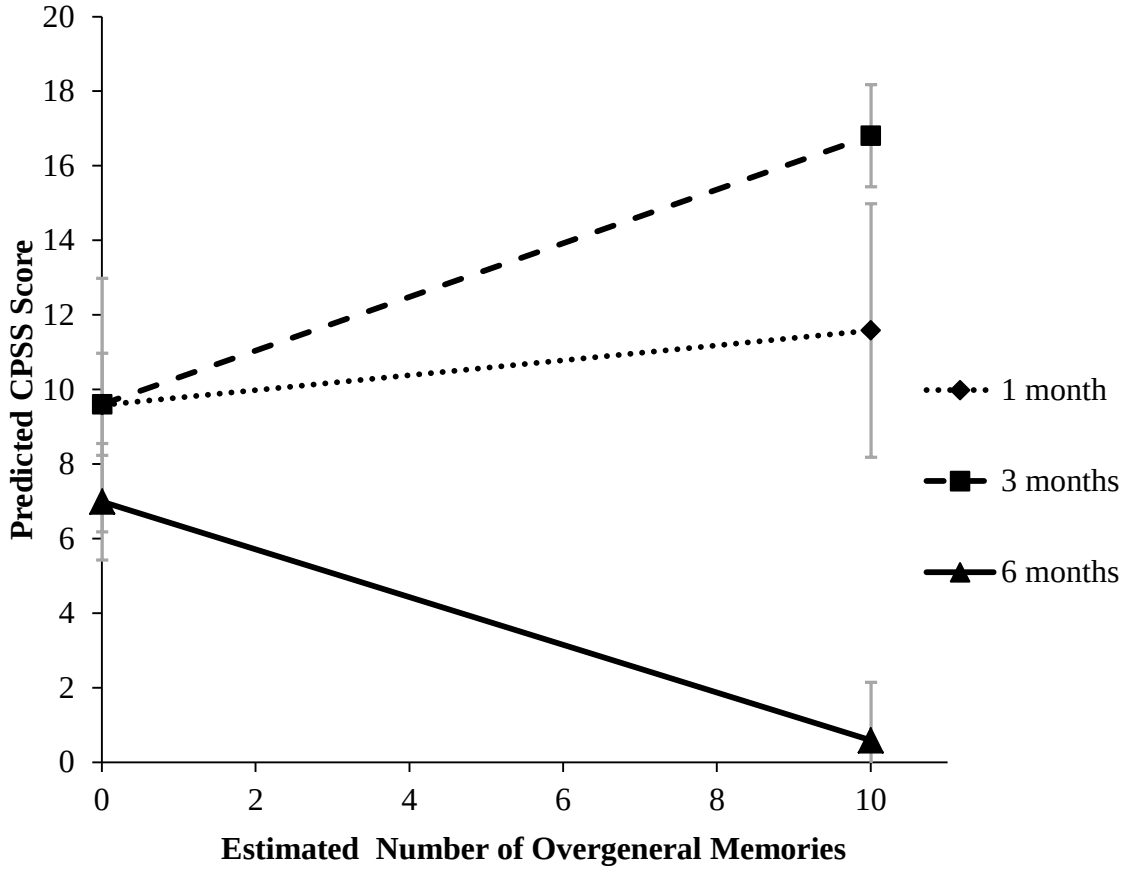

Figure 1. Plotted mixed model for interaction between time since trauma exposure and estimated number of overgeneral memories on predicted PTSD symptoms, represented by score on the Child Posttraumatic Stress Scale (CPSS). Error bars indicate standard error. Random effect of participant was included in the model, *SD* = 1.11.

Depression symptoms. The above analyses were repeated with depression symptoms as the dependent variable. The initial model was formed by participant as a random effect (*SD* = 7.03), and age as a fixed effect. The next step added time and OGM which significantly improved the fit of the model, $\chi^2(2) = 17.11$, $p < .001$. This model indicated a significant coefficient for time (T2: $b = 0.02$, $SE_b = 0.06$, $p = .98$; T3: $b = -2.59$, $SE_b = -2.55$, $p = .02$), although OGM was not significantly related to symptoms ($b = 0.08$, $SE_b = 0.46$, $p = .53$). Adding the interaction term did not significantly improve the fit of the model, $\chi^2(1) = 1.55$, $p = .21$. Hence, no evidence was

found for a significant relationship between depression symptoms and OGM at any of the time points.

Discussion

This study is the first to examine the trajectory of OGM immediately after childhood trauma exposure and how this relates to psychopathology. I also provided the first examination of multiple mechanisms of the CaR-FA-X model in an acute trauma-exposed sample. Results suggested that the depression-OGM relationship and the PTSD-OGM relationship may show different patterns following childhood trauma. Further, I demonstrated that the relationship between OGM and PTSD symptoms changed over time. Intriguingly, a significant negative relationship at T3 suggested that OGM was adaptive six months after trauma. Finally, no evidence was found for the capture and rumination or executive control components of the CaR-FA-X model in trauma-exposed children.

My findings contribute to understanding of how OGM relates to childhood psychopathology. There was no evidence of a relationship between OGM and symptoms of depression at any time point. This is in direct contrast to previous studies that have found that OGM predicts depression symptoms in adolescents. The key difference between this study and previous studies is that I used a trauma-exposed sample, whereas studies demonstrating that OGM predicted depression symptoms did not investigate the role of trauma. Indeed, my findings are consistent with previous research demonstrating that a (positive) relationship between OGM and depression symptoms is only apparent for those without abuse histories (Valentino et al., 2012). The relationship between childhood trauma, OGM and psychopathology is likely to be complex. This study is the first to examine change in both psychological symptoms and OGM on multiple occasions over six months following the experience of trauma. I

have therefore provided unique information about how OGM relates to psychological symptoms immediately following childhood trauma.

I have provided support for Williams' (1996) theory of how OGM relates to emotional disturbance following childhood trauma. Williams (1996) proposed that OGM would regulate negative affect produced by a trauma memory. As such, OGM would be functional initially following trauma and only become maladaptive at a later point, once the style became chronic in use. The lack of relationship between OGM and depression symptoms in my sample is consistent with this view. That is, OGM may have aided my participants to regulate negative affect produced by their trauma memory, and would therefore not be related to depression. Further, my findings indicated that OGM was actually adaptive for PTSD symptoms at six months post-trauma. Hence, the relationship between OGM and psychological symptoms may be different initially following trauma (compared to those who had not been exposed, or those who had been exposed years ago) as proposed by Williams (1996). This will be an intriguing area for future research as results indicated that OGM will not be associated with negative outcome immediately post-trauma. Theoretical understanding of OGM will benefit from exploring whether the relationship between OGM and symptoms changes with time since trauma exposure. In particular, it will be important to determine whether OGM becomes associated with depression symptoms if maintained over the longer term. In addition to time since exposure, future research will also need to examine whether other factors may need to be present, such as the experience of another trauma or stress (i.e., accumulative effects) for OGM to become maladaptive.

My findings also have implications for the mechanisms underlying OGM in young people. No support was found for either the capture and rumination or executive control mechanisms of the CaR-FA-X model in this sample. This may indicate that these mechanisms do not operate in trauma-exposed children. However, measurement

differences between this and previous studies may also contribute to discrepant findings. The only study to demonstrate that inhibition was related to childhood OGM used a personality (i.e., ability to keep a secret) measure of inhibition (Raes et al., 2010). Hence, my use of a more objective measure of inhibition may influence results. In line with this, the non significant effect of inhibition replicates that of Valentino et al. (2012), who also used an objective, cognitive measure of inhibition. In terms of rumination, the only previous study to report an effect of rumination on OGM in young people involved an experimental manipulation, which influenced state rumination (Park et al., 2004), whereas I assessed trait rumination. As such, measurement differences may contribute to discrepant findings.

Alternatively, functional avoidance, which I did not assess, may drive OGM initially following childhood trauma (as originally theorised), with the other mechanisms becoming influential once the child has mastered those cognitive skills, or once OGM becomes maladaptive. First, given that rumination and executive control are not fully developed in childhood (e.g., Gathercole, 2004; Hampel & Petermann, 2005) it is possible that OGM is driven by functional avoidance during childhood and that the other mechanisms become influential when the individual has further developed those skills (i.e., in late adolescence or adulthood). Indeed, my findings lend support to this proposition. Results demonstrated that age moderated the effect of working memory capacity on OGM, such that greater working memory capacity was related to a reduction in overgeneral memories in older children (as proposed by the CaR-FA-X model), while in younger children, having greater working memory capacity was related to reporting a higher number of overgeneral memories.

A plausible explanation for this finding is that younger children with well-developed working memory capacity may be more efficient at retrieving any type of memory. However, this is discounted by the fact that the effect was not evident when

number of specific memories was the dependent variable, indicating that the interaction only produced an increase in OGM, not all types of memories. It is possible that those younger children who develop high working memory capacity have not yet developed the control skills needed to use the expanded capacity to greatest effect. That is, although working memory capacity is higher, it may be used for different tasks than the controlled memory search, such as engaging in elaboration of irrelevant cues. As such, in the absence of adequate control, higher working memory capacity may actually increase potential for being distracted from the task at hand, or more simply, be used to engage in processes that interrupt the search. Although age did not moderate the effect of other mechanisms on OGM, this highlights the importance of considering developmental effects that may influence maintaining factors of OGM. This study was the first to examine whether age of the child interacted with the assessed mechanisms in predicting OGM. Future studies will need to include age interactions in analyses to accurately account for developmental effects.

Second, support for the CaR-FA-X model in explaining OGM in mood disordered adults (see Sumner, 2012) suggests that the model is able to account for the end-point of maladaptive development, but may not offer an adequate description of the intermediate development of OGM towards this point. As OGM was negatively related to PTSD symptoms in this sample, participants may have been actively trying to be overgeneral. If this were the case, errors in their desired retrieval style would relate to increased specific memories, as I observed. For example, if the child was trying to be overgeneral, poor inhibition would relate to an unwanted specific memory coming to mind. In line with this, the results indicated that executive control (i.e., greater working memory capacity and better ability to inhibit irrelevant information) was positively related to OGM, not negatively related as the CaR-FA-X model predicts. As such, the CaR-FA-X mechanisms may best describe OGM once it has become maladaptive, or in

those experiencing significant symptoms of mood disturbance. This would be in line with support for the mechanisms in mood disordered samples (e.g., Park et al., 2004). Indeed, Williams et al. (2007) stated that psychological symptoms may increase activation of the mechanisms. For example, emotional disturbance may reduce executive control. Thus, I suggest that the CaR-FA-X model may not adequately account for ongoing OGM development.

I make several observations in conclusion. Considering the prevalence of mental health difficulties in young people, identifying protective or vulnerability factors is important in improving intervention and preventative programs. Clinical research with adolescents has demonstrated that reducing OGM can lead to a reduction in depression symptoms (Neshat-Doost et al., 2013). However, my findings highlight the need to establish timelines underlying the relationship between psychopathology and OGM before interventions are used in clinical practice as there may be a time that OGM is in fact adaptive, at least in trauma-exposed young people. Theoretical underpinnings of the development of OGM and the mechanisms through which it occurs in children are also in need of refinement, particularly as the role of OGM in psychopathology appears to change over time. Study 2 addressed these needs by exploring the relationship between OGM and psychopathology years after childhood trauma exposure, along with replicating this study's findings on the cognitive mechanisms underlying OGM.

CHAPTER 4

Study 2

This study built upon Study 1 by providing a more thorough examination of the CaR-FA-X mechanisms. I expanded my previous investigation of the executive control, and capture and rumination mechanisms by also assessing the third proposed mechanism, functional avoidance. Potential interactions between all three mechanisms, depression and trauma history in predicting OGM were also assessed. Simultaneous examination of aspects of each mechanism, namely rumination, inhibition and cognitive avoidance, along with depression and trauma exposure created a unique opportunity to untangle the effects of these factors. Two assessments were completed eight months apart to determine the direction of the relationship between OGM and each of these factors. A secondary aim of this study was to advance understanding of how OGM related to symptoms of depression and PTSD a number of years after childhood trauma had occurred. There were both cross-sectional and prospective elements to the study design. To examine the first aim, I completed a prospective examination of whether the mechanisms predicted future OGM, and whether OGM predicted future symptoms. However, to explore the second aim, this study also included a cross-sectional assessment of how time since trauma exposure impacted the relationship between OGM and symptoms.

An adolescent sample was specifically used. Examination of psychological symptoms and mechanisms of OGM in adolescents offered two benefits. First, clinical interventions are being developed to reduce depression symptoms by increasing memory specificity in adolescents (Neshat-Doost et al., 2013). As such, the present study provided further theoretical understanding of the operation of OGM in the population these interventions target. Second, this study was interested in how OGM related to symptoms *years* after childhood trauma exposure. As such, adolescents offer

a sample that is likely to have experienced childhood trauma years previously. Variation in the number of years since exposure would allow a preliminary, cross-sectional examination of whether the relationship might be different between those exposed to trauma (for example) one, three, or six years previously. Any differences would have implications for whether OGM may be adaptive if used following recent trauma, but maladaptive if used years after exposure.

This study aimed to advance our understanding of the CaR-FA-X mechanisms in three particular ways. First, this study addressed a noticeable gap in the field by providing the first examination of the functional avoidance mechanism in young people. In the context of autobiographical memory theory, functional avoidance is considered a type of cognitive avoidance (Williams et al., 2007). No previous study has examined whether OGM is predicted by cognitive avoidance, or any other measure of the functional avoidance construct. As the functional avoidance mechanism is proposed to underlie the childhood onset of OGM, it is important to assess the mechanism in young people. Due to a scarcity of research of the functional avoidance mechanism, it has not been determined whether the three CaR-FA-X mechanisms do interact with one another, as predicted by Williams et al. (2007). Interactions between different executive control processes have been assessed in young samples, but not supported (e.g., Study 1 of this thesis; Valentino et al., 2012). Research with adult samples has only recently begun to explore interactions between different mechanisms, and strong evidence for interactions has not been produced (see Sumner, 2012, for review).

The model does not make predictions regarding how the mechanisms will interact. However, if cognitive avoidance, poor inhibition, and rumination are each unhelpful in retrieving specific memories, it should follow that someone who frequently engages in all of these processes will perform more poorly when retrieving specific memories than someone who only engages in one of these processes. That is, high

cognitive avoidance and high rumination would be expected to combine to increase the number of retrieval errors, and therefore OGM. Similarly, low inhibition and high rumination should also interact to produce OGM. In addition to exploring such interactions, the prospective study design allowed me to test for directionality between the mechanisms and OGM. This is particularly important as previous examination of the mechanisms has been primarily correlational (e.g., de Decker et al., 2003; Valentino et al., 2012). Accordingly, the present study was in a position to make an important contribution to the field by providing the first simultaneous examination of cognitive avoidance, rumination, and inhibition.

Second, I explored whether trauma history interacted with the mechanisms of interest in predicting OGM. An interaction with trauma history is fundamental in the description of how functional avoidance operates. Williams et al. (2007) stated a child will engage in functional avoidance following trauma in order to regulate negative affect brought about by their trauma memory. This avoidance will later generalise to all memory searches. Thus, functional avoidance is proposed to mediate the relationship between trauma exposure and OGM, such that children exposed to trauma will demonstrate greater cognitive avoidance, and thus OGM. There are also research findings to suggest that trauma exposure may moderate the other mechanisms. Inhibition has received support in predicting OGM in a normative sample (Raes et al., 2010), but not in trauma-exposed samples (Study 1 of this thesis; Valentino et al., 2012). Similarly, rumination has been found to predict OGM in a clinical sample (Park et al., 2004), but not a trauma-exposed sample (Study 1 of this thesis).

Initial theory of OGM began with the idea that childhood trauma exposure would lead to OGM due to increased functional avoidance (Williams, 1996). The expanded CaR-FA-X model introduced the capture and rumination, and executive control mechanisms to account for OGM in those without childhood trauma histories.

Thus, it possible that the functional avoidance mechanism initiates OGM immediately following childhood trauma, and is later maintained by the other two processes. Alternatively, functional avoidance may be more integral in trauma-exposed samples while the capture and rumination, and executive control mechanisms may be more integral in non trauma-exposed samples. Indeed, previous authors have proposed that the CaR-FA-X mechanisms may be more important in one context compared to another (Crane, Barnhofer, Visser, Nightingale, & Williams, 2007; Sumner, 2012). This may contribute to inconsistent support for the mechanisms between samples. I explored this idea by examining whether trauma history interacted with any of the mechanisms in predicting OGM.

This study also explored whether the CaR-FA-X mechanisms interact with depression to predict OGM. Current evidence for the capture and rumination, and executive control mechanisms has largely occurred in the context of depression. In terms of capture and rumination, the one study demonstrating that rumination predicts OGM used a sample of adolescents with Major Depressive Disorder (MDD; Park et al., 2004). The executive control mechanism has received less support. Many studies have reported no evidence for working memory capacity and updating in predicting OGM (de Decker et al., 2003; Kuyken et al., 2006; Nixon, Ball, et al., 2013; Valentino et al., 2012). Evidence for inhibition has been less consistent. Although Study 1 and Valentino et al. (2012) demonstrated no evidence for a main effect of inhibition on OGM, inhibition has been found to interact with depression in predicting OGM (Raes et al., 2010). Thus, some support has been found for rumination and inhibition in interaction with depression. Williams et al. (2007) stated that symptoms of depression may increase activation of the mechanisms. For example, emotional disturbance may reduce inhibition, increase an individual's tendency to ruminate, or increase cognitive avoidance, thereby increasing activation of the mechanisms. I aimed to investigate these

effects further by determining if cognitive avoidance, inhibition, and rumination interacted with depression. In sum, this study advanced understanding of the CaR-FA-X model by exploring aspects of all three mechanisms and their potential moderators in predicting adolescent OGM.

In assessing trauma history, this study was in a unique position to provide a preliminary exploration of whether the number of years since trauma exposure may impact the relationship between OGM and psychopathology. Williams (1996) proposed that time since trauma exposure would moderate the relationship between OGM and psychological symptoms. OGM would be an adaptive mood regulation strategy when engaged immediately following trauma but become associated with psychopathology if maintained over the long term.[9] Inconsistent reports regarding whether OGM is unrelated to symptoms of PTSD in young people (Brennen et al., 2010; de Decker et al., 2003; Vrielynck et al., 2007), or related to lower symptoms of PTSD (Kuyken et al., 2006; Nixon, Ball, et al., 2013), or related to increased symptoms of PTSD (Stokes et al., 2004), may reflect that the relationship changes with time since trauma exposure. Each of these studies may have measured the relationship at different points of the OGM trajectory. Despite representing a key prediction of OGM theory, this proposal has not been previously assessed over years since exposure. Increased understanding of the trajectory of OGM may indicate the optimal timing of clinical intervention into OGM. If OGM begins as adaptive, and changes to become maladaptive, an intervention would be optimally timed just before the change occurs. A cross-sectional examination of whether time since trauma exposure impacts the relationship between OGM and symptoms is a cost effective way to determine if further longitudinal research is

[9] In studies of adult samples with childhood trauma histories (e.g., Crane & Duggan, 2009), current psychological symptoms (e.g., depression, posttraumatic stress) may be anchored to an adulthood event (e.g., relationship breakdown, an assault during adulthood), rather than the noted childhood experience. However, if OGM is a cognitive vulnerability, OGM resulting from adverse childhood events would be expected to influence current symptoms.

warranted. Thus, while the key aims of this study were achieved through prospective examination of OGM, I also completed a secondary, cross-sectional examination of whether the relationship between OGM and symptoms differed depending on how long ago the individual was exposed to trauma.

Finally, this study aimed to close a gap in prospective research of OGM and depression symptoms in adolescents. Chapter 1 emphasised that previous examination of the causal relationship between OGM and depression is very limited. Understanding the direction of the relationship is of clinical importance by indicating whether OGM may offer a potential intervention point to reduce recurrent depression symptoms, or even present a target for preventative programs. Although a number of longitudinal studies have indicated that OGM is able to predict depression symptoms up to a year later, the vast majority have used samples with elevated depression symptoms at baseline (e.g., Hipwell et al., 2011; Sumner, Griffith, Mineka, et al., 2011). Although these studies indicate that OGM is able to predict recurrent symptoms, they do not clarify whether OGM is a pre-existing risk factor for depression. That is, as samples were currently depressed, the direction of the relationship is unable to be determined.

Only one previous study (Rawal & Rice, 2012) has examined the ability of OGM to predict future symptoms in those who did not demonstrate elevated depression symptoms at baseline. The authors reported that OGM to negative cues predicted depression symptoms one year later in female participants. That is, OGM was a unique predictor of depression symptoms. Although this sample was not currently depressed, they were at increased familial risk of depression due to having a parent with the disorder. As such, the finding may be limited to those already at increased risk of developing the disorder. Further prospective research is needed with regular community samples in order to determine whether OGM offers a pre-existing cognitive vulnerability in unselected adolescents that can be targeted to prevent depression. As I

recruited a school-based sample, not all the current participants had a trauma history. I was therefore able to examine how OGM predicted future depression symptoms (and vice versa) in a non-clinical, community sample. Thus, this study offered important implications for whether OGM may be a vulnerability factor for depression in those not already at increased risk.

In summary, this study had three key aims. The primary aim was to determine if the CaR-FA-X mechanisms accounted for OGM in young people. In particular, I assessed whether the mechanisms predicted OGM uniquely, in interaction with one another, or in interaction with trauma exposure or depression. I completed two assessments eight months apart to assess whether any of these effects predicted later OGM. The secondary aim was to provide a preliminary exploration of whether the relationship between OGM and psychological symptoms may be different depending on time since trauma exposure. This cross-sectional examination is a cost effective method to determine whether further longitudinal examination is warranted. Finally, I aimed to clarify the direction of the relationship between OGM and depression in a non trauma-exposed, non-clinical sample. This was investigated by assessing whether OGM at baseline predicted later depression symptoms.

I hypothesised that inhibition, rumination, and cognitive avoidance would each account for unique variance in OGM. In addition, I expected that inhibition and rumination, and cognitive avoidance and rumination, would interact in predicting OGM. I also predicted that trauma exposure would interact with cognitive avoidance, rumination, and inhibition. Specifically, the effects of rumination and inhibition would be moderated by trauma exposure. Both processes were expected to have a stronger effect on OGM for those without a trauma history, compared to those with a trauma history. Similarly, cognitive avoidance was hypothesised to mediate the relationship between trauma exposure and OGM. I also predicted that time since trauma exposure

would moderate the relationship between both OGM and depression symptoms, and OGM and PTSD symptoms. A negative relationship was expected between OGM and symptoms when trauma was relatively recent, and a positive relationship was expected for those who had experienced trauma many years ago. I did not make specific predictions regarding when the relationship would change, as Williams (1996) does not outline a specific timeline. Finally, it was hypothesised that OGM at baseline would predict depression symptoms at follow-up, controlling for baseline symptoms

Method

Participants

Two hundred and one students between the ages of 12 and 17 years (M = 14.18 years, SD = 1.58 years) were recruited from three metropolitan public high schools.[10] The sample was almost exclusively Caucasian (95.9%) which reflects the demographics of the schools' geographic locations (Australian Bureau of Statistics, 2011); other ethnic backgrounds included Asian (2%), Pacific Islander (1%) and African (1%). Two students withdrew during data collection. Data were also excluded from one student who was above the age range and two students who did not comply with the research protocol. Hence, I analysed data from 196 (104 female) students. Students with existing psychological diagnoses (e.g., anxiety, depression) were included. Before participation, informed consent was gained from legal guardians, along with consent from the student. All participants received an ice cream or pen for their involvement.

Participants completed two sessions: Time 1 (T1) at the beginning of the Australian academic year (February or March 2012) and Time 2 (T2) at the end of the academic year (October or November 2012). Mean number of days between assessments was 230.61 days (SD = 40.14). Attrition between T1 and T2 was 21.40%.

[10] I did not recruit a sample that excluded those who were at familial risk. Rather, I sought to recruit a sample that was representative of the general population to make findings generalizable to other school-attending adolescents. I therefore acknowledge that some of the participants may have been at familial risk, however, the overall sample did not represent a high-risk group.

Those who did not complete T2 demonstrated significantly more symptoms of depression at T1 (*M* = 57.92, *SD* = 13.75, [53.46, 62.38]) compared to those who completed T2 (*M* = 50.70, *SD* = 12.04, [48.74, 52.60]), *p* = .001, *d* = 0.58. They also demonstrated significantly more PTSD symptoms at T1 (did not complete T2 *M* = 16.45, *SD* = 11.34, [12.72, 20.17]; did complete T2 *M* = 9.84, *SD* = 9.51, [8.28, 11.40]), *p* = .001, *d* = 0.67. However, they did not significantly differ in OGM at T1 (did not complete T2 *M* = 5.98, *SD* = 2.35, [5.25, 6.71]; did complete T2 *M* = 5.78, *SD* = 2.27, [5.42, 6.14]), *p* = .62, *d* = 0.09.

The majority of the hypotheses were interactions, and a priori estimation of power for interactions is difficult and not recommended (Cohen, 1988). Therefore estimation of required sample size was based on that needed to detect a small effect. Cohen's (1988) conventions state that to detect a small effect with power of .80, 175 participants are required. Hence, with the current sample size I can be confident this study was adequately powered.

Measures

Autobiographical Memory Test (AMT): Written form. The written form of the AMT has been used with adolescents in this age group (e.g., Kuyken & Dalgleish, 2011; Smets, Griffith, Wessel, Walschaerts, & Raes, 2013). Students were presented with ten cue words and asked to provide a specific memory for each. Cues were printed in a booklet with lines underneath for their response. Positive- and negative-valence words were alternated. Different cue words were used at T1 and T2, with lists balanced for imagery and familiarity (full word lists are presented in Appendix B). Instructions were taken from the written AMT employed by Kuyken and Dalgleish (2011) and written on top of the page. In order to minimise omissions, a problem encountered by Kuyken and Dalgleish, instructions were read to the participants, the task requirements were clarified, and each cue was read aloud by the researcher. Participants were given

one minute to record their response for each word. Responses were coded as specific, overgeneral (extended or categoric), semantic associate, omission, or violation. Violations were defined as repeating a memory previously reported, or if what was written was unable to be coded (e.g., one child wrote a story about a friend). Interrater agreement on scoring of 25% of the AMTs was adequate ($\kappa = .85$). I used number of specific memories as the measure of OGM.

Walk, Don't Walk task. Inhibition errors were assessed using the Walk, Don't Walk subtest of the Test of Everyday Attention for Children (Manly, Robertson, Anderson & Nimmo-Smith, 1999). Psychometric properties of this task are presented in Study 1. The test is standardised for individual or group administration (Manly et al., 1999). Unlike Study 1, in this study the measure was administered in groups. To ensure comprehension of the task, the researcher demonstrated an example on a whiteboard in front of the class. As norms were only available up until the age of 15, data were only included for children under this age. This measure was chosen as it is an objective, standardised measure of inhibition errors, and Williams et al. (2007) stated that errors in inhibition will produce OGM.

Child Posttraumatic Stress Scale (CPSS; Foa et al., 2001). Psychometric properties of this task are presented in Study 1. Children were asked to rate their experience of each symptom in relation to their most frightening or stressful life experience. In my sample, this included sexual or physical assault, domestic violence, death or life threatening injury of a family member, accidental injury, and childhood experiences such as getting lost, seeing a scary movie, or going on a scary amusement park ride.

Spence Children's Anxiety Scale - Child Report (Spence, 1998). The general anxiety subscale was administered to measure symptoms of anxiety. As anxiety was unrelated to number of specific memories ($r = .06$, $n = 161$, $p = .45$), and number of

overgeneral memories (r = .14, n = 161, p = .08), the measure was not used in any further analyses.

The Child Depression Inventory – Short Form (CDI-S; Kovacs, 1992). As in Study 1, the CDI-S was used to assess depression symptoms. In line with recommendations made by Kovacs (1992), participants who obtained a normed score (T score) of 65 or higher were classified as having clinically significant symptoms.

The Children's Response Style Scale (CRSS; Ziegert & Kistner, 2002). As in Study 1, I used the CRSS to measure rumination.

The Cognitive Avoidance Questionnaire (CAQ; Sexton & Dugas, 2008). The CAQ was used to tap functional avoidance. Although this measure was designed for adults, I used it because a valid, reliable measure of cognitive avoidance has not been developed for young people. The measure has adequate internal consistency (α = .95) in adult samples (Sexton & Dugas, 2008), and has been used with those in their late teens (i.e., 17-19 years old; McEvoy, Mahoney, & Moulds, 2010). Nevertheless, I slightly altered wording on some items to make it easier for younger participants to read. For example, 'I think about trivial details so as not to think about important subjects that worry me' was changed to 'I think about small things so that I don't think about important things that worry me'. Scores were calculated for the total (out of 125) and subscales (out of 25), with higher scores indicating more cognitive avoidance. Although some words were changed, internal consistency was adequate in this sample. Internal consistency was comparable with the original CAQ for the total score (α = .96), and all subscales; thought substitution (α = .84), transformation of images into thoughts (α = .81), distraction (α = .86), avoidance of threatening stimuli (α = .87) and thought suppression (α = .88).

Trauma history. A life events checklist was used to gain parent report of trauma history. The checklist consisted of the 15 potentially traumatic experiences from

the life events checklist in the Clinician Administered PTSD Scale - Child Version (Nader et al., 1996). I collected data on frequency of occurrence and age at each event. In this sample, endorsed events were sexual assault, physical abuse, and war exposure. Ethics committee constraints required us to obtain this information via parental (not student) report. This checklist was changed from Study 1 as it directly asked about sexual assault, and provided further information on the age at experienced events. Trauma history was used dichotomously in analyses (i.e., trauma-exposed or non-exposed). Students were classed as having a trauma history if their parent reported they had experienced sexual assault/abuse, physical assault/abuse, or war exposure. Time since trauma exposure was recorded at each assessment as the number of months since trauma exposure.

Procedure

University and education department ethics committees approved data collection. Parental consent and student assent were gained prior to data collection. The same procedure was followed at T1 and T2. Parents completed the Life Events Checklist prior to each session. Data collection was completed in group format at the school. Students completed the AMT and inhibition task as a group. Following this, they worked through the CPSS, CDI-S, CRSS, CAQ and anxiety scale at their own pace. A researcher was available to answer questions.

Results

I used linear mixed-effects modelling to test my hypotheses. Except where noted, each model had participant, assessment (i.e., T1, T2), and school as random effects, and age as a fixed effect entered in the first step. Random effects are reported for the model of best fit. Individual predictors (i.e., main effects) were then entered in the second step, followed by interaction terms in the third step. Number of specific

memories was used when examining AMT performance.[11] As the CaR-FA-X processes are proposed to impair retrieval of specific memories, they may be better assessed by examining number of specific memories, rather than the erroneous production of overgeneral memories. As such, OGM is represented by a low number of specific memories. All models were repeated with number of overgeneral memories and are reported when different from models with specific memories as the dependant variable.

Descriptive statistics for AMT performance, psychological symptoms, and trauma characteristics are presented in Table 3. At T1, 55 participants (25.5%) reached the clinical cut-off of 16 on the CPSS (i.e., PTSD symptoms),[12] and 31 (15.8%) reached the clinical cut-off of 65 on the CDI-S (i.e., depression symptoms). For 28 participants their parents reported that they had experienced a traumatic event (i.e., sexual assault, physical assault, or war exposure). One parent reported that their child had been sexually assaulted, but did not report the age of their child at exposure. Thus, when examining age at exposure and time since exposure, data was only available for 27 participants. Trauma history was only available for 160 participants (82%), due to this questionnaire not being returned by a caregiver.

I completed a mixed-effects model to examine whether memory performance differed between assessments. School and participant were entered as random effects, and age and assessment were entered as fixed effects. Assessment did not significantly predict mean number of specific memories (i.e., no difference was observed between T1

[11] Although I analysed number of overgeneral memories in Study 1, the number of overgeneral memories reported in this sample was quite low, and there was more variation in reporting of specific memories (see Table 3 for descriptive statistics). This is not uncommon when using the AMT in a non-clinical setting, as instructions explicitly ask participants to retrieve a specific memory (for discussion see Debeer, Hermans, & Raes, 2009). As such, it is common to use number of specific memories in analysis (e.g., Sumner, Griffith, Mineka, et al., 2011). I therefore used number of specific memories as our dependent variable, although all models were repeated for number of overgeneral memories.

[12] The discrepancy between the percentage of participants who reached the clinical cut-off on the CPSS and those with a parent-reported trauma history may be due to three reasons. First, it may reflect elevated general anxiety symptoms in those without trauma histories. Indeed, the CPSS index event described by some participants who reached the clinical cut-off did not constitute a Criterion A stressor. Second, it may be due to failure of some of these participants to return the parent forms. That is, they may have formed part of the sample for which I did not have parent-report trauma history. Finally, the discrepancy may reflect inaccurate/false report of trauma history by the parent.

and T2), participant $SD = 1.48$, school $SD < 0.01$, $\chi^2(1) = 0.10$, $p = .75$. Similarly, assessment was not seen to predict overgeneral memories when the model was repeated, participant $SD = 1.16$, school $SD = 0.22$, $\chi^2(1) = 0.04$, $p = .84$. This is consistent with research suggesting OGM is a stable cognitive factor (Heron et al., 2012).

Table 3

Summary of Means, Standard Deviations, and Range of Scores for AMT Performance, Cognitive Variables, Psychological Symptoms and Trauma Characteristics by Assessment

	Time 1				Time 2			
Variable	*n*	*M* (*SD*)	95% CI	Range	*n*	*M* (*SD*)	95% CI	Range
Specific Memories	196	5.79 (2.42)	[5.46, 6.12]	0-10	153	5.78 (2.56)	[5.38, 6.19]	0-10
Overgeneral Memories	196	2.41 (1.85)	[2.15, 2.67]	0-9	153	2.44 (2.14)	[2.10, 2.78]	0-9
Inhibition	160	7.31 (3.90)	[6.70, 7.92]	0-19	129	9.11 (3.61)	[8.48, 9.74]	1-15
Rumination	191	4.18 (2.55)	[3.82, 4.54]	0-10	148	4.32 (2.59)	[3.90, 4.74]	1-10
CAQ	186	58.91 (21.63)	[55.78, 62.04]	25-110	143	55.63 (22.29)	[51.95, 59.32]	25-117
CDI-S	188	51.93 (13.24)	[50.03, 53.87]	39-90	152	52.83 (14.32)	[50.53, 55.12]	39-97
CPSS	184	11.15 (10.25)	[9.66, 12.64]	0-48	150	10.46 (10.54)	[8.76, 12.16]	0-44
Months since trauma exposure	27	85.88 (52.45)	[65.14, 106.64]	12-187	27	93.89 (52.45)	[73.14, 114.64]	20-195
Age in years at exposure	27	7.52 (4.43)	[5.77, 9.27]	1-14	-	-	-	-

Note. CI = Confidence interval; CAQ = Cognitive Avoidance Questionnaire; CDI-S = Child Depression Inventory- Short Form; CPSS = Child Posttraumatic Stress Scale. Number of specific memories and overgeneral memories are reported.

Do Rumination, Inhibition, and Cognitive Avoidance Predict Specificity?

First I examined whether rumination, inhibition, and cognitive avoidance predicted specificity. I hypothesised that each process would uniquely predict specificity. Further, I hypothesised interactions between inhibition and rumination, and cognitive avoidance and rumination. To test these hypotheses, I created a mixed-effects model with number of specific memories as the outcome. The first step included assessment, participant and school as random effects, and age as a fixed effect, $\chi^2(1) = 15.27$, $p < .001$. Next, I added rumination, inhibition and cognitive avoidance, which significantly improved the fit of the model, assessment $SD < 0.01$, participant $SD = 1.41$, school $SD < 0.01$, $\chi^2(3) = 362.86$, $p < .001$. Contrary to expectations, specificity was not significantly predicted by rumination, $b = 0.04$, $SE_b = 0.08$, $p = .70$, inhibition, $b = 0.06$, $SE_b = 0.04$, $p = .13$, or cognitive avoidance, $b = 0.01$, $SE_b = 0.01$, $p = .70$. Adding interactions between the processes did not significantly improve the fit of the model, $\chi^2(3) = 0.06$, $p = .99$, nor did adding interactions with age, $\chi^2(3) = 2.55$, $p = .47$, or assessment, $\chi^2(9) < 0.01$, $p = .99$. The lack of interactions with assessment indicated that the CaR-FA-X processes did not predict current or future OGM. In sum, there was no evidence for unique effects of rumination, inhibition, or cognitive avoidance in predicting OGM. Further, I found no evidence for significant interactions between the processes. Thus, the CaR-FA-X model was not supported across the entire sample.

I also examined the subscales of the Cognitive Avoidance Questionnaire (CAQ) to determine whether the relationship differed for different types of cognitive avoidance. First, I examined correlations between the subscales (i.e., replacing images with thoughts, thought suppression, distraction, thought substitution, and avoidance of threatening stimuli) to ensure that they were in fact distinguishable subscales. However, correlations between the subscales were strong, $r_s = .61$ to $.75$, $n = 188$, ps $< .001$.

Given this evidence I was reluctant to use these scores as measures of distinct constructs.[13]

Next, I was interested in whether trauma exposure influenced the effect of the mechanisms on specificity. I hypothesised that trauma exposure would moderate the effect of both rumination and inhibition on specificity. To test these hypotheses, I completed a model predicting number of specific memories. The first step included assessment, participant and school as random effects and age as a fixed effect, $\chi^2(1) = 15.08$, $p < .001$. I next added rumination, inhibition and trauma exposure, assessment $SD < 0.01$, participant $SD = 1.43$, school $SD < 0.01$, $\chi^2(3) = 315.87$, $p < .001$. The interaction between rumination and trauma exposure, $b = 0.14$, $SE_b = 0.17$, $p = .40$, and inhibition and trauma exposure, $b = -0.12$, $SE_b = 0.11$, $p = .27$, were entered in the last step, $\chi^2(2) = 1.99$, $p = .37$. The non significant model indicated that trauma exposure did not interact with rumination or inhibition in predicting specificity.

The next hypothesis predicted that cognitive avoidance would mediate the relationship between trauma exposure and specificity. Trauma exposure is proposed to increase functional avoidance. Therefore, I first determined whether trauma exposure predicted score on the CAQ. If the theory were correct, I would expect that those with a trauma history would demonstrate greater cognitive avoidance than those without a trauma history. I created a mixed-effects model predicting score on the CAQ with trauma exposure entered as a dichotomous variable in the first step. Trauma exposure, $b = 7.06$, $SE_b = 3.21$, $p = .03$, contributed to the model of best fit, assessment $SD < 0.01$, participant $SD = 9.52$, school $SD = 0.01$, $\chi^2(1) = 4.84$, $p = .03$. The coefficient indicated that those with a trauma history on average scored 7.06 points higher on the CAQ (a

[13] When a mixed model was created to examine the ability of the subscale scores to predict specificity, the fitted model included significant effects of thought substitution, $b = 0.17$, $SE_b = 0.05$, $p = .001$, and transforming images to thoughts, $b = -0.15$, $SE_b = 0.05$, $p = .002$, assessment $SD < 0.01$, participant $SD = 1.53$, school $SD < 0.01$, $\chi^2(5) = 105.35$, $p < .001$. However, due to the strong correlation between these scores, the positive and negative coefficients effectively cancel each other out. Thus, the model describes no real relationship between cognitive avoidance and specificity; the coefficients are essentially a statistical artefact.

125 point scale) than those without trauma histories. This is consistent with trauma exposure increasing functional avoidance. However, I was unable to assess whether CAQ score mediated the relationship between trauma exposure and specificity as score on the CAQ was not related to number of specific memories. Thus, the prerequisites for mediation were not met. In sum, although cognitive avoidance was higher in trauma-exposed participants, there was no evidence for functional avoidance mediating the relationship between trauma exposure and specificity.

Finally, I determined whether the mechanisms interacted with depression symptoms. The first step of the model included assessment, participant and school as random effects, and age as a fixed effect, $\chi^2(1) = 15.08$, $p < .001$. Rumination, inhibition, cognitive avoidance, and depression symptoms (i.e., CDI-S score) were entered in the second step, assessment $SD < 0.01$, participant $SD = 1.39$, school $SD < 0.01$, $\chi^2(4) = 377.52$, $p < .001$. Interactions between depression and each of the mechanisms were entered in the final step, $\chi^2(3) = 1.77$, $p = .62$. The non significant model indicated that depression did not interact with any of the CaR-FA-X processes to predict specificity (inhibition $b = -0.003$, $SE_b = 0.003$, $p = .49$; rumination $b = -0.003$, $SE_b = 0.01$, $p = .73$; cognitive avoidance $b = -0.001$, $SE_b = 0.001$, $p = .77$).

Relationship with Psychological Symptoms

Time since trauma exposure. I hypothesised that time since trauma exposure would moderate the relationship between specificity and psychological symptoms. To assess this prediction, data was only used from those participants who had a trauma history ($n = 27$). Age at exposure and time since exposure were strongly correlated, $r = .95$, $n = 27$, $p < .001$. This is unsurprising; Given that all participants were teenagers, the younger a child was at exposure, the longer it will have been since the incident. As such, the two variables are too closely related to be considered separate effects. Hence,

although I examine time since trauma exposure, the effect may be attributable to age at exposure.

First I examined the effect of specificity on depression symptoms by creating a mixed model with score on the CDI-S as the outcome. Participant, school and assessment were entered as random effects. Age was entered in the first step, $\chi^2(1) = 2.71$, $p = .10$. The second step included time since the trauma and specificity, $\chi^2(2) = 1.46$, $p = .48$. The final step added an interaction between time since the trauma and specificity, assessment $SD < 0.01$, participant $SD < 0.01$, school $SD < 0.01$, $\chi^2(1) = 3.46$, $p = .06$. Contrary to expectations, neither specificity, $b = 3.09$, $SE_b = 1.68$, $p = .07$, nor an interaction between the two, $b = -0.04$, $SE_b = 0.01$, $p = .07$, significantly predicted depression symptoms**.** Although the overall model and the specificity and interaction coefficients were approaching significance, there was no evidence that OGM was significantly related to depression symptoms in trauma-exposed participants.

Next I repeated the same steps with CPSS score as the outcome to determine if specificity influenced PTSD symptoms. Participant, school and assessment were entered as random effects. Age was entered in the first step, $\chi^2(1) = 0.73$, $p = .39$. The second step included time since the trauma and specificity, $\chi^2(2) = 7.42$, $p = .02$. The final step included an interaction between time since the trauma and specificity, participant $SD = 7.90$, school $SD = 2.41$, assessment $SD < 0.01$, $\chi^2(1) = 4.75$, $p = .03$. The model indicated that specificity, $b = 2.61$, $SE_b = 1.12$, $p = .03$, and time since exposure, $b = 0.26$, $SE_b = 0.08$, $p = .002$, significantly predicted symptoms. The specificity coefficient indicated that an increase in number of specific memories related to an increase in score on the CPSS, though the effect was small. As hypothesised, specificity and time since

exposure did interact, b = -0.03, SE_b = 0.01, p = .04.[14] Time since exposure was entered as a continuous variable in the model. However, to illustrate the predicted relationship, I plotted the regression line for different lengths of time since trauma exposure (see Figure 2). That is, although Figure 2 presents different lines, these are not distinct groups. Rather, the line simply represents the predicted relationship for that period of time. Therefore the relationship between groups is not being compared. The figure is simply a visual representation of how the model predicts a different relationship at different time points.

When trauma had occurred in the 1-3 year period prior to assessment, the model predicted a negative relationship between number of specific memories and score on the CPSS. This relationship was not as strong at 4-6 years post-trauma. The model predicted that at 7-9 years after exposure, specificity did not influence CPSS score. However, over 10 years after exposure, a low number of specific memories was associated with higher score on the CPSS. That is, OGM appeared protective against symptoms for 1-3 years post-trauma, however, the nature of the relationship was different as time since exposure increased, such that over ten years after exposure, OGM was associated with poorer outcome.

[14] When the same steps were repeated with number of overgeneral memories, OGM did not significantly predict CPSS, b = -1.28, SE_b = 0.70, p = .07, participant SD = 7.90, school SD = 4.41, assessment SD < 0.01, $\chi^2(2) = 9.83$, p = .01. Further, adding the interaction did not improve the fit of the model, $\chi^2(1) = 0.24$, p = .62. As such, these effects were not found to significantly predict retrieval of overgeneral memories.

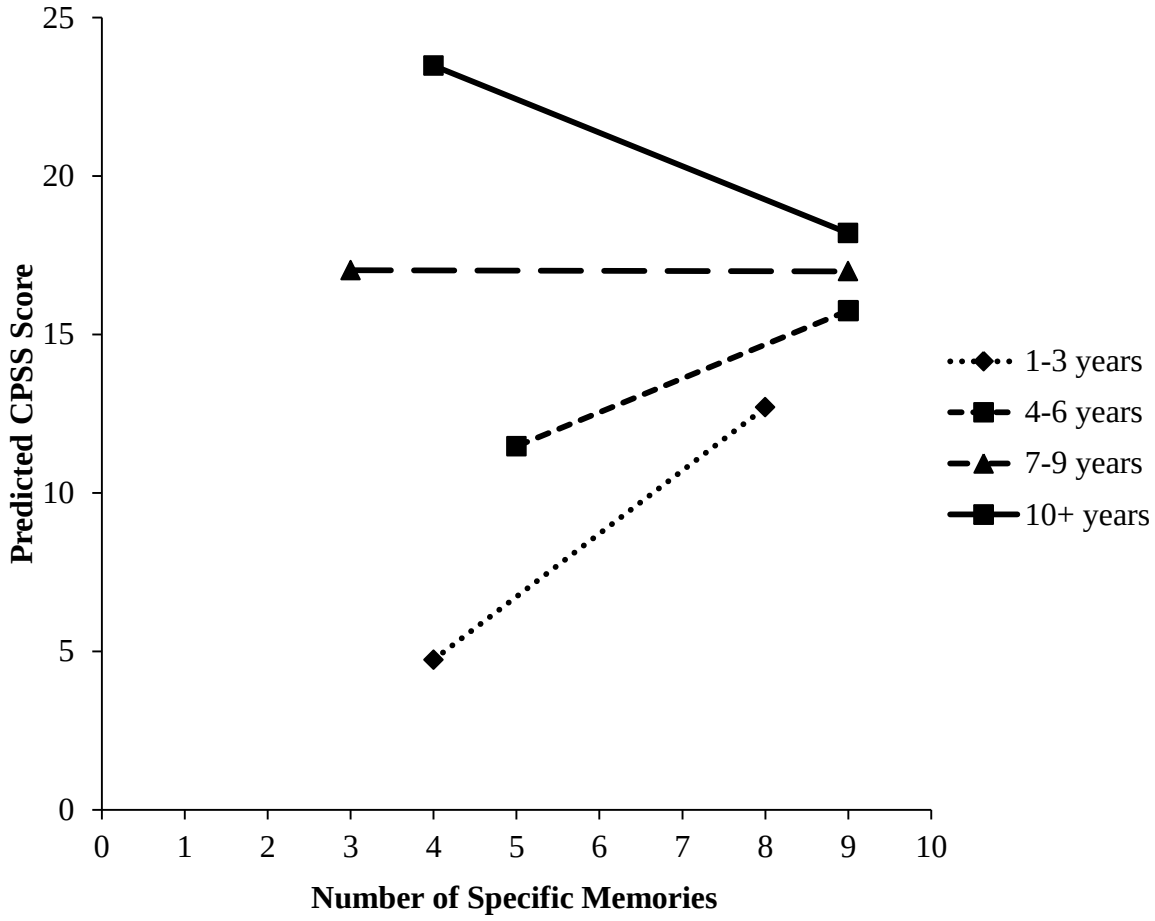

Figure 2. Plotted mixed model for interaction between time since trauma exposure and estimated number of specific memories on predicted PTSD symptoms, represented by score on the Child Posttraumatic Stress Scale (CPSS). Standard errors, which indicate the error of parameter estimates, are presented in the text.[15] Random effects included in the model were participant *SD* = 7.90, school *SD* = 2.41, and assessment *SD* < 0.01.

Prospective analyses in non-clinical participants. Finally, I determined whether specificity predicted depression symptoms in the non-clinical participants. This would indicate whether specificity was a risk factor for later depression. As I wished to examine the relationship in a truly non-clinical sample, data was excluded from those

[15] Calculating confidence intervals on a continuous predictor (as opposed to the coefficient itself) is complicated and appropriate methods are uncertain (Gelman & Hill, 2007). Plotted models for continuous predictors should therefore be interpreted with the standard error of the coefficient to take variability into account.

with a trauma history (and for those missing trauma history data), and from participants with scores equal to or above the CDI-S clinical cut-off. This left 136 participants with no trauma history and non-clinical depression symptoms at T1. I created a mixed-effects model predicting depression symptoms at T2. School was entered as a random effect, along with fixed effects of age and depression symptoms at T1 to control for baseline depression symptoms, school $SD = 0.94$, $\chi^2(2) = 91.63$, $p < .001$. Specificity ($b = 0.44$, $SE_b = 0.32$, $p = .17$) was then entered as a fixed effect. The predicted model was non significant, $\chi^2(1) = 1.30$, $p = .25$. That is, specificity did not predict later depression symptoms.[16]

The previous prospective study reported that it was only specificity to negative cues that predicted later depression symptoms, and only for females (Rawal & Rice, 2012). I therefore determined whether specificity to negative cues would predict depression, and if specificity to negative cues would interact with gender. The above model was repeated, with specificity to negative cues and gender entered in the second step, school $SD < 0.01$, $\chi^2(1) = 188.14$, $p < .001$. The model indicated that baseline depression symptoms significantly predicted depression at T2, $b = 0.86$, $SE_b = 0.12$, $p < .001$, however specificity to negative cues, $b = 0.90$, $SE_b = 0.62$, $p = .15$, and gender, $b = -0.07$, $SE_b = 1.64$, $p = .97$, did not predict symptoms. Adding the interaction between specificity to negative cues and gender did not improve the fit of the model, $\chi^2(1) = 2.55$, $p = .11$. As such, specificity to negative cues and gender did not predict later depression symptoms uniquely, or in interaction.

As the direction of the relationship between OGM and depression is currently unclear, I also assessed whether depression at T1 predicted OGM at T2. This would

[16] As specificity at baseline has been found to predict future depression symptoms in those with elevated symptoms at baseline (e.g., Hipwell et al., 2011; Sumner, Griffith, Mineka, et al., 2011), I did attempt to examine whether depression symptoms at baseline moderated the effect of specificity on symptoms at T2. However, as there were only 31 participants who reached the clinical cut-off (relative to the 165 who did not), this study was not adequately powered to allow this analysis (Rosenthal & Rosnow, 2008). This is not surprising given that I aimed to recruit a sample that was not at elevated risk of depression.

indicate whether depression symptoms influenced the development of OGM. I created a mixed-effects model predicting OGM at T2. School was entered as a random effect, along with fixed effects of age and OGM at T1, school $SD = 0.01$, $\chi^2(2) = 19.45$, $p < .001$. Depression symptoms at T1 ($b = 0.01$, $SE_b = 0.03$, $p = .74$) were then entered as a fixed effect. The predicted model was non significant, $\chi^2(1) = 0.12$ $p = .73$. That is, depression symptoms did not predict later OGM.

Discussion

This study is the first to simultaneously examine all three CaR-FA-X processes (Williams et al., 2007) in young people. Findings indicated that the model was not well supported. Specifically, there was no evidence that the processes predicted OGM uniquely, in interaction with one another, or in interaction with depression or trauma exposure. The secondary aim was to provide a preliminary exploration of whether the number of years since trauma exposure may impact the relationship between OGM and psychological symptoms. I found that time since trauma exposure moderated the relationship between specificity and PTSD symptoms, and that specificity did not predict depression symptoms at any point. Finally, OGM did not predict future depression symptoms in non-clinical participants. These findings offer important implications for both theoretical understanding of OGM and emerging clinical interventions.

This study contributes to accumulating evidence that the CaR-FA-X model as a whole is unable to account for OGM in young people, although it may offer a reliable explanation of OGM in adulthood (see Sumner, 2012). The lack of support for the mechanisms may have been influenced by two aspects of the methodology; measurement and context of retrieval. With reference to the first issue, I only assessed certain aspects of the CaR-FA-X mechanisms. For example, I did not measure the capture aspect of the capture and rumination mechanism. Some support for the capture

aspect has been found in children (Valentino et al., 2009). Thus, simultaneous assessment of both the capture and rumination aspects of the mechanism may produce different results. In addition, my measure of inhibition assessed errors in response inhibition, as Williams et al. (2007) describe OGM as occurring due to errors inhibiting a shift of attention during the retrieval process. Previous OGM research with young people has also measured inhibition errors (e.g., Valentino et al., 2012). Measuring the ability to inhibit a shift in attention, as opposed to inhibiting a conditioned response, may result in a different relationship with OGM. Measures of inhibition generally assess ability to inhibit a response, rather than ability to inhibit a shift in attention. Therefore the inhibition construct defined by the theory may not be adequately tapped by existing measures of inhibition. However, previous support for inhibition in adult samples has been obtained using similar measures (e.g., Piolino et al., 2010). It would therefore appear that measurement does not account for the non significant relationship. Rather, this would appear to reflect that inhibition is not a strong predictor of OGM in adolescents.

In relation to the context of retrieval, recent research suggests that the relationship between the CaR-FA-X processes and OGM may only become evident in certain contexts. For example, Debeer et al. (2011) demonstrated that OGM was only associated with avoidance in a threatening context. Similarly, Smets et al. (2013) suggested that a relationship between rumination and OGM may only be found if state rumination is activated. As OGM was not tested under either of these conditions, this could explain the lack of support for the mechanisms in the present data. In future, contextual factors may need to be considered when examining the processes underlying OGM. Although the results may have been influenced by methodological design, previous examination of the model's processes in both clinical and non-clinical adolescent samples (e.g., Study 1; Nixon, Ball, et al., 2013; Valentino et al., 2012) has

failed to produce strong support for the model. The lack of evidence for a significant role of the mechanisms in the present study adds to a growing number of studies that have failed to support the key tenets of the model in accounting for OGM in young people.

Although the CaR-FA-X model as a whole was not fully supported, there were two findings that are potentially consistent with the proposed role functional avoidance. First, I found a significant effect of trauma history on cognitive avoidance. Williams (1996) stated that trauma exposure would increase functional avoidance. Hence, the finding that trauma-exposed participants demonstrated more cognitive avoidance than non-exposed participants is consistent with predictions drawn from the functional avoidance mechanism. Second, the observed relationship between OGM and PTSD symptoms was consistent with functional avoidance. Williams (1996) proposed that OGM would be adaptive immediately following trauma due to functional avoidance regulating negative affect. However, if maintained in the longer term, OGM would become associated with emotional disturbance. As predicted by the theory, I found that OGM was initially related to lower PTSD symptoms, and that time since trauma exposure did moderate this relationship. The adaptive nature of OGM changed with time since exposure, and appeared to be detrimental approximately ten years after trauma exposure. In addition, high specificity was related to poor outcome for those participants who were in the first six years after exposure. Observation of this effect in a cross-sectional examination with a small sample size suggests the relationship is likely to be robust. Future longitudinal with greater sample sizes will strengthen conclusions regarding the effect of time since exposure. In turn, this will strengthen support for Williams' (1996) prediction.

This moderation was consistent with the hypothesised role of functional avoidance, however, I was unable to distinguish between the effects of age at exposure

and time since trauma. This was due to a low amount of variation in age at exposure. As participants were a similar age when assessed, this meant that the length of time since exposure was strongly related to how old the child was when exposed. That is, I was unable to establish whether time since exposure was a unique effect due to little variance in age at exposure (e.g., a child exposed to trauma when aged 10, which was 2 years ago vs. a child exposed to trauma when aged 10, which was 4 years ago). It is therefore important to note that the findings may reflect age at trauma exposure. If this were the case, this would reflect that OGM was worse for psychological functioning if the child was younger when exposed to trauma. Williams (1996) stated that younger age at exposure will lead to increased OGM due to heightened engagement of the functional avoidance mechanism, as younger children will have a restricted range of other coping strategies (Cole et al., 1994). However, he did not make predictions as to whether age at exposure will influence how OGM relates to psychological symptoms. These findings suggest that future research should consider time since exposure, and age at exposure, when exploring whether OGM predicts psychological symptoms. Such research will need to recruit a large age range to address the limitation of the present study. In summary, although this study was unable to offer firm evidence for role of functional avoidance, preliminary evidence for the mechanism suggests that functional avoidance warrants further investigation.

This study offers important implication for the ability of OGM to predict depression in trauma-exposed and non-clinical samples. There was no evidence for a relationship between OGM and depression symptoms in either trauma-exposed or non-clinical participants. Results with trauma-exposed participants are consistent with Study 1 and previous research that has demonstrated no relationship between depression and OGM in trauma-exposed samples (de Decker et al., 2003; Meesters et al., 2000; Stange et al., 2013). Valentino et al. (2012) also indicated that the relationship between

depression and OGM was weaker in trauma-exposed versus non-exposed participants. The lack of relationship may reflect the adaptive role of OGM post-trauma. However, as the relationship between specificity and depression did not change from adaptive to maladaptive, as observed for PTSD symptoms, it would appear that OGM will relate differently to depression than PTSD following childhood trauma. That is, while OGM may be adaptive for PTSD symptoms initially following trauma, and later become maladaptive, there are consistent findings (including the current study and Study 1) that suggest that OGM may not predict depression symptoms in trauma-exposed young people. Further exploration of the predictive nature of OGM in PTSD and depression will inform theory by clarifying whether separate models of OGM may be needed for PTSD and depression. In turn, such research will indicate how OGM relates to clinical presentation following trauma, and whether OGM is of clinical utility for both conditions.

The current study builds on previous research by providing prospective data in non-clinical young people. The finding of a non significant relationship between OGM and depression symptoms in non-clinical participants differs from previous research. I found that specificity did not predict depression, suggesting that the relationship may not be as robust in children and young adolescents as has been previously found with adults. The effect of OGM on depression symptoms in adult samples is moderated by age. That is, the relationship is stronger with increasing age (Sumner, Griffith, & Mineka, 2010). As such, it may be that OGM is simply a stronger predictor of depression symptoms in adults, relative to adolescents. Alternatively, as my sample was non-clinical and previous studies with adolescents have used those at increased risk of depression (e.g., Hipwell et al., 2011; Rawal & Rice, 2012; Sumner, Griffith, Mineka, et al., 2011; Valentino et al., 2012), it is possible that the relationship may be more pronounced in at-risk populations. For example, the relationship may be stronger in

those at familial risk due to having a mother with depression. Valentino (2011) proposed that mother-child reminiscing would play a central role in the development of OGM. If the mother is experiencing depression, she may be likely to use an overgeneral retrieval style herself (given that OGM has been strongly related to depression maintenance in adulthood; Sumner et al., 2010). Thus, the mother might teach the child to use overgeneral retrieval in a chronic manner, which is proposed to lead to psychopathology (Williams et al., 2007). This idea is consistent with findings of a relationship between OGM and depression in adolescents who have a parent with a depression history (Rawal & Rice, 2012).

A predictive relationship between OGM and depression in samples with elevated symptoms (Hipwell et al., 2011) or a history of depression (Sumner, Griffith, Mineka, et al., 2011) but not the current sample may also indicate that OGM is a maintenance factor for adolescent depression. If OGM is a causal risk factor, it would be expected that OGM would predict future symptoms. However, if OGM maintains depression symptoms, a non significant relationship would be expected as our sample was not experiencing clinical levels of depression. Our findings therefore contribute to growing research which indicates that OGM maintains depression symptoms, rather than representing a vulnerability for the disorder (e.g., Kuyken & Dalgleish, 2011; Valentino et al., 2009). Indeed, longitudinal findings that OGM in those experiencing an MDD episode at baseline will predict future depression symptoms (Rawal & Rice, 2012; Sumner, Griffith, Mineka, et al., 2011) indicates that OGM may emerge, or become active in maintaining symptoms during the first episode, and remain a stable cognitive predictor of later depression. Future prospective research with non-clinical samples will clarify whether OGM is a causal or maintenance factor for adolescent depression, and indicate for which samples OGM intervention will be most effective.

To conclude, this study offers important implications for clinical intervention. First, the relationship between OGM and adolescent depression may not be as robust as first thought. Interventions that seek to modify OGM may be best targeted towards populations at-risk of depression, such as those with family histories or sub-threshold symptoms. Further exploration of how OGM influences depression in clinical samples, in comparison with at-risk and normative samples will indicate for whom intervention will be most effective. Second, the effect of specificity on symptoms was found to vary between disorders. Further investigation of the effects of specificity training on PTSD symptoms is clearly needed. In addition, my results suggested that the adaptive nature of OGM may change to become maladaptive somewhere between four and ten years after trauma exposure. This finding may indicate that OGM intervention is optimally timed within this period. Finally, interventions will also be aided by greater knowledge of the cognitive processes that underlie OGM in young people. This study provided some limited support for the role of functional avoidance in OGM, but not for the CaR-FA-X model as a whole. As such, the next goal of this thesis was to further explore the functional avoidance mechanism.

CHAPTER FIVE

Introduction

This chapter reports on two experiments that assessed the functional avoidance mechanism of OGM. To provide the rationale for assessing functional avoidance, I summarise the accumulated evidence so far for the thesis research questions. First, I asked how OGM related to psychopathology following childhood trauma. Studies 1 and 2 indicated that OGM was not related to depression symptoms. Both studies also demonstrated that time since trauma exposure moderated the relationship between OGM and PTSD symptoms. Specifically, Study 1 reported a negative relationship between OGM and PTSD symptoms at six months post-trauma. Study 2 also suggested that OGM would be negatively related to PTSD symptoms for approximately four years following trauma, however, a positive relationship may emerge after this point. Hence, I have consistent evidence that OGM is negatively related to PTSD symptoms, although this relationship is moderated by time since exposure, and unrelated to depression symptoms in trauma-exposed young people.

Evidence for my other research question was less consistent. I examined whether the CaR-FA-X mechanisms account for childhood OGM. Study 1 and Study 2 offered no support for the capture and rumination, and executive control mechanisms. However, some findings have been consistent with assumptions of the functional avoidance mechanism. To remind the reader, Williams (1996) stated that functional avoidance can initially promote emotional stability following childhood trauma. This is due to functional avoidance regulating the negative affect produced by the trauma memory. Consistent with this proposal, Study 1 and Study 2 demonstrated that OGM was negatively related to PTSD symptoms in the shorter term following trauma. I therefore have some evidence that OGM may be beneficial following childhood trauma. If I found evidence that functional avoidance of specific memories regulated negative

affect, this may indicate that the functional avoidance mechanism underlies the relationship between OGM and good adjustment following childhood trauma. Therefore, I next completed two experiments assessing the functional avoidance account of childhood OGM.

These experiments provided the first empirical assessment of two key theoretical assumptions of functional avoidance. Theory of autobiographical retrieval states that the voluntary retrieval of an autobiographical memory will occur through a controlled (i.e., generative) retrieval process (Conway & Pleydell-Pearce, 2000). Autobiographical information is proposed to be stored in a series of nested indices. These indices store information in a hierarchical manner, such that lifetime periods are contained at the top of the index, moving down to categories of general events and finally, to finer, event-specific details that are stored at the bottom of the index. A specific autobiographical memory is produced by a controlled, top-down search of the indices which is guided by a retrieval cue. Williams (1996) stated that overgeneral retrieval will occur when the search is terminated prior to reaching event-specific information. Avoidance of specific details will occur in two stages; the first proposed to be adaptive, and the second proposed to be maladaptive. First, avoidance will begin with the specific details of one adverse memory (i.e., for searches guided by a cue related to an adverse event). This is thought to occur in particular relation to childhood trauma. As finer detail of an adverse event is proposed to be distressing to a child, the child will avoid the specific memory of the event to manage the negative affect produced by the memory (Williams, 1996). For example, recalling how fearful they were the first time their father hit them will produce stronger negative affect than if the child simply recalled that their father had been abusive. As such, the child will actively abort a search guided by the cue ‘fear’ before it reaches event-specific details. This process was termed functional avoidance, and leads to two key predictions: (i) specific details of an adverse event will be

distressing for a child, and (ii) avoidance of these specific details will avoid negative affect. My primary aim was to assess these predictions in children's autobiographical memory.

Assessing these hypotheses is important for two reasons. First, these predictions form the basis of the dominant theory of overgeneral retrieval (Williams et al., 2007), however, they have not been directly assessed. Although evidence has been provided for an affect regulation role of overgeneral retrieval following adverse events in adulthood (e.g., Hermans et al., 2008; Raes et al., 2003), no study has examined these predictions in relation to adverse childhood events. As children's skill in cognitively regulating their own emotions is still developing, their ability to use overgeneral retrieval to regulate affect may not be as effective as mature adults. Children use others, particularly adults, to help regulate their emotions (e.g., Cole et al., 1994). Further, their cognitive ability to independently regulate their emotions increases with age (e.g., Stegge & Terwogt, 2007). Due to these developmental effects, direct examination of the key predictions of functional avoidance needs to be completed in children to accurately assess the theory. Experimental evidence for these predictions in children's autobiographical retrieval would provide the strongest support for the role of functional avoidance to date.

The findings will also have implications for the relationship between childhood trauma and overgeneral retrieval.[17] Childhood trauma is thought to be fundamental in increasing or initiating overgeneral retrieval. Williams (1996) proposed that after experiencing trauma a child will begin to avoid specific details of the trauma memory to regulate the negative affect produced by the memory. This will then lead to an overgeneral retrieval tendency. Hence, functional avoidance is proposed to mediate the

[17] I deliberately use the term overgeneral retrieval, rather than overgeneral memory, throughout this chapter. This is because OGM is thought to be a cognitive trait, and I experimentally manipulated the children's retrieval strategy. In doing so, I have examined state overgeneral retrieval rather than an ingrained, more consistent overgeneral retrieval tendency.

relationship between childhood trauma and overgeneral retrieval. Consistent relationships have been observed between childhood trauma and overgeneral retrieval during childhood (e.g., de Decker et al., 2003; Valentino et al., 2009) and adulthood (Moore & Zoellner, 2007). Yet, assessment of a mediating role of functional avoidance has not been completed (for review see Sumner, 2012). Before mediation can be assessed, we need to determine that avoidance of specific memories of an adverse event will indeed regulate negative affect. In addition to establishing the prerequisites for mediation, evidence that avoiding specific memories regulates affect would also explain why overgeneral retrieval is adaptive following adverse childhood events. Therefore, examining these predictions will have important implications for the functional avoidance explanation of overgeneral retrieval. This includes whether childhood trauma may increase functional avoidance, and whether overgeneral retrieval may be adaptive in the short term following adverse childhood events.

In addition to testing whether the functional avoidance predictions were supported in retrieval of an adverse childhood memory, I also explored whether the predictions applied to the second stage of overgeneral retrieval. In this second stage overgeneral retrieval will begin to be used for memories other than that adverse event. Williams et al. (2007) proposed that avoidance of negative affect produced by a specific adverse memory will be negatively reinforced and children will begin to abort all cued memory searches prior to retrieving specific details. Importantly, OGM theory states the child will begin to avoid specific details of not only other negative events, but also positive events. This is because cues used to guide the search for a positive memory (e.g., ‘chocolate’) may still activate a specific memory of a negative event (e.g., ‘Dad gave me chocolate after the first time he hurt me’). Hence, avoidance will generalise to all specific memories, regardless of whether the cue used to guide a search is positive or negative.

Although overgeneral retrieval of an adverse memory was proposed to regulate negative affect, Williams et al. (2007) were unclear whether broader use of overgeneral retrieval will continue to regulate affect. That is, it was not outlined whether retrieval of all specific memories will be distressing, or perhaps more importantly, whether avoidance of specific memories of other autobiographical events will regulate negative affect. If retrieval of a specific memory induces the affect of that memory, and avoiding specific memories avoids that affect, the overall affect experienced by the individual will depend on which type of memories are retrieved. For example, if only negative specific memories are avoided, negative affect will be avoided, but positive affect will still be experienced. However, if the individual avoids both positive and negative specific memories, both positive and negative affect will be reduced. In this circumstance, the advantage of minimising negative affect might come at a significant cost, that is, by reducing the degree to which positive affect is experienced.

Research in adult samples does not suggest an obvious resolution to this ambiguity. Findings on the impact of overgeneral retrieval on affect differ between correlational and experimental studies. Correlational research has indicated those who naturally retrieved a high number of specific memories experienced more distress following a negative event compared to those who were naturally overgeneral (Hermans et al., 2008; Raes et al., 2003; Study 1, Raes, Hermans, Williams, & Eelen, 2006). These studies did not examine differences in the specificity of retrieval between positive and negative cues. As such, it is possible that participants were only being overgeneral to the negative event, and not for broader autobiographical retrieval. These results would be consistent with overgeneral retrieval of an adverse event memory regulating negative affect. In contrast, experimental research has demonstrated that those induced to be overgeneral to both positive and negative cues experienced more distress following a negative event or negative mood induction than those instructed to be

specific (Philippot, Schaefer, & Herbette, 2003; Study 2, Raes et al., 2006). It is possible that this discrepancy in findings may be due to different use of overgeneral retrieval between samples. That is, these experimental studies induced overgeneral retrieval to both positive and negative cues, while correlational studies may have only observed overgeneral retrieval in relation to the adverse event. Discrepancy in findings may therefore suggest that avoidance of all specific memories impacts affect differently than avoidance of a specific adverse memory. No previous research has distinguished between overgeneral retrieval for one adverse event versus broader autobiographical memory when examining the impact on affect. I completed two experiments to further investigate this issue. The first experiment examined how affect was impacted by broader overgeneral retrieval. The second experiment examined how affect was impacted by overgeneral retrieval of an adverse event.

Examining the functional avoidance predictions in retrieval of other autobiographical memories may also provide insight into the theorised change of the style from an adaptive to maladaptive factor. Williams (1996) stated that if overgeneral retrieval was maintained over the longer term, generalising to most, if not all, autobiographical experiences, it would become associated with psychopathology. Indeed, findings from Study 2 supported this proposition. However, the model was not clear as to why broader overgeneral retrieval would become maladaptive (although it was proposed that the style would become associated with impaired problem solving and difficulty imagining the future; Williams et al., 2007). I suggest that use of overgeneral retrieval for positive and negative memories may lead to emotional disturbance by impairing ability to repair low mood. That is, avoiding positive specific memories, and hence positive affect, will impair the child's ability to repair low mood. As such, a key contribution of these experiments was to determine whether the manner

in which broader overgeneral retrieval influences affect may explain why the retrieval style can change from being adaptive to maladaptive.

Therefore, the goal of these experiments was to determine: (a) whether retrieval of specific memories was distressing for children, and (b) if avoidance of specific memories led to avoidance of induced negative affect. The first experiment assessed these predictions when children were asked to retrieve positive and negative specific memories, whereas the second experiment assessed these predictions when children retrieved specific memories of a single, negative affect inducing event. In both experiments I manipulated the specificity of autobiographical retrieval in upper primary school aged (i.e., 9-13 year old) children. By this age children have some skill in cognitively regulating their internal emotional state (Stegge & Terwogt, 2007). I induced negative affect prior to retrieval to examine how retrieval style influenced negative affect. If retrieval of specific memories is distressing for children, I would expect those children instructed to retrieve specific memories would experience a decrease in positive affect from pre- to post-retrieval. In addition, if avoidance of specific memories minimises negative affect, I would expect that children instructed to avoid specific memories would experience negligible change in affect. It is important to note that the theory would not predict an improvement in affect, as it states that avoiding specific memories will avoid negative affect, not increase positive affect.

Study 3

This study determined whether avoidance of all specific memories in the experiment influenced negative affect. I manipulated instructions on the Autobiographical Memory Test (AMT; Williams and Broadbent, 1986) to create three retrieval conditions. When presented with a cue, participants were required to produce an autobiographical memory related to that cue. However, instructions were altered to direct participants to either avoid specific memories (Avoid condition), retrieve specific

memories (Specific condition), or gave no instruction regarding specificity (Control condition). I measured affect before and after the task to determine how change in affect differed between retrieval conditions.

Examination of change in affect from pre- to post-AMT allowed me to determine (a) whether retrieving specific memories was distressing for the child, and (b) whether avoidance of all specific memories would avoid negative affect. Although Williams (1996) was unclear as to how avoidance of all specific memories will impact affect, I based my predictions on the original functional avoidance hypotheses. If retrieval of specific memories is distressing, I would expect the Specific condition to demonstrate a decrease in positive affect. In addition, if avoiding specific memories leads to avoidance of negative affect, I would expect that the Avoid condition would experience negligible change in affect. As I left the Control condition to choose their own retrieval strategy, I expected that they would experience an improvement in affect, as they would use whichever type of retrieval usually benefitted them when they were experiencing negative affect.

In line with these predictions, I hypothesised an interaction between retrieval condition and time (i.e., pre-AMT and post-AMT) on affect. Specifically, those in the Specific condition would demonstrate a decrease in positive affect from pre- to post-AMT. However, those in the Avoid condition would not significantly change from pre- to post-AMT. Finally, the Control condition would demonstrate an increase in positive affect from pre- to post-AMT.

Method

Participants

Eighty three Year 5 students (37 females) were recruited from three private metropolitan schools in suburbs of middle socio-economic status. Students were aged 9-11 years (M = 10.36, SD = 0.35). Ninety six per cent of participants were Caucasian,

2.4% (n = 2) were of Asian ethnicity, and 1.2% (n = 1) were Eurasian. All participants received a pen as compensation for their time.

Power analysis for the linear mixed-effects modelling approach I used is not well established. Therefore, I estimated the necessary number of participants needed per condition to detect a medium effect using a typical parametric analysis. Cohen (1988) indicated this required 33 participants per condition for power of .80. The final sample size was determined by how many consent forms were returned. Although the recruited number of participants was lower than this goal, mixed-effects modelling is more powerful than typical parametric analysis. Hence, it was deemed appropriate to cease recruiting. Following completion of data collection, I completed post hoc analysis of effective power to ensure the sample size was sufficient. The observed effect size in the smallest condition (d = 1.27, n = 23) was associated with power of .70 (Rosenthal & Rosnow, 2008). Therefore, analyses were adequately powered.

Measures

Autobiographical Memory Test: Written Form (AMT). The AMT (Williams & Broadbent, 1986) contains a series of trials in which the participant is presented with a cue and asked to recall an autobiographical memory pertinent to the cue. I used five positive and five negative cue words (happy, sad, lucky, lonely, proud, scared, brave, angry, playing, hurt). Both written and verbal forms of the AMT are available. I used the written form which required participants to write their response. Similar proportions of specific memories have been reported between written and verbal versions of the AMT (Raes et al., 2006). I used different task instructions between conditions to manipulate retrieval strategy (see Appendix C). Three retrieval conditions (Specific, Avoid, and Control) were created. The Specific condition used traditional AMT instructions that encourage participants to retrieve a specific memory (i.e., provide a memory of a particular time or one specific event). Two examples of correct

memories, along with one incorrect memory were given to ensure the task instructions were clear. The Avoid condition were asked to avoid thinking of a specific event or one particular time that something happened (as instructed in the reversed version of the AMT; Dalgleish et al., 2007). Instead, they were instructed to recall 'things that happen lots of times, not something that happened one time'. As in the Specific condition, two examples of correct and one incorrect memory were given. Finally, a Control condition was created by asking the child to retrieve a memory without giving any instruction regarding response specificity. This was done to provide an indication of the degree of retrieval specificity a child would naturally use when experiencing negative affect.

The AMT was administered in group format. All instructions were printed at the top of the response page, and read aloud by the researcher. Cues were also presented in written form and read aloud by the researcher. After each cue, children were instructed to write their memory below the cue on the response page. Children were allowed one minute to record their memory before the next cue was administered. Responses were scored as specific, overgeneral (categoric or extended), sematic associate, violation (i.e., the response was illegible), or omission. Use of this written AMT with the same scorers in Chapter 3 demonstrated good inter-rater reliability ($\kappa = .85$).

Mood induction. To determine how retrieval strategy impacted negative affect, sad mood was induced in all participants prior to the AMT. Children were asked to identify a time when they felt "really sad", and instructed to think and write about it for three minutes. During this time, a piece of sad string music (Samuel Barber's Adagio for Strings, 1936) was played. This track and instructions have been used to induce sadness in children (Romens & Pollak, 2012). After the writing task, participants completed two minutes of guided imagery focussing on the sad event. Again, this guided imagery task has been used for sad mood induction in children of this age (e.g., Joormann, Gilbert, & Gotlib, 2010; Joormann, Talbot, & Gotlib, 2007). Events

described in this sample included nightmares, death of a pet, and difficulties with friends.

Psychopathology. Depression symptoms were measured using the short form of the Child Depression Inventory (CDI-S; Kovacs, 1992). Adequate alternate form reliability exists between full and short forms (r = .89; Kovacs, 1992). I used normed scores in all analyses. Symptoms of posttraumatic stress were measured using the Child Posttraumatic Stress Scale (CPSS; Foa et al., 2001). The CPSS is a self-report measure in which children are required to rate their experience of all seventeen symptoms of PTSD in relation to their most frightening life experience. The measure offers adequate internal consistency (α = .89). In addition to the total score, scores were also calculated for the hyperarousal, avoidance, and re-experiencing subscales.

White Bear Suppression Inventory (WBSI). The WBSI (Wegner & Zanakos, 1994) was used to assess children's tendency to avoid unwanted thoughts. In particular, I wished to determine if avoiding unwanted thoughts was related to specificity in the Control condition as this may provide an indication of whether overgeneral retrieval is a more general cognitive avoidance strategy. The scale has been used with children of this sample's age, and has adequate internal consistency and reliability (Vincken, Meesters, Engelhard, & Schouten, 2012).

Adverse event history. Parents were given a list of negative life experiences and asked to indicate if any of the events had ever happened to their child (scored dichotomously). Events included motor vehicle accidents, physical assault, and neglect. The list was based on the life events scale used in the Clinician Administered PTSD Scale- Child Version (Nader et al., 1996) . The child's age at the time of endorsed events was also collected. The ethics committee restricted us from directly asking the child these questions.

Affect. Children were asked to circle the face that best represented how they were currently feeling from a series of five faces of varying emotional intensity: two frowning (big frown and small frown), one neutral, and two smiling faces (small smile and big smile). Based on the commonly used criteria created by Taylor and Ingram (1999), selections were then converted to a score of one to five, where 1 = very sad, 3 = neutral, and 5 = very happy. Thus, a score of 1 indicates strong negative affect and a score of 5 indicates strong positive affect. This rating scale was administered directly before the mood manipulation, pre-AMT, post-AMT and at the end of the session.

Procedure

Data collection was approved by the institution's human research ethics committee. Consent forms were sent home to parents to gain informed consent, along with the adverse event history checklist. Assent was gained from the child immediately before beginning the session. Sessions were completed in group format, with no more than 18 students in any one session. Each group was randomly allocated to the Control, Avoid, or Specific condition. Children completed demographic questions and an affect rating. The mood manipulation was completed, and then the pre-AMT affect rating was taken. Next, the AMT was administered followed by the post-AMT affect rating. Finally, the CDI-S, CPSS and WBSI were completed, along with the last affect rating. During the session, a researcher circulated around the room to answer any questions. The session lasted approximately 45 minutes. At the end of the session, all students were debriefed and a mood lifting exercise was completed.

Results and Discussion

Data Analysis Approach

All Cohen's *d*s were calculated using the standard deviation of the particular variable from pre-manipulation scores. Ninety five per cent confidence intervals are reported for each effect. All analyses were completed using a linear mixed-effects

modelling approach. This approach allowed me to control for random effects of school, as I recruited from three different sites. Each model had a random effect of school entered in the first step. Key variables were entered in the second step, followed by any interactions in the final step. The standard deviation for the random effect is reported for the model of best fit. The standard deviation reflects the estimated variance in model intercept between schools. Effect sizes are indicated by *b* coefficients, which display the amount of change expected in the outcome variable for a one unit increase in the predictor. As in regression, entering a categorical predictor into a mixed-effects model requires use of a reference group. As such, when entering condition as a predictor into the models, I needed to create a reference group. I used the Control condition as the reference group, as I was interested in how the induced retrieval strategies influenced affect relative to the child's preferred choice of retrieval strategy.

Manipulation Checks

I completed manipulation checks prior to beginning analyses. The retrieval strategy manipulation aimed to induce a higher number of specific memories in the Specific condition than the Avoid condition. To check that this occurred, I created a mixed-effects model predicting number of specific memories with school as a random effect in the first step. I next added group as a fixed effect, school $SD = 0.62$, $\chi^2(2) = 115.03$, $p < .001$. As intended, those in the Specific condition ($M = 8.61$, $SD = 1.12$, [8.13, 9.09]) demonstrated a higher number of specific memories than those in the Avoid condition ($M = 1.07$, $SD = 1.61$, [0.43, 1.71], $d = 5.47$, [5.01, 6.08]), $b = -7.57$, $SE_b = 0.51$, $p < .001$, and Control condition ($M = 7.12$, $SD = 2.41$, [6.27, 7.97]), $d = 0.76$, [0.31, 1.59], $b = -1.61$, $SE_b = 0.57$, $p = .01$. As such, manipulation of retrieval specificity was successful.

I also determined that the sad mood manipulation successfully induced negative affect. I created a mixed-effects model predicting affect with school entered as a random

effect. The next step included time (i.e., immediately pre-induction versus immediately post-induction) as a fixed effect, school $SD < 0.01$, $\chi^2(1) = 124.36$, $p < .001$. Consistent with successful manipulation, the mean affect rating decreased after the mood induction (pre-induction $M = 4.04$, $SD = 0.79$, [3.87, 4.21]; post-induction $M = 2.23$, $SD = 0.93$, [2.03, 2.43], $d = 1.89$, [1.72, 2.06]), $b = -1.81$, $SE_b = 0.13$, $p < .001$. The mean post-induction was representative of selection of a sad face. As such, the mood induction was successful.

Hypothesis Testing

I hypothesised an interaction between condition and time (i.e., pre- or post-AMT) on affect. I predicted that those in the Specific condition would demonstrate a decrease in positive affect from pre- to post-AMT. In contrast, those in the Avoid condition would not experience a significant change in affect from pre- to post-AMT. To test this hypothesis, I created a mixed-effects model predicting affect. School was entered as random effect in the first step. Time (i.e., pre- or post-AMT) and condition were entered in the second step, school $SD = 0.28$, $\chi^2(3) = 69.46$, $p < .001$. As I was interested in how the Avoid and Specific conditions experienced change in affect relative to the Control condition, the Control condition was used as the reference group. The best fitting model included significant coefficients for time, $b = 1.26$, $SE_b = 0.14$, $p < .001$, and the Specific condition, $b = 0.43$, $SE_b = 0.20$, $p = .04$, but not the Avoid condition, $b = 0.18$, $SE_b = 0.20$, $p = .36$. Adding the interaction between time and condition did not significantly improve the fit of the model, $\chi^2(2) = 0.93$, $p = .63$. As such, I had no evidence for a significant interaction between time and condition in their influence on affect. This is reflected in the aggregate descriptive statistics (see Table 4). Confidence intervals for the mean difference from pre- to post-AMT overlap substantially between conditions. This indicates there is no evidence for a meaningful difference between groups. When examining the means of affect ratings for pre- and

post-AMT, it is apparent that each condition experienced an increase in positive affect. Contrary to predictions, the Specific condition demonstrated improved affect, as did the Avoid condition. Thus, my hypothesis was not supported. This indicates there was no evidence that retrieving specific memories changed affect any differently than avoiding specific memories.

Table 4

Mean Affect Rating Pre- and Post- Autobiographical Memory Test (AMT) by Condition

		Pre-AMT		Post-AMT		Mean difference	
Condition	*n*	*M* (*SD*)	95% CI	*M* (*SD*)	95% CI	*d*	95% CI
Control	33	2.27 (0.88)	[1.96,2.60]	3.39 (0.75)	[3.12,3.66]	1.27	[0.97, 1.57]
Avoid	27	2.11 (0.85)	[1.77, 2.45]	3.42 (0.70)	[3.14,3.70]	1.54	[1.21, 1.87]
Specific	23	2.30 (1.11)	[1.82,2.78]	3.74 (1.14)	[3.25,4.23]	1.30	[0.84, 1.75]

Note. *d* = within-group effect size. All *d*s are absolute values. CI = Confidence Interval. Affect rating was on a scale of 1 – 5, where 1 = very sad and 5 = very happy.

Psychological Symptoms

Overall, the sample demonstrated low symptoms of depression (*M* = 48.79, *SD* = 9.99, [46.48, 51.10], Range = 40-85). The mean was well within the normal range. The mean CPSS score was high for a non-clinical sample (*M* = 15.24, *SD* = 11.06, [12.71, 17.76], Range = 0- 46). A score of 16 or above falls into the clinical range (Nixon,

Meiser-Stedman, et al., 2013), and 40.3% of the sample reached this cut-off. This is much higher than the community prevalence rate of child and adolescent PTSD of approximately 10% (Kilpatrick et al., 2003). The sad mood induction may have contributed to the unusually high ratings on the CPSS, and the events listed by children as causing the symptoms did not always qualify for a PTSD-inducing event (e.g., a nightmare). To ensure that the experimental conditions did not significantly differ in reported symptoms, I created a mixed-effects model predicting CPSS score. School was entered as a random effect. When condition was added to the model (Avoid $b = 1.50$, $SE_b = 3.05$, $p = .62$; Specific $b = -0.94$, $SE_b = 3.09$, $p = .76$), it did not significantly improve the fit of the model, school $SD < 0.01$, $\chi^2(1) = 0.06$, $p = .81$. I also created a model predicting CDI-S score with the same predicting factors. Again, adding condition (Avoid $b = 1.05$, $SE_b = 2.67$, $p = .69$; Specific $b = 0.16$, $SE_b = 2.76$, $p = .95$) did not significantly improve the fit of the model, school $SD < 0.01$, $\chi^2(1) = 0.01$, $p = .92$. Hence, there was no evidence that the experimental conditions significantly differed in depression or PTSD symptoms.

I found no evidence that WBSI score, $b = -0.01$, $SE_b = 0.03$, $p = .61$, was related to number of specific memories produced in the Control condition, school $SD = 0.85$, $\chi^2(1) = 8.38$, $p < .001$. This indicates that suppression of unwanted thoughts was not related to specificity. As such, I did not include WBSI in further analyses.

Conclusion

I predicted that retrieving specific memories would be distressing for children. The findings demonstrated that retrieving specific memories cued by both positive and negative cues did not decrease positive affect. This indicates retrieval of specific memories was not distressing. Williams (1996) proposed that an overgeneral retrieval style would begin in response to a specific memory of an adverse event, and later generalise to other autobiographical experiences. Thus, an alternative explanation for

the current findings is that it may only be retrieval of specific memories of a particular adverse event that will cause a child distress, not specific retrieval of all autobiographical memories.

I also predicted that avoidance of specific memories would minimise negative affect. Results suggested that those instructed to avoid specific memories experienced an increase in positive affect from pre- to post-AMT. Although this is consistent with avoiding negative affect, the Specific condition also experienced an increase in positive affect. Thus, the Avoid condition did not need to engage in avoidance to minimise distress. Further, Williams (1996) predicts no significant decrease in affect, as opposed to an improvement in positive affect, thus, results did not support the proposal that overgeneral retrieval will avoid negative affect.

Interestingly, all conditions experienced an increase in positive affect from pre- to post-AMT. This indicates that although overgeneral retrieval will not regulate affect any more than specific retrieval, neither style will have a detrimental impact on mood. As all three conditions experienced an increase in affect, this would suggest that it was something that all conditions experienced, not the experimental manipulation, which caused the change. It is possible that all participants experienced natural recovery, as completing the AMT may have distracted them from the induced negative affect. That is, it may not be that retrieving either specific or overgeneral memories improved affect, but that the effect is simply attributable to distraction. The induced negative affect was not particularly long lasting, thus with minimal distraction, it may have lifted naturally. I aimed to explore this potential explanation in the second experiment by examining the influence of distraction on affect.

To conclude, I found no evidence for the functional avoidance assumptions that retrieving specific memories would cause distress, and that avoidance of specific memories would avoid this distress when retrieving positive and negative memories. As

such, it may be that the impact of functional avoidance is only seen in the retrieval of specific memories of a single negative event. Thus, my next goal was to assess whether functional avoidance was supported in the retrieval of specific memories of an adverse event, as originally outlined by Williams (1996).

Study 4

This experiment provided a more stringent test of the role of functional avoidance in overgeneral retrieval. I determined (a) whether retrieval of specific memories of a *particular* negative event was distressing for children and (b) if avoidance of specific memories of that event led to avoidance of negative affect. I asked participants to describe one of three common childhood events that are distressing for the child (Goodyer et al., 1997). These were death of a friend or family member, death of a pet, and being bullied by other children. I anticipated that describing the event would induce negative affect. To examine the research questions, I then explored how retrieval style impacted this affect. I was most interested in whether retrieving specific memories of the event would make the children feel worse, and if avoiding specific memories of the event would help the children to avoid further negative affect. As in the first experiment, I created a Specific condition that instructed children to retrieve specific memories of the event, and an Avoid condition that had children avoid specific memories of the event. In addition to examining the impact of the retrieval strategies on affect, I was also interested in how distracting from the mood-inducing memory would influence affect. This was because a potential explanation of Study 3 findings was that the task might have resulted in distraction, which in turn buffered children from negative affect. I therefore created a Distraction condition, as opposed to the traditional control group used in the first experiment. This group completed a cued-recall task designed to distract them from the event memory.

If the key assumptions of functional avoidance are correct, I expected that retrieving specific memories of an event that induced negative affect would intensify that affect. However, avoiding specific memories of this event should buffer children from further negative affect. In addition, based on the findings of the Study 3, I expected that distracting the children from the negative memory would improve affect. I therefore hypothesised that retrieval condition and time would interact in their influence on affect. Specifically, the Specific condition would demonstrate a decrease in positive affect from pre- to post-AMT. However, the Avoid condition would demonstrate no significant change in affect from pre- to post-AMT. Finally, it was anticipated that the Distraction condition would demonstrate an increase in positive affect from pre- to post-AMT.

Method

Participants

One hundred and two students (56 females) were recruited from two private metropolitan schools in suburbs of middle socio-economic status. Students were aged 10-13 years. Mean age was 11.93 years (*SD* = 0.59). Ninety per cent of participants were Caucasian, 2% (*n* = 2) were of Indian heritage, and 1% (*n* = 1) was Eurasian. Ethnicity information was missing for 7% of students due to parental omission on the form. All participants received a pen as compensation for their time. As in the previous experiment, I estimated the necessary sample size (33 per condition for a power of .80) based on recommendations of Cohen (1988). The final sample size was determined by the number of consent forms that were returned. Although one condition fell two participants short of the desired number, mixed-effects modelling is more powerful than typical parametric analyses. Further, the study was still adequately powered to detect a moderate effect (Cohen, 1988).

Materials

Negative event description. I gave participants three minutes to write a description of one of the three events (when a friend or family member had passed away, or when a pet had passed away, or when they had been bullied by other children). Participants were instructed to choose which event was most upsetting for them, and to then write a description of the event, which could be as brief or detailed as they liked. Of the three options, 42.2% wrote about a friend or family member dying, 31.4% wrote about a pet dying, 21.6% wrote about being excluded by others, and 3.9% described an adverse event that did not fit into one of the categories (data was included for these participants as the events were still discrete, negative events). I also collected data on how distressing the nominated event was to the child. Therefore participants completed two 10 point scales (1 = not upset, 10 = very upset): (a) How upset does this memory make you feel when you think about it now? and, b) How upset did this event make you feel right when it happened? Participants were also asked to state how old they were when the event occurred. Mean rating of how upsetting the memory is now was 5.92 out of 10 (*SD* = 2.34, [5.46, 6.38], Range = 1-10). Not surprisingly, children reported that the event was substantially more distressing at the time it occurred (*M* = 8.72, *SD* = 1.85, [8.36, 9.09], Range = 2-10). On average, the chosen event occurred when the participant was approximately 9.10 years old (*SD* = 2.19, [8.62, 9.58], Range = 1-12 years), or 2.39 years ago (*SD* = 2.06, [1.94, 2.84], Range = 0-10 years).

AMT. The AMT I used was similar to the previous experiment (see Appendix D for instructions). However, the Specific condition was explicitly instructed to retrieve a specific memory of the adverse event they had identified. This included the example "For the word 'bad', it would be OK to say 'I felt bad when my dog got put down', because this is a specific memory about the time your dog passed away. Another example is if I said the word 'pain' you could say 'My dog was in pain so we took him

to the vet, and then the vet said he had to be put down'." The Avoid condition was asked to avoid thinking of the specific event they had identified, and instead identify repeated categories of events. I specifically instructed students to be categorical to facilitate termination of the search at the general event level of information (e.g., to terminate the search at 'when I used to walk my dog' which could have led to the specific memory 'when my dog was put down'). For each condition, two examples of correct memories were given, along with one example of an incorrect memory. All cues were negative (sad, lonely, scared, angry, hurt, upset, tears) to encourage the child to retrieve autobiographical information on the nominated adverse event. Further, this guarded against positive cues resulting in positive affect, which may negate the impact of retrieving specific memories to negative cues.

The Distraction condition completed a cued-recall task about movies and TV shows. Participants were presented with seven cues (a movie or TV show with a villain/bad guy, animals, friends, magic, a cartoon, superhero, monsters or aliens) and asked to name and describe the plot of a movie or TV show in response to the cue. A cued-recall task was used to replicate the cognitive load required in the AMT. As in the AMT, cues were read aloud by the researcher, and one minute was given for each response.

Psychopathology. The same self-report measures of depression and posttraumatic stress were used as in the first experiment. Mean score on the CDI-S was 50.59 (*SD* = 11.34, [48.28, 52.90], Range = 40-99). Mean CPSS score was 10.88 (*SD* = 10.45, [8.77, 13.00], Range = 0-38). Both means were within the normal range.

Adverse event history. Three extra questions were added to the questionnaire from the previous experiment. These asked parents to indicate if their child had experienced any of the three adverse events I examined (i.e., death of a friend or family member, death of a pet, being excluded by other students), with parents also providing a

rating (out of 10) as to how distressing they believed that event had been for their child and how old their child was when the event occurred. The mean number of adverse events reported by parents was 2.14 (*SD* = 1.40, [2.85, 3.42], Range = 0-7) per child. Seventy-eight per cent of students had had a pet pass away, 71.9% had experienced the death of a family member or friend, and 56.4% had been excluded by other children. Other adverse events endorsed included natural disaster (7.3%), motor vehicle accident (19.8%), and accident or illness that may have caused death or serious injury (30.2%).

Affect. The same affect ratings from the previous experiment were used, with higher scores indicating more positive affect.

Procedure

I followed the same procedure as the previous experiment, with the exception that participants completed the negative event description as the mood induction. The thought suppression measure (White Bear Suppression Inventory) was also omitted.

Results and Discussion

Manipulation Checks

I again completed manipulation checks prior to beginning analyses. The manipulation aimed to induce a higher number of specific memories in the Specific condition than the Avoid condition. I created a mixed-effects model predicting number of specific memories with school as a random effect in the first step and condition as a fixed effect in the second step, school $SD = 0.02$, $\chi^2(1) = 116.00$, $p < .001$. As intended, those in the Specific condition ($M = 4.97$, $SD = 1.68$, [4.39, 5.56]) demonstrated a higher number of specific memories than those in the Avoid condition ($M = 0.11$, $SD = 0.32$, [0.003, 0.22], $d = 4.02$, [3.47, 4.13]), $b = 4.86$, $SE_b = 0.28$, $p < .001$. As such, manipulation of retrieval specificity was successful.

I also determined that completing the negative event description successfully induced negative affect. I created a mixed-effects model predicting affect with school

entered as a random effect in the first step and time (i.e., immediately before versus immediately after describing the adverse event) as a fixed effect in the second step, school $SD = 0.14$, $\chi^2(1) = 54.52$, $p < .001$. As intended, the mean affect rating decreased after the child described their adverse event (before description $M = 4.04$, $SD = 0.71$, [3.90, 4.18]; after description $M = 3.20$, $SD = 0.84$, [3.06, 3.37], $d = 1.19$, [1.05, 1.33]), $b = -0.84$, $SE_b = 0.11$, $p < .001$. Hence, describing the negative event induced negative affect.

Hypothesis Testing

I hypothesised an interaction between condition and time (i.e., pre- or post-AMT) on affect. I predicted that those in the Specific condition would demonstrate a decrease in positive affect from pre- to post-AMT. In contrast, those in the Avoid condition would not experience a change in affect from pre- to post-AMT, and the Distraction condition would experience an increase in affect. To test this hypothesis, I created a mixed-effects model predicting affect. As in regression, entering a categorical predictor into a mixed-effects model requires use of a reference group. I used the Distraction condition as the reference group as I was interested in how retrieval strategy influenced affect relative to distracting from the memory. The first step included school as a random effect. Next I added condition and time (i.e., pre- or post-AMT), $\chi^2(3) = 8.91$, $p = .03$. Adding the interaction significantly improved the fit of the model, school $SD = 0.23$, $\chi^2(1) = 11.52$, $p = .003$.

The coefficients for the final model are presented in Table 6. The plotted model (see Figure 3) indicates that the Specific condition demonstrated a decrease in positive affect from pre- to post-AMT. This is reflected in the aggregate descriptive statistics and associated confidence intervals (see Table 5). As predicted by Williams (1996), retrieving specific memories increased distress. I also hypothesised that the Avoid condition would demonstrate negligible change in affect from pre- to post-AMT. As

hypothesised, Figure 3 indicates that the Avoid condition did not experience a substantial change in affect. Again, this is reflected in the aggregate statistics. Confidence intervals on the aggregate means show almost complete overlap, indicating that there is no evidence of meaningful change in affect for that condition. Further, the *d* for the mean difference is small, and the associated confidence interval includes zero, consistent with no significant effect, or at best, a small effect. Hence, my hypothesis was supported. This indicates that avoiding specific memories did minimise negative affect. Finally, I predicted that the Distraction condition would experience an increase in positive affect from pre- to post-AMT. The plotted model is in the predicted direction. Examination of the aggregate statistics indicates no overlap in the confidence intervals around pre- and post-AMT means, consistent with a meaningful effect. Hence, the Distraction condition did demonstrate an increase in positive affect from pre- to post-AMT. This indicates that distracting from the negative memory will improve affect.

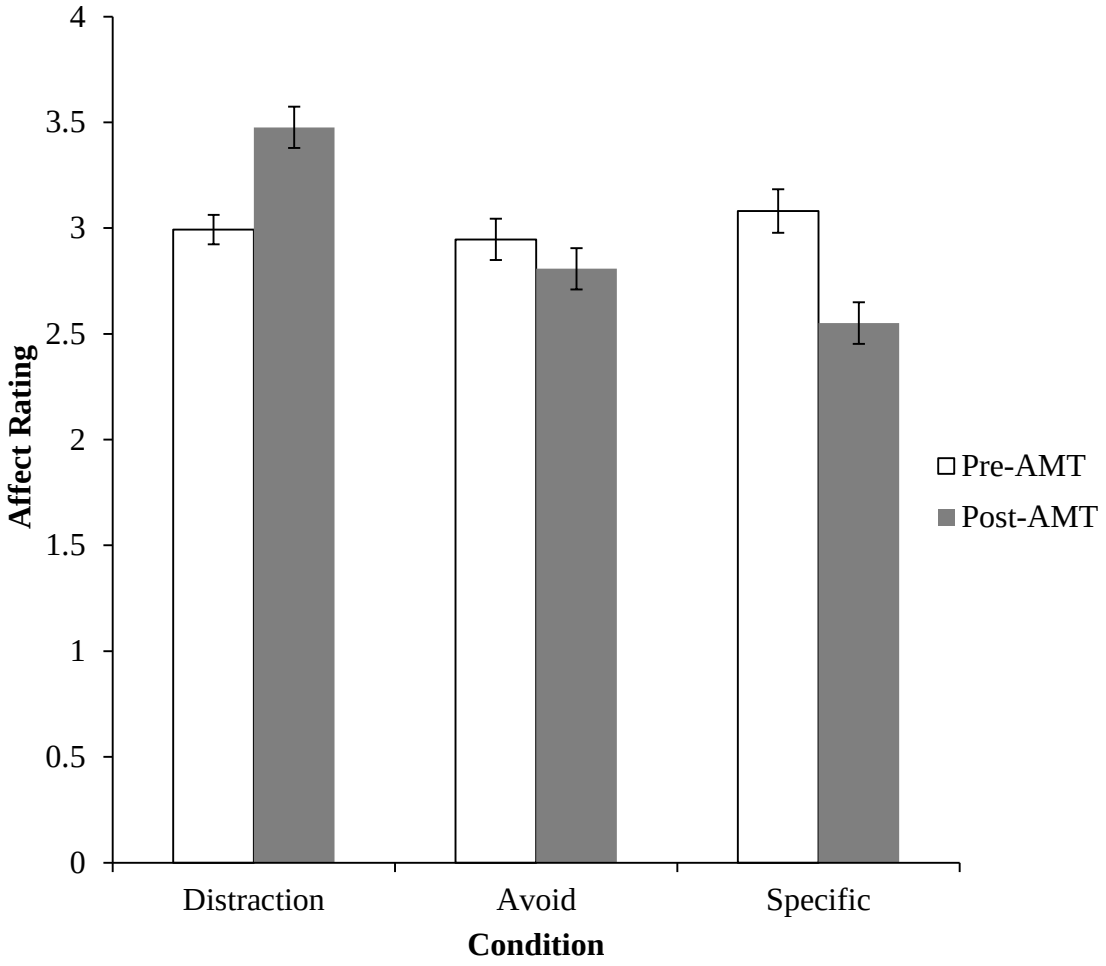

Figure 3. Plotted mixed model for interaction between time and condition on affect rating.[18] Random effect of school was included in the model, *SD* = 0.23. Error bars indicate 95% confidence intervals around the coefficient. Affect rating was on a scale of 1 – 5, where 1 = very sad and 5 = very happy.

[18] Technically, this is predicted affect rating as the mixed-model has predicted affect based on condition and time.

Table 5

Mean (Standard Deviation) Affect Rating for Pre- and Post- Autobiographical Memory Test (AMT) by Condition

		Pre-AMT		Post-AMT		Mean difference	
Condition	*n*	*M* (*SD*)	95% CI	*M* (*SD*)	95% CI	*d*	95% CI
Distraction	31	3.26 (0.77)	[2.97, 3.54]	3.74 (0.68)	[3.49, 3.99]	0.62	[0.35, 0.89]
Avoid	36	3.11 (0.78)	[2.85, 3.38]	2.97 (0.84)	[2.68, 3.26]	-0.18	[-0.08, 0.43]
Specific	34	3.24 (0.96)	[2.90, 3.57]	2.71 (1.22)	[2.28, 3.13]	-0.55	[-0.23, -0.87]

Note. *d* = within-group effect size; CI = Confidence Interval. Affect rating was on a scale of 1 – 5, where 1 = very sad and 5 = very happy.

Table 6

Estimates of Fixed Effects for Mixed-Effects Model Predicting Affect

Parameter	*b*	SE_b	*p*
Intercept	2.99	0.38	< .001
Time	0.48	0.22	.03
Avoid	-0.05	0.21	.83
Specific	0.09	0.21	.68
Avoid × Time	-0.62	0.30	.04
Specific × Time	-1.01	0.30	.001

Note. The Distraction condition was coded as the reference group. Random effect of school *SD* = 0.48.

Conclusion

These findings provide firm support for Williams' (1996) theory of functional avoidance. I have provided evidence that retrieving specific memories of an adverse event will cause children distress by demonstrating that children instructed to be specific experienced a decrease in positive affect. I have also provided evidence that avoidance of specific memories of a negative event will minimise negative affect as those instructed to use an overgeneral retrieval style did not experience substantial change in affect. This experiment has been the first direct examination of the affect regulating properties of overgeneral retrieval in the population that the theory was based on (i.e., children), as previous research has only tested these assumptions with adult samples. This experiment also built upon the findings of the previous experiment by demonstrating that overgeneral retrieval impacts affect differently when used for a particular negative event, as opposed to when this retrieval style is applied indiscriminately to multiple autobiographical events of varying valence. In addition, the meaningful improvement in the Distraction condition suggests that all Study 3 conditions may have increased positive affect due to the AMT distracting from the sad mood induction. The findings from both experiments are now discussed further along with their broader implications.

Discussion

I completed the field's first experimental assessment of functional avoidance in children. This investigation tested two key assumptions of autobiographical memory theory; is retrieval of specific memories distressing for children? And, does avoidance of specific memories result in avoidance of negative affect? The first experiment examined these assumptions in the context of broader overgeneral retrieval, that is, when overgeneral retrieval was used for both positive and negative cues across different events. Contrary to expectations, results demonstrated that retrieval of specific

memories to positive and negative cues resulted in an improvement in affect. Further, there was no evidence that avoiding positive and negative specific memories influenced affect differently from retrieving specific memories, as all conditions experienced an improvement in affect. The second experiment examined these predictions in relation to the retrieval of specific memories of a negative affect inducing event. This provided a more direct assessment of the functional avoidance hypotheses. As predicted, retrieving specific memories of a negative event resulted in a decrease in positive affect, while avoidance of specific memories did not result in a meaningful change in affect. Distracting from the negative event also improved affect. Taken together, the results suggest that children directed to use an overgeneral retrieval style will not experience the decrease in positive affect experienced by children directed to retrieve specific memories. However, this will only occur when retrieving memories of a negative affect inducing event.

These experiments have provided valuable insight into how functional avoidance operates in children. The first experiment suggested that the functional avoidance mechanism does not offer an accurate explanation of overgeneral retrieval once it has generalised to retrieval of all memories. Both the Avoid and Specific conditions experienced an increase in positive affect, which is inconsistent with predictions drawn from the model of how this mechanism operates. A number of alternate explanations for these findings may be considered. One possible explanation is that children employing overgeneral retrieval did avoid negative affect, but that specific retrieval also avoided negative affect, as the AMT consisted of both positive and negative cues. That is, retrieving positive specific memories may have produced strong positive affect, which repaired the negative affect produced by retrieving negative specific memories. However, the Specific condition experienced an increase in positive affect from pre- to post-AMT. Therefore, retrieving both positive and negative

memories did not simply balance each other out, or we would have seen no change in affect. A more probable explanation is that overgeneral and specific retrieval did not influence affect per se, but that the increase in positive affect may be accounted for by distraction. Completing the AMT may have distracted the children from the negative event they described in the mood induction task. I tested this potential explanation in Study 4 by creating a Distraction condition. The Distraction condition demonstrated an increase in positive affect, which indicates that distracting from the mood-inducing event does improve affect. Thus, overgeneral retrieval in response to both positive and negative cues may not minimise negative affect due to avoiding specific memories of a negative event, as predicted by Williams (1996), but engaging in retrieval of other memories may distract from the memory of the negative event. Indeed, distraction has been demonstrated to shorten the duration of negative affect (e.g., Compton, 2000; Nolen-Hoeksema & Morrow, 1993). Together, these results suggest that functional avoidance does not account for overgeneral retrieval when it is used for both positive and negative memory cues.

The second experiment did provide support for the buffering role of functional avoidance in the retrieval of memories of a negative affect inducing event. This study was the first to assess whether specific retrieval of a negative memory influenced affect differently compared to avoiding specific memories of a negative event. The results indicated that retrieval of specific memories of an adverse event was distressing, and that avoiding specific memories of the event will minimise negative affect. Given that distracting from the event memory may improve affect, it was important to determine that encouraging children to be overgeneral did not improve affect by simply distracting from the negative event. However, a different pattern of results was observed between the Distraction and Avoid conditions. The Distraction condition experienced an increase in positive affect while the Avoid condition experienced no significant change. This

suggests that avoiding specific memories has an effect unique from distracting from the memory of the event. Thus, Study 4 has provided the first empirical demonstration of the fundamental assumptions of the effect of functional avoidance on affect following retrieval of adverse childhood memories.

Experimental evidence for these predictions in a non-clinical sample suggests that overgeneral retrieval will influence affect. In this experiment, participants retrieved specific memories of events that were distressing, but were not always traumatic enough to qualify for a diagnosis of PTSD (e.g., dog dying). In addition, the target event occurred a long time ago (on average, 2.39 years previously). Further, the sample was not experiencing PTSD as a result of the event. When considering that the model was proposed to account for functional avoidance in those who had experienced adverse life events such as childhood trauma, the fact that this sample experienced a meaningful decrease in affect despite these factors provides strong evidence for the theory. That is, the observation of an effect in a non-clinical sample who retrieved memories of an adverse, but normal, childhood experience indicates that the effect is robust, and may be stronger in clinical samples retrieving trauma memories. In addition, the effect may be stronger when overgeneral retrieval is used for memories of a more recent event. Indeed, participants indicated that the negative affect inducing event was more distressing right when it occurred, compared with when it was recollected at the time of the experiment. In sum, my findings suggest that engaging in overgeneral retrieval may help a child to manage affect produced by more traumatic memories.

These experiments address what has been until now a significant lack of empirical study of the proposition that childhood trauma increases overgeneral retrieval. Williams (1996) proposed that an overgeneral tendency would develop as a result of functional avoidance of a trauma memory, typically beginning in childhood. Consistent relationships have been observed between childhood trauma and an overgeneral

retrieval style in both child (e.g., de Decker et al., 2003; Valentino et al., 2009) and adult samples (Moore & Zoellner, 2007). However, research has yet to determine that functional avoidance offers an accurate explanation of OGM, and that childhood trauma exposure is likely to increase functional avoidance. My findings suggest that avoiding specific memories of a negative event may regulate affect, thus supporting the functional avoidance explanation of overgeneral retrieval. In addition, in demonstrating that avoiding specific memories of a negative childhood event will minimise negative affect, my findings are consistent with the proposition that childhood trauma may increase a child's use of functional avoidance. As I have provided initial experimental evidence for the aspects of the theory that link childhood trauma to functional avoidance, and how functional avoidance will produce overgeneral retrieval, one critical next step for the field will be to examine longitudinally the process of functional avoidance in children recently exposed to trauma. This will allow determination of whether children naturally avoid specific memories of an adverse event, and will also enable direct examination of the proposed mediational relationship. Completing such a study was beyond the scope of this thesis, but presents an important area for future research.

My findings also have theoretical implications for whether overgeneral retrieval may be adaptive following childhood trauma. Williams (1996) proposed that OGM would be adaptive in the short term following childhood trauma as it would regulate strong negative affect. By demonstrating that avoiding specific memories of an adverse event can regulate negative affect, I have been the first to directly test and support this proposal. Interestingly, Study 1 and Study 2 findings suggested that under certain conditions (e.g., at six months following exposure, or one year following exposure) overgeneral retrieval may be adaptive for posttraumatic adjustment, as it was associated with fewer symptoms of PTSD. These experiments have therefore expanded theoretical

knowledge by indicating that functional avoidance may underlie the adaptive role of overgeneral retrieval following trauma.

My results build on previous research in a number of ways. First, I have explicitly tested whether overgeneral retrieval is a form of functional avoidance. Previous research has studied associations between avoidance and overgeneral retrieval (Kuyken et al., 2006; Stokes et al., 2004). However, this study has been the first to demonstrate that overgeneral retrieval is a form of functional avoidance by providing experimental evidence that overgeneral retrieval will avoid negative affect. In addition, I have expanded theoretical understanding by demonstrating that it is overgenerality to a specific event, as opposed to being overgeneral to multiple events, which appears to regulate negative affect. Previous studies have only examined how overgeneral retrieval related to affect following a negative event, rather than directing participants to be overgeneral in their retrieval of memories of that event (Hermans et al., 2008; Raes et al., 2003). I have built upon these findings by demonstrating that it is what the child is being overgeneral to (i.e., a single event versus multiple experiences) that appears to be a critical factor. As such, these experiments have been important in advancing theoretical understanding of overgeneral retrieval.

These findings also raise a number of questions for future research. A limitation of these experiments is that the task used in the distraction condition may have functioned as a positive mood induction, as we asked children to describe movies or TV shows, which are likely to be entertaining. Replication of this experiment with an alternate reference group may impact results. Further, although the negative mood induction did have a large effect on mood, the level of negative affect experienced by participants was not severe, and the effect of overgeneral retrieval may be different for clinical levels of low mood. Similarly, although I have demonstrated that short term use of overgeneral retrieval has a beneficial effect on mood, I did not explore the long term

consequences of maintaining the retrieval style. This is important to determine as Williams (1996) suggested that overgeneral retrieval will become associated with emotional disturbance if used over the longer term. Indeed, Study 2 results were consistent with this proposal. Hermans et al. (2008) proposed that overgeneral retrieval may become associated with emotional disturbance when not used exclusively for an adverse memory (i.e., when used across wider autobiographical memory). Although Williams (1996) and Hermans et al. (2008) speculate why overgeneral retrieval will become associated with emotional disturbance (e.g., by impairing problem solving), no strong evidence for their speculations has been provided to date.

The experiments presented in this chapter make an important contribution to attempts to distinguish the factors that lead to overgeneral memory being problematic for healthy adjustment. I examined whether broader use of overgeneral retrieval may be maladaptive by impairing ability to repair low mood. However, results indicated that overgeneral retrieval to positive and negative cues improved positive affect, and thus did not limit the ability to repair low mood. Alternate explanations for why overgeneral retrieval becomes maladaptive therefore need to be considered. Previous research has focussed on exploring flexible versus inflexible use of overgeneral retrieval (e.g., Debeer, Raes, Williams, Craeynest, & Hermans, 2013). However, it has yet to be established that inflexible use of overgeneral retrieval will be associated with psychological symptoms any more than flexible use of the style. Williams et al. (2007) also stated that overgeneral retrieval would become maladaptive by impairing problem solving, or ability to imagine the future. Further exploration of how overgeneral retrieval relates to these factors may offer a potential explanation. Establishing the mechanism for change will be an important area for future research.

In conclusion, this chapter has indicated that functional avoidance offers a promising theoretical explanation of overgeneral retrieval in children. To build upon

these findings, future research will need to examine whether children naturally engage in functional avoidance following an adverse event. In addition, further understanding of how overgeneral retrieval may change from an adaptive to maladaptive emotion regulation strategy is needed. Current clinical intervention (e.g., Neshat-Dooet et al., 2013) using memory specificity training aims to reduce overgeneral retrieval in relation to all cues, typically months or years after trauma exposure. However, my findings indicate that overgeneral retrieval may have a temporary benefit on negative affect if used in relation to an adverse memory that has been recently cued. This will need to be considered when developing and refining interventions that seek to modify memory retrieval styles. In addition, further exploration of *exactly how* overgeneral retrieval becomes maladaptive will highlight processes to target in intervention. Hence, advanced theoretical understanding of OGM will shape effective intervention, which may help to reduce the occurrence of emotional disturbance.

CHAPTER 6

General Discussion

This thesis has been innovative in providing the first simultaneous examination of the relationship between OGM, trauma exposure and multiple psychopathologies in childhood. Further, this thesis represents the first time all three mechanisms of the CaR-FA-X model have been evaluated concurrently in childhood OGM. I addressed two key research questions. First, how does OGM relate to psychopathology following childhood trauma exposure? In particular, I explored how OGM related to symptoms of PTSD, and whether this may change over time. It was established in Study 1 (presented in Chapter 3) that the relationship between OGM and PTSD symptoms changed over six months since trauma exposure. Study 2 (presented in Chapter 4) built upon these findings by providing preliminary, cross-sectional evidence that the relationship between OGM and PTSD symptoms may continue to change years after trauma exposure. No evidence was found for a relationship between OGM and depression in any of the studies.

The second research aim was to determine whether the CaR-FA-X mechanisms accounted for childhood OGM. No evidence was found for the CaR-FA-X model as a whole, however, some findings were consistent with predictions drawn from the functional avoidance mechanism regarding how OGM relates to psychological symptoms. Studies 3 and 4 (presented in Chapter 5) provided stronger evidence for the mechanism by demonstrating that functional avoidance of specific memories of an adverse event may regulate negative affect. To conclude this thesis, this chapter will evaluate the evidence provided for each of the research questions. How the findings advance our theoretical understanding of OGM will be outlined, including potential limitations to the CaR-FA-X model in explaining childhood onset OGM. Finally,

recommendations are made for future research that will build upon the findings of this thesis, and ultimately refine clinical intervention for childhood OGM.

How time since trauma exposure influences OGM-symptom relationships

This thesis has provided preliminary evidence that time since trauma exposure is important in the relationship between OGM and child psychopathology. Williams (1996) proposed that OGM will be adaptive for regulating negative affect initially following childhood trauma, but that OGM will become maladaptive if maintained over the longer term. Hence, the theory predicts that OGM will be negatively related to psychopathology immediately post-trauma, but that this relationship will change to become positive with increasing time since trauma exposure. This thesis provided preliminary evidence of this change for symptoms of PTSD. Together, Studies 1 and 2 indicated that time since trauma did significantly moderate the relationship between OGM and PTSD symptoms.

Study 1 findings were consistent with the proposal that OGM will be associated with fewer PTSD symptoms at six months post-trauma. This is consistent with the only other study to examine OGM immediately following trauma, which demonstrated that OGM was associated with lower posttraumatic stress in the first month post-trauma (Nixon, Ball, et al., 2013). Study 2 suggested that OGM may continue to be negatively related to PTSD symptoms up until three years post-trauma. Study 2 also indicated that the relationship may change at approximately six years post-trauma, and that OGM was positively associated with PTSD symptoms by ten years post-trauma. The only previous study to examine OGM at approximately ten years following childhood trauma indicated that OGM was not correlated with symptoms of PTSD (Brennen et al., 2010). This previous examination was also cross-sectional. Importantly, the current study indexed time since exposure for each individual, whereas Brennen et al. recorded all participants as having been exposed to trauma within the range of 3 years old to 7 years

old (i.e., a four year period), as time since exposure was not a key variable in their analyses. Interestingly, my results indicated that the relationship between OGM and PTSD is likely to change over a four-year period. Developmental differences between 3 year olds and 7 year olds are also likely to impact the development of both PTSD symptoms (Salmon & Bryant, 2002) and OGM (Valentino, 2011). The difference in results is therefore likely to be influenced by more precise measurement of time since trauma exposure in the present study, although the relationship between OGM and PTSD may also vary between war trauma (as in Brennen et al., 2010) and more interpersonal trauma such as sexual or physical abuse (as in this thesis). Future research may need to use finer measurement of time since trauma, that is, indexing how many months or years have passed since exposure for each individual, to accurately examine the effect on OGM. In sum, my findings indicated that the relationship between OGM and PTSD symptoms may change from negative to positive over a number of years (and is likely to be non significant at some point during this change). A moderating effect of time may account for the inconsistency in previous literature as to whether OGM is positively related (e.g., Stokes et al., 2004), negatively related (Kuyken et al., 2006; Nixon, Ball, et al., 2013), or unrelated (Brennen et al., 2010; de Decker et al., 2003; Vrielynck et al., 2007) to PTSD symptoms.

While both Study 1 and Study 2 findings were consistent with the proposal that time since trauma moderates the relationship between OGM and PTSD symptoms, the moderating effect observed in Study 2 could possibly be explained by age at exposure. I was unable to separate the effects of time since exposure and age at exposure. As such, it might not be how long it is since trauma exposure, but how old the child was when exposed which influenced the relationship. For example, the longer it has been since trauma exposure, the younger the child was likely to be at exposure (if measurement is taken at one static time point, as done in Study 2). Williams (1996) suggested that

younger age at exposure may increase OGM due to heightened engagement of the functional avoidance mechanism. That is, young children will be more likely to engage in functional avoidance than older children due to younger children having lower ability to independently regulate emotions. This is consistent with findings that children's ability to regulate emotion increases with age (Cole et al., 1994; Stegge & Terwogt, 2007). However, Williams is unclear as to whether age at exposure will influence the relationship between OGM and psychological symptoms. It is possible that age at exposure could have confounded the relationship between OGM and PTSD symptoms. That is, younger age at exposure may have increased both OGM and PTSD symptoms. Research with adult samples has indicated that younger age at the time of childhood trauma exposure is related to greater OGM measured in adulthood (Crane & Duggan, 2009; Hermans et al., 2004). In turn, younger age at exposure may result in more severe psychological symptoms (Crane & Duggan, 2009; Salmon & Bryant, 2002). Thus, I cannot rule out age at exposure as the variable influencing the relationship between OGM and PTSD symptoms. This potential confound and the cross-sectional nature of the analysis impairs the ability to firmly conclude that the relationship does change over years since trauma exposure. However, the results are consistent with the prediction that time since trauma exposure moderates the relationship between OGM and PTSD symptoms.

A key aim of this thesis was to provide the first examination of whether time since childhood trauma impacted the relationship between OGM and psychopathology, particularly PTSD. This aim was achieved. Study 1 was longitudinal and adequately powered, thus I can be confident in findings of the relationship for the first six months following exposure. That is, there was strong evidence that OGM is negatively related to PTSD symptoms at six months post-trauma. As Study 2 was cross-sectional, I cannot draw definitive conclusions about the nature of the OGM-symptom relationship in the

years since trauma exposure. However, in providing evidence, albeit cross-sectional, consistent with the theorised change, this thesis has indicated that further longitudinal research is warranted. Taking into account the caveats described above in relation to the findings of Studies 1 and 2, the fact that a significant interaction was observed between OGM and time since exposure on PTSD symptoms, in relatively small sample sizes, with a mix of severe and relatively low grade traumas indicates that the effect is likely to be robust. As such, this presents an exciting avenue for future longitudinal research.

This thesis indicates a number of directions for future research into the trajectory of OGM following childhood trauma. Future longitudinal exploration of the relationship between OGM and PTSD symptoms will strengthen conclusions drawn from the current cross-sectional data, and improve theoretical understanding. Findings suggest that this longitudinal examination will need to span a number of years (perhaps even up to ten years) to provide an accurate indication of OGM's theorised change. Future research will also need to determine the unique effects of time since exposure and age at exposure on the relationship between OGM and PTSD symptoms, particularly as age at exposure has been found to impact OGM (e.g., Crane & Duggan, 2004; Valentino et al., 2009). Distinction between these effects will be needed before we can accurately determine that the relationship between OGM and psychological symptoms does change, as hypothesised by Williams (1996). This may be achieved by recruiting a larger sample over a wider age range than was recruited in Study 2. Precise measurement of time since trauma exposure for each individual will also be important. Further exploration of the trajectory of the relationship between OGM and psychopathology will also indicate when clinical intervention into memory specificity is optimally timed. Memory specificity training has been completed with adolescents who had been exposed to war approximately nine years previously (although they were currently living in countries of refuge, and as such may have still been experiencing

ongoing trauma; Neshat-Doost et al., 2013). Further understanding of potential changes in the relationship between OGM and psychological symptoms will indicate whether intervention will be more effective if delivered prior to this point. This thesis has demonstrated that OGM was adaptive for PTSD symptoms in the short term. However, exploration of these future directions will build upon the current findings by clarifying how OGM influences long term prognosis for PTSD, along with indicating whether memory specificity training may improve outcomes for trauma-exposed young people.

Although the relationship between OGM and PTSD symptoms was as predicted by Williams (1996), I did not observe a significant relationship between OGM and depression symptoms in trauma-exposed participants. This is in direct contrast to a quite consistent body of research that has demonstrated a positive relationship between depression and OGM, as reviewed in Chapter 1. However, the samples used in these previous studies were not selected based on trauma exposure. That is, the key distinction between this thesis and previous studies that have reported a relationship is that the current participants were all trauma-exposed. Chapter 1 highlighted that the four previous studies reporting no relationship between OGM and depression have used trauma-exposed samples (de Decker et al., 2003; Meesters et al., 2000; Stange et al., 2013; Valentino et al., 2012). Therefore the lack of relationship in this thesis may be due to different relationships between OGM and depression in those with and without significant trauma histories. As participants in the four previous studies and the current studies were trauma-exposed, Williams (1996) would predict that OGM would be adaptive for affect regulation, and unrelated to depression. A non significant relationship shortly following childhood trauma is still consistent with the observed relationship between OGM, childhood trauma and adult onset depression, as OGM will become maladaptive if maintained until adulthood (Williams et al., 2007). Interestingly, there was variation in the time since trauma (i.e., whether trauma had occurred months

or years previously) between the studies presented in this thesis. However, no relationship between OGM and depression was observed in any study, suggesting that time since trauma did not influence the relationship. As such, the lack of relationship in trauma-exposed participants in the present studies may reflect that OGM and symptoms were assessed in a period following childhood trauma when OGM plays a protective role against negative affect, and therefore represents an adaptive processing method.

It is possible that other differences in sample characteristics may have contributed to discrepant findings. Across all studies in this thesis, participants did not demonstrate overly high symptoms of depression as the mean number of depression symptoms was consistently within the normal range. Hipwell et al. (2011) previously highlighted that findings of the relationship between OGM and continuous symptoms of depression are inconsistent relative to findings with diagnosed samples. Indeed, previous studies that reported a relationship between OGM and adolescent depression have primarily used samples with elevated depression symptoms (e.g., Hipwell et al., 2011; Rawal & Rice, 2012). A recent meta-analysis indicated that OGM was a stronger predictor of later depression symptoms in adults with clinical depression diagnoses compared to non-clinical adults (Sumner et al., 2010). As such, the relationship is likely to be stronger in those with clinical levels of depression, and the current participants may not have been depressed enough for a relationship to emerge. This would be consistent with OGM representing a maintenance rather than vulnerability factor for depression. There were not enough participants with clinical levels of depression to directly assess the maintenance explanation in this thesis, but the combination of previous research and the findings reported in this thesis suggest this is a promising explanation.

Similarly, previous reports of OGM predicting depression symptoms have been in at-risk samples, such as those with elevated baseline symptoms (e.g., Hipwell et al.,

2011), or at familial risk (e.g., Rawal & Rice, 2012). Hence, the predictive nature of OGM may be more robust in at-risk populations. For example, the relationship may be stronger in those at familial risk due to having a mother with depression. Valentino (2011) proposed that mother-child reminiscing would play a central role in the development of OGM. If the mother uses an overgeneral retrieval style herself (consistent with OGM being strongly related to depression maintenance in adulthood; Sumner et al., 2010), she may teach the child to use OGM in a chronic manner. In turn, chronic use of OGM has been proposed to increase depression (Hermans et al., 2008; Williams et al., 2007). Thus, the relationship between OGM and depression may be stronger in those at familial risk. This is consistent with findings of a relationship between OGM and depression in adolescents who have a parent with a depression history (Rawal & Rice, 2012). In addition, previous research has indicated that the relationship between OGM and depression is stronger in females than males (Hipwell et al., 2011; Rawal & Rice, 2012). Thus, young women may form a population where OGM is a stronger risk factor. Indeed, women are at greater risk for depression than men, a difference which emerges in early adolescence (Wade, Cairney, & Pevalin, 2002). In sum, the lack of relationship between depression and OGM is consistent with OGM being adaptive following trauma, but could also reflect that the relationship is stronger in at-risk samples or for those with clinical depression.

This thesis was primarily interested in the relationship between OGM and depression in those exposed to childhood trauma, however, the results also offer implications for the relationship in non trauma-exposed samples. In particular, Study 2 provided the first prospective examination of OGM and depression in an adolescent sample that was not at increased risk of depression (compared to Rawal & Rice, 2012, who did use an at-risk sample). This closed an important gap in the literature by providing preliminary data on whether OGM is a causal risk factor or maintenance

factor for depression. If OGM is a causal risk factor, it would be expected that OGM would predict future symptoms. If OGM only maintained depression symptoms, no significant relationship would be expected as the sample was not clinically depressed. Findings indicated that OGM did not predict depression eight months later. These results contribute to accumulating research that demonstrates that OGM maintains depression, rather than representing a pre-existing vulnerability for the disorder (Hipwell et al., 2011; Sumner et al., 2011; Rawal & Rice, 2012). Further prospective research in non-clinical community samples of adolescents will help to clarify the nature of the relationship between OGM and depression, and in turn, highlight samples for which clinical intervention will be most effective.

In light of these findings, theoretical understanding of the relationship between OGM and childhood psychopathology is in need of refinement. This thesis indicated that OGM may relate differently to depression versus PTSD following childhood trauma. Current models (Williams et al., 2007) generally describe poor emotional adjustment as a consequence of OGM, but are not specific as to the exact presentation of the maladjustment (i.e., whether this would present as depression versus PTSD). That is, the current theory does not outline whether depression and PTSD may differ in their relationship to OGM.

A potential explanation for the different relationships observed in the present thesis centres on how the children used OGM. Recent research has begun to explore whether the flexibility of OGM may underlie the relationship between OGM and psychopathology (e.g., Debeer et al., 2013). It is important to remember that Williams (1996) proposed that children would use OGM flexibly initially following trauma, that is, only in response to the trauma memory. However, over time, OGM could become inflexible, such that it was then used for retrieval of memories of events other than the trauma. Each of these uses (i.e., flexible versus inflexible application of OGM) is

proposed to relate differently to symptoms. Overgeneral retrieval of the trauma memory (that is, flexible use of OGM) is thought to assist emotional adjustment (Williams, 1996; Williams et al., 2007). The negative relationship between PTSD and OGM observed in Study 1, and for those recently exposed to trauma in Study 2, is consistent with this proposal. As such, it is possible that children were still using OGM flexibly. Hermans et al. (2008) propose that once OGM is used inflexibly, that is, for other autobiographical memories, it will become associated with depression. Thus, if the current sample was still using OGM flexibly, we would not expect a relationship between OGM and depression. In sum, flexible use of OGM may buffer PTSD symptoms, while inflexible use of OGM may lead to depression.

As the current participants were recently exposed to trauma, OGM may still have been used flexibly, and thus resulted in the negative relationship with PTSD, and non significant relationship with depression. It was unclear whether the current samples were only overgeneral to trauma-related memories in Study 1 and Study 2, or whether they may have also been overgeneral to memories unrelated to the trauma. Future research will need to carefully delineate whether overgeneral retrieval is being applied to both trauma-related and unrelated cues. Although I was unable to examine this in Study 1 and Study 2, Studies 3 and 4 demonstrated that use of overgeneral retrieval for memories of one adverse event may relate to mood differently than use of overgeneral retrieval for a variety of events, consistent with this explanation.

If we consider that inflexible use of OGM will involve overgeneral retrieval for both positive and negative cues, while flexible use will involve overgeneral retrieval for only negative, trauma-related cues, differences in the OGM-depression relationship between different cue valences may also be consistent with this explanation. That is, OGM to positive cues (in addition to negative cues) may reflect inflexible use of OGM whereas OGM to negative cues alone may reflect flexible use. Williams et al. (2007)

calculated effect sizes across eight studies that reported interactions between cue valence and group (depressed or control) in predicting OGM. The effect sizes indicated that there was a larger effect of depressed group (relative to control) on positive cues (d = 0.91) than negative cues (d = 0.48). In an adolescent sample, Hipwell et al. (2011) indicated that it was only OGM to positive cues that was related to current depression symptoms, and that only OGM to positive cues uniquely predicted depression symptoms one year later.

A stronger relationship for positive cues may indicate that depression is more closely related to inflexible OGM, and therefore overgeneral retrieval to both positive and negative cues, while flexible use of OGM in relation to negative (trauma-related) cues alone will not be as strongly related to depression. Indeed, this thesis demonstrated that overgeneral retrieval of a variety of events using both positive and negative cues may impact mood differently than OGM to an adverse event using only negative cues. Thus, the flexibility of OGM may be important in how the style relates to psychological symptoms. Based on the previous literature, and current theoretical understanding (Hermans et al., 2008; Williams, 1996; Williams et al., 2007), flexible use of OGM may be beneficial for PTSD symptoms following recent childhood trauma, while inflexible use of depression may be associated with symptoms of depression, and possibly poorer prognosis for PTSD.

Similarly, OGM following childhood trauma may only be beneficial for symptoms directly related to the trauma (i.e., posttraumatic stress symptoms). For example, OGM may help to minimise re-experiencing or hyperarousal symptoms that are triggered by trauma-related stimuli. That is, specific details of the trauma may act as triggers of re-experiencing or hyperarousal symptoms, therefore avoiding specific memories of the trauma would avoid triggers. This would be consistent with the vicious cycle between trauma memories and these symptom clusters described by cognitive

models of PTSD (e.g., Ehlers & Clark, 2000). Thus, we would expect a negative relationship between OGM and PTSD, and no relationship with depression, as observed. As this thesis has indicated that the relationship between OGM and PTSD symptoms follows a different pattern than the relationship between OGM and depression following childhood trauma, theory will need to be further developed to reflect these different relationships. This may include describing how PTSD symptoms will relate to flexible or inflexible use of OGM, as current theory has only outlined how the flexibility of OGM will relate to depression (Hermans et al., 2008). Potential mechanisms underlying the two different relationships will be later discussed.

In conclusion, this thesis has demonstrated different relationships between OGM and PTSD, and OGM and depression following childhood trauma. Theory will need to be advanced to account for different relationships. A number of potential explanations for these differences have been presented, including the flexibility of OGM use, and that OGM may only benefit trauma-related symptoms. Future directions for research include exploring these potential explanations, or uncovering whether other mechanisms, not identified to date, play a role in different relationships for PTSD and depression. Alternate mechanisms may be drawn from clinical literature by exploring unique factors that have been demonstrated to predict prognosis for each disorder. Other avenues for future research include examining how OGM relates to comorbidity of PTSD and depression. There are likely to be elevated symptoms of both PTSD and depression in trauma-exposed samples. Attempting to recruit samples of children with high PTSD but low depression symptoms, and high PTSD and high depression symptoms may help to untangle some of the underlying mechanisms, although these samples will be difficult to recruit. It may be easier to complete regression or mixed model analyses which allow us to partition unique effects. However, OGM itself may represent a transdiagnostic processes underlying the comorbidity of PTSD and depression. Previous research would

suggest that this is the case, given that OGM is able to predict prognosis for both PTSD and depression in adults (Moore & Zoellner, 2007; Williams et al., 2007). Further exploration of the mechanisms underlying OGM in depression versus PTSD, and whether some of these overlap, may contribute to increased understanding of the frequent comorbidity between PTSD and depression. In turn, this will improve our knowledge of how OGM relates to clinical presentation and prognosis.

The CaR-FA-X Model in Childhood OGM

The second aim of this thesis was to determine whether the CaR-FA-X model accounted for OGM in young people. Overall, the findings indicated that the CaR-FA-X model as a whole did not offer an adequate account of childhood OGM. Williams et al. (2007) outlined three mechanisms through which OGM may occur: capture and rumination, functional avoidance, and reduced executive control. This thesis consistently demonstrated no support for the capture and rumination, and executive control mechanisms. Both Study 1 and Study 2 indicated that OGM was not significantly predicted by rumination, or measures of executive control; namely, inhibition, working memory updating and verbal fluency. Study 1 did indicate that working memory capacity significantly predicted OGM, however, the relationship was in the opposite direction to that predicted by the model. As such, there was no evidence to support the proposed role of the capture and rumination mechanism, and some evidence that contradicted the theorised nature of executive control processes in OGM. None of the mechanisms interacted with trauma exposure to predict OGM. Thus, two of the three CaR-FA-X mechanisms were not supported in this thesis. Conceptually, this indicates that the model does not offer an adequate account of childhood OGM. As the majority of previous support for the model has been found in interaction with depressive symptoms (Raes et al., 2010) or in samples with clinical levels of depression (Park et al., 2004; Valentino et al., 2012), current results suggest that the CaR-FA-X model as a

whole does not explain OGM in trauma-exposed children, nor the non-exposed (and not clinically depressed) participants in this thesis.

There are a number of aspects of methodology that may have led to these findings. Although all measures were objective and standardised, they may not have accurately assessed the CaR-FA-X processes. For example, my measure of inhibition assessed errors in response inhibition. This was due to Williams et al. (2007) describing OGM as occurring due to errors inhibiting a shift of attention during the retrieval process, and inhibition errors being measured in previous OGM research with young people (e.g., Valentino et al., 2012). Measuring the ability to inhibit a shift in attention, as opposed to inhibiting a conditioned response, may result in a different relationship with OGM. There is currently a lack of standardised measures of subtypes of inhibition. That is, measures of inhibition generally assess ability to inhibit a response, rather than ability to inhibit a shift in attention. Therefore the inhibition construct defined by the theory may not have been tapped by the measure used. However, similar measures of inhibition errors have been previously correlated with OGM in adult samples (e.g., Piolino et al., 2010), but not adolescents (e.g., Valentino et al., 2012), as I observed. As such, the non significant relationship is likely to be due to use of paediatric samples, rather than a methodological issue. This is consistent with the argument that aspects of the CaR-FA-X model do not have utility to explaining childhood OGM.

Similarly, I did not measure the capture aspect (i.e., negative self-representations or self-beliefs capturing attention) of the capture and rumination mechanism, although one previous study has indicated this aspect is related to OGM in children (Valentino et al., 2009). As only one aspect of the mechanism was assessed in the present thesis, this limits the ability to firmly conclude that the entire mechanism offers an inadequate description of childhood OGM. In addition, recent research has suggested that the relationship between the CaR-FA-X processes and OGM may only emerge in certain

contexts. For example, Smets et al. (2013) suggested that a relationship between rumination and OGM may only be found if state rumination is activated. However, I assessed trait rumination rather than state rumination, as Williams et al. (2007) discuss how errors in retrieval are more likely to occur in those prone to rumination. My measures of the mechanisms and the retrieval context in which the mechanisms were assessed may therefore have contributed to non significant results. Future research will need to address these concerns before we can confidently refute the utility of the CaR-FA-X model to explain childhood OGM. However, despite the caveats raised above, the current results do provide a pattern of findings that are not supportive of the model.

Although the CaR-FA-X model as a whole was not fully supported, some findings were consistent with the proposed role of functional avoidance in OGM. Study 2 reported that cognitive avoidance was greater in those with trauma histories relative to those without trauma histories, consistent with trauma exposure increasing functional avoidance (Williams, 1996). This builds upon previous support for the mechanism provided by studies demonstrating a greater OGM tendency in trauma-exposed versus non-exposed children (e.g., Brennen et al., 2010; Stokes et al., 2004; Valentino et al., 2009). Further, functional avoidance is proposed to underlie the adaptive nature of OGM following childhood trauma as avoidance of specific memories will regulate negative affect. The findings presented in Study 1 and Study 2 that OGM was potentially adaptive for PTSD symptoms following recent trauma were therefore consistent with functional avoidance. This replicates a previous finding that OGM is negatively related to posttraumatic stress in the first month following childhood trauma (Nixon, Ball, et al., 2013). Most importantly, Study 4 provided empirical evidence that avoidance of specific memories of an adverse event may buffer a child from negative affect, a key assumption of the functional avoidance mechanism. This is consistent with findings in adult samples that OGM can help to regulate negative affect (e.g., Hermans

et al., 2008; Raes et al., 2003). Overgeneral retrieval of an adverse memory being adaptive for emotional management is also consistent with findings that OGM is unrelated to clinical levels of PTSD (Study 1 & Study 2, Brennen et al., 2010; de Decker et al., 2003; Vrielynck et al., 2007) and depression (de Decker et al., 2003; Meesters et al., 2000; Stange et al., 2013; Valentino et al., 2012) in trauma-exposed young people.

This thesis has therefore provided support for how the functional avoidance mechanism operates, and the prediction related to this mechanism, that is, that OGM will be adaptive for emotional adjustment immediately following childhood trauma exposure. Together, these findings offer support for the functional avoidance account of childhood OGM. This thesis has been the first body of research to examine and support the functional avoidance mechanism in children and adolescents. When the current findings are considered in relation to previous research, this thesis has provided a clear indication that functional avoidance is an important mechanism in OGM in young people.

Support for functional avoidance but not the other CaR-FA-X mechanisms may indicate that separate mechanisms of the model operate under different conditions. Study 1 introduced the idea that functional avoidance may drive OGM initially following childhood trauma, and that the other CaR-FA-X mechanisms may become operational at a later point. Indeed, functional avoidance is the only mechanism specifically proposed to emerge in childhood, and Williams et al. (2007) introduced the other mechanisms to account for OGM when functional avoidance was not applicable. Thus, the capture and rumination, and executive control mechanisms may only contribute to OGM in certain contexts. I suggest two conditions in which this may occur. First, the capture and rumination, and executive control mechanisms may not be developmentally appropriate for children, and may only be used in adulthood. This

thesis has highlighted that children may not possess adequate skill in the cognitive processes that underlie these mechanisms. The lack of evidence provided for the overall model in children would suggest that this is likely. Instead of observing the expected interactions with age, this thesis reported that rumination, inhibition, verbal fluency, and working memory measures did not predict OGM at all. This adds to many previous studies that did not support these mechanisms in young samples (de Decker et al., 2003; Kuyken et al., 2006; Nixon, Ball, et al., 2013; Valentino et al., 2012). As such, it would not appear that the age of the child influences the operation of the mechanisms, but rather that the rumination, and executive control mechanisms are not important in childhood OGM. Support for the CaR-FA-X model in adult samples (see Sumner, 2012) indicates that the model may provide an adequate account of OGM during adulthood. As such, achieving adult-like ability in the cognitive processes underlying the mechanisms may see that the capture and rumination, and executive control mechanisms come into operation in mid- to late- adolescence. This would be consistent with studies that have supported the rumination, and inhibition processes of OGM in 18 and 19 year old university students (e.g., Raes, Watkins, Williams, & Hermans, 2008; Sumner, Griffith, & Mineka, 2011; Sutherland & Bryant, 2007).

Second, as alluded earlier, I suggest that different mechanisms may underlie OGM in PTSD than OGM in depression, and that this may be due to flexible use of OGM relating to PTSD, and inflexible use of OGM underlying depression. Previous researchers have indicated that certain CaR-FA-X mechanisms may more accurately account for OGM in one type of psychopathology compared to another (Crane et al., 2007; Sumner, 2012). Support for functional avoidance suggests that it may be a critical mechanism underlying OGM following childhood trauma. That is, functional avoidance may account for the adaptive use of OGM in the initial stages following childhood trauma, and the other CaR-FA-X mechanisms may become active once OGM becomes

maladaptive. Most simply, when OGM is flexible and adaptive, functional avoidance may be the key mechanism underlying the retrieval style. Williams et al. (2007) proposed that functional avoidance underlies the deliberate avoidance of a specific trauma memory in order to regulate strong negative affect. In turn, avoiding intense negative affect produced by a recent trauma memory is thought to be adaptive for posttraumatic emotional adjustment in the short term. Thus, functional avoidance, by definition, accounts for flexible use of OGM, and appears to underlie adaptive use of OGM. In line with this proposal, this thesis demonstrated that functional avoidance was beneficial for mood regulation when used for an adverse memory. At this point, OGM is likely to be beneficial for PTSD symptoms (although as natural recovery occurs, and the emotional intensity of the trauma memory reduces, functional avoidance of the memory may not be as important in emotional management). The lack of support for the capture and rumination, and executive control mechanisms in trauma-exposed young people reported in the current and previous research (e.g., de Decker et al., 2003; Nixon, Ball, et al., 2013; Valentino et al., 2012) would be consistent with these mechanisms not contributing to OGM when it is flexible and adaptive.

Once used inflexibly, OGM is thought to become maladaptive, and associated with symptoms of depression (Hermans et al., 2008). At this point, the other mechanisms of the CaR-FA-X model, that is, executive control, and capture and rumination, may begin to contribute to OGM. There are two aspects of theory that suggest this to be the case. First, both of these mechanisms describe overgeneral retrieval as a result of errors in the attempted retrieval of specific memories. That is, errors in the retrieval of any specific memory will result in OGM. Thus, capture and rumination, and executive control may underlie inflexible use of OGM. Second, theory would suggest that depression may increase activation of these mechanisms. Williams et al. (2007) proposed that depression may increase OGM by impairing executive

control, and that the negative self-beliefs that are evident in depression may see that those who are depressed are more prone to capture errors. This proposal has been supported in previous literature with adult samples (for review see Sumner, 2012). In paediatric samples, this would be consistent with research supporting a relationship between rumination and OGM in adolescents with MDD (Park et al., 2004), and findings that inhibition mediates the relationship between OGM and depression symptoms in primary school children (Raes et al., 2006). As the current samples were not clinically depressed, it is perhaps not entirely surprising that the capture and rumination, or executive control mechanisms did not relate to OGM. Similarly, as Study 2 only had a small number of participants with clinical levels of depression, this may account for non significant interactions between depression and the mechanisms in predicting OGM. Although not all participants were at a clinical level of PTSD symptoms, it is trauma exposure that is proposed to increase activation of the functional avoidance mechanism (rather than symptoms of emotional disturbance, as proposed for the capture and rumination, and executive control mechanisms). Study 2 did not report an interaction between cognitive avoidance and trauma exposure in predicting OGM, however, cognitive avoidance was higher in trauma-exposed participants. This is consistent with trauma exposure increasing the mechanism. As such, it is likely that functional avoidance will occur at subclinical levels of PTSD.

Considering both of these explanations, developmental effects on the capture and rumination, and executive control mechanisms may also underlie why OGM relates differently to posttraumatic stress in the short term following childhood trauma compared to exposure in adulthood. Studies with adult samples with Acute Stress Disorder indicate that OGM is associated with increased symptoms of posttraumatic stress shortly following trauma exposure (Harvey et al., 1998; Kangas, Henry, & Bryant, 2005; Kleim & Ehlers, 2008). This is in contrast to the negative relationship

observed between OGM and posttraumatic stress in children (Study 1 of this thesis; Nixon, Ball, et al., 2013). This may potentially be due to the activation of the capture and rumination, and executive control mechanisms in adult samples. As described earlier, it would appear that childhood trauma will not increase the role of these mechanisms in children's OGM, possibly due to children's ongoing cognitive development. However, these mechanisms may be activated following trauma in adulthood due to adults having fully developed executive control, and more ingrained self-representations and a greater tendency to ruminate compared to children. In turn, activation of these mechanisms may see that OGM is related to poorer outcome (perhaps through the mechanisms' association with inflexible use of OGM), as described above. It is therefore important to note that this thesis draws a distinction between trauma exposure in childhood versus adulthood when considering the mechanisms of OGM and how OGM may relate to psychopathology post-trauma.

Theory (Hermans et al., 2008; Williams, 1996; Williams et al., 2007) and previous research findings therefore suggest that the mechanisms may account for different use of OGM (i.e., flexible and inflexible use), and that in turn, the flexibility of OGM may influence symptoms. In sum, functional avoidance may best explain OGM initially following childhood trauma, and the other CaR-FA-X mechanisms may best describe OGM when it is inflexible and associated with depression. At this point, OGM may also be related to poorer prognosis for PTSD. It is important to recognise that while there is some broad overlap between cognitive models of PTSD and depression, models for both have unique and disorder-specific elements, and as such, separate models may be needed for how OGM relates to each disorder (although both models may simply be variations of the CaR-FA-X model). As previously mentioned, comorbidity of PTSD and depression may see that both sets of mechanisms are activated in an individual. Further exploration of whether separate mechanisms are more important in one

pathology compared to another will advance theoretical understanding of OGM, and may lead to the improvement of current models of OGM.

Theoretical Underpinnings of OGM's Change from Adaptive to Maladaptive

Bringing together the findings on symptoms and OGM mechanisms, this thesis has highlighted the lack of clarity regarding *why* OGM changes from being adaptive to maladaptive. It has not yet been determined what needs to happen (or not happen) for the change to occur. This thesis attempted to close this gap in the literature by exploring the mechanism underlying why the style becomes maladaptive. That is, I began to explore the process through which OGM may lead to increased psychological symptoms. Williams (1996) suggested that an OGM style would become maladaptive if maintained over the longer term. Hermans et al. (2008) expanded this proposal by stating this may be due to OGM becoming inflexible in use. Based on this idea, Study 3 assessed whether overgeneral retrieval may impair ability to repair low mood when used for a variety of autobiographical events. The results did not support this prediction. It has not yet been assessed whether inflexible OGM does indeed relate differently to symptoms compared to flexible OGM. Future research will therefore need to measure whether individuals use OGM in relation to an adverse event memory alone or in relation to wider autobiographical memory when examining the effect of OGM on psychological symptoms. Experimental manipulation of flexible and inflexible use of OGM may also shed light on the mechanism underlying this relationship. Establishing whether inflexible use of OGM leads to psychopathology will be a fundamental area for future research.

Previous research has also highlighted other potential catalysts for change. These include factors that may trigger a dormant relationship between OGM and depression, such as interpersonal stress (e.g., Sumner et al., 2011). Further exploration of the process through which these triggers initiate a positive relationship between

OGM and psychopathology may provide insight into why the style becomes maladaptive. Alternatively, OGM may be part of a host of other avoidant coping strategies, and the use of avoidant coping more generally (i.e., not only in autobiographical retrieval, but also avoidant thinking and problem solving) may lead to psychological symptoms. Indeed, Hermans and colleagues (2005) demonstrated that OGM was related to other types of avoidant coping, such as experiential, cognitive, and behavioural avoidance, although these relationships have yet to be assessed in children. As such, it may be broader use of avoidance across memory retrieval, thoughts, behaviours and problem solving which combine to predict psychological symptoms. This may reflect that for some children, OGM forms the weakest link in the child's use of cognitive processes, as defined by the weakest link hypothesis. Support for the weakest link hypothesis has been found in childhood depression (Abela & Sarin, 2002), however, simultaneous examination of OGM and other cognitive vulnerabilities for childhood psychopathology have not been completed. Further understanding of the mechanisms underlying OGM's theorised change from an adaptive to maladaptive factor will not only advance theory, but also highlight processes we can target in intervention.

Clinical Implications

Finally, this thesis has important implications for clinical intervention into OGM. First, we need to establish how clinical intervention targeting depression symptoms by reducing OGM will impact comorbid PTSD symptoms. This thesis has suggested that OGM may be adaptive for PTSD symptoms immediately post-trauma, and as such, it will be important to determine that interventions do not negatively impact PTSD symptoms. Similarly, my results have indicated that intervention may be best timed a number of years following trauma exposure as OGM may be adaptive for PTSD symptoms prior to this point. In addition, this thesis has highlighted that OGM

interventions for depression may be best targeted to at-risk populations, such as those with subclinical symptoms, or at increased familial risk. Finally, this thesis has suggested that clinical intervention should promote flexible use of OGM. Results indicated that overgeneral retrieval of a negative memory may benefit affect in the short term following retrieval. That is, flexible use of OGM may be beneficial for children when used in response to a negative event memory. Intervention may therefore be best aimed at promoting flexible use of OGM, rather than eradicating the style. This may involve encouraging the child to be specific in their retrieval of positive memories only, rather than focussing on improving specificity to negative cues, or all cues, as current interventions do (e.g., Neshat-Doost et al., 2013). Further consideration of each of these factors will improve the efficacy of intervention.

Conclusion

In conclusion, this thesis has advanced the field in three significant areas. First, my findings have indicated that OGM may be adaptive for mood regulation, and also PTSD symptoms, initially following childhood trauma. At this initial stage, functional avoidance may offer an accurate account of OGM. Second, OGM may become maladaptive if used in the longer term. Results indicated that this may be seen a number of years after exposure, and that at this point OGM may become associated with depression, and possibly poorer prognosis for PTSD. Once maladaptive and associated with depression, the capture and rumination, and executive control mechanisms of the CaR-FA-X model may become operational. Finally, the mechanism underlying OGM's proposed change from adaptive to maladaptive is unclear. However, theory and previous findings suggest that this may occur due to OGM generalising from flexible use in response to a trauma memory, to inflexible use, across wider autobiographical memory. Further research on the trajectory of OGM following childhood trauma, and the cognitive processes underlying the retrieval style will improve the efficacy of

emerging clinical intervention. Ultimately, further understanding of OGM may help to reduce the impact and severity of psychopathology following childhood trauma.

References

Abela, J. R. Z., & Sarin, S. (2002). Cognitive vulnerability to hopelessness depression: A chain is only as strong as its weakest link. *Cognitive Therapy and Research, 26*, 811-829. doi:10.1023/A:1021245618183

Abela, J. R. Z., Vanderbilt, E., & Rochon, A. (2004). A test of the integration of the response styles and social support theories of depression in third and seventh grade children. *Journal of Social and Clinical Psychology, 23*, 653-674. doi:10.1521/jscp.23.5.653.50752

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th, text revision ed.). Washington, DC: Author.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Australian Bureau of Statistics. (2011). Greater Adelaide Ancestry by Birthplace of Parents Retrieved 28 February 2013, from www.censusdata.abs.gov.au/census_services/getproduct/census/2011/communityprofile/4GADE

Baayen, R. H., Davidson, D. J., & Bates, D. M. (2008). Mixed-effects modeling with crossed random effects for subjects and items. *Journal of Memory and Language, 59*, 390-412. doi:10.1016/j.jml.2007.12.005

Bates, D. M., Maechler, M., & Bolker, B. (2011). lme4: Linear mixed-effects models using S4 classes. R package version 0.999375-41 from http://CRAN.R-project.org/package=lme4

Beck, A. T., & Steer, R. A. (1993). *Beck Depression Inventory: Manual*. Orlando, FL: Harcourt Brace.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *The Beck Depression Inventory-Second Edition.* San Antonio, TX: The Psychological Corporation.

Birleson, P. (1981). The validity of depression disorder in childhood and the development of a self-rating scale: A research report. *Journal of Child Psychology and Psychiatry, 22*, 73–88. doi:10.1111/j.1469-7610.1981.tb00533.x

Brennen, T., Hasanovi, M., Zotovi, M., Blix, I., Skar, A.-M. S., Preli, N. K., et al. (2010). Trauma exposure in childhood impairs the ability to recall specific autobiographical memories in late adolescence. *Journal of Traumatic Stress, 23*, 240-247. doi:10.1002/jts.20513

Brocki, K., Eninger, L., Thorell, L., & Bohlin, G. (2010). Interrelations between executive function and symptoms of hyperactivity/Impulsivity and inattention in preschoolers: A two year longitudinal study. *Journal of Abnormal Child Psychology, 38*, 163-171. doi:10.1007/s10802-009-9354-9

Broderick, P. C. (1998). Early adolescent gender differences in the use of ruminative and distracting coping strategies. *Journal of Early Adolescence, 18*, 173-191. doi:10.1177/0272431698018002003

Cohen, J. (1988). *Statistical Power for the Behavioural Sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Cole, P. M., Michel, M. K., & Teti, L. O. D. (1994). The development of emotion regulation and dysregulation: A clinical perspective. *Monographs of the Society for Research in Child Development, 59*, 73-102. doi:10.1111/j.1540-5834.1994.tb01278.x

Compton, R. J. (2000). Ability to disengage attention predicts negative affect. *Cognition & Emotion, 14*, 401-415. doi:10.1080/026999300378897

Conway, M. A., & Pleydell-Pearce, C. W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review, 107*, 261-288. doi:10.1037/0033-295X.2.261

Crane, C., Barnhofer, T., Visser, C., Nightingale, H., & Williams, J. M. G. (2007). The effects of analytical and experiential rumination on autobiographical memory specificity in individuals with a history of major depression. *Behaviour Research and Therapy, 45*, 3077-3087. doi:10.1016/j.brat.2007.05.009

Crane, C., & Duggan, D. S. (2009). Overgeneral autobiographical memory and age of onset of childhood sexual abuse in patients with recurrent suicidal behaviour. *British Journal of Clinical Psychology, 48*, 93-100. doi:10.1348/014466508x370600

Crone, E. A., Wendelken, C., Donohue, S., van Leijenhorst, L., & Bunge, S. A. (2006). Neurocognitive development of the ability to manipulate information in working memory. *Proceedings of the National Academy of Sciences, 103*, 9315-9320. doi:10.1073/pnas.0510088103

Dalgleish, T., Rolfe, J., Golden, A.-M., Dunn, B. D., & Barnard, P. J. (2008). Reduced autobiographical memory specificity and posttraumatic stress: Exploring the contributions of impaired executive control and affect regulation. *Journal of Abnormal Psychology, 117*, 236-241.

Dalgleish, T., Williams, J. M. G., Golden, A., Perkins, N., Barrett, L., Barnard, P. J., et al. (2007). Reduced specificity of autobiographical memory and depression: The role of executive control. *Journal of Experimental Psychology: General, 136*, 23-42. doi:10.1037/0096-3445.136.1.23

Daviss, W. B., Racusin, R., Fleischer, A., Mooney, D., Ford, J. D., & McHugo, G. J. (2000). Acute stress disorder symptomatology during hospitalization for

pediatric injury. *Journal of the American Academy of Child & Adolescent Psychiatry, 39*, 569-575. doi:10.1097/00004583-200005000-00010

de Decker, A., Hermans, D., Raes, F., & Eelen, P. (2003). Autobiographical memory specificity and trauma in inpatient adolescents. *Journal of Clinical and Adolescent Psychology, 32*, 22-31. doi:10.1207/15374420360533031

Debeer, E., Hermans, D., & Raes, F. (2009). Associations between components of rumination and autobiographical memory specificity as measured by a Minimal Instructions Autobiographical Memory Test. *Memory, 17*, 892-903. doi:10.1080/09658210903376243

Debeer, E., Raes, F., Claes, S., Vrieze, E., Williams, J. M. G., & Hermans, D. (2011). Context-dependent activation of reduced autobiographical memory specificity as an avoidant coping style. *Emotion, 11*, 1500-1506. doi:10.1037/a0024535

Debeer, E., Raes, F., Williams, J. M. G., Craeynest, M., & Hermans, D. (2013). Operant conditioning of autobiographical memory retrieval. *Memory*. doi:10.1080/09658211.2013.774419

Drummond, L. E., 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 & Emotion, 20*, 488-505.

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38*, 319-345. doi:10.1016/S0005-7967(99)00123-0

Ellis, L. K., & Rothbart, M. K. (2001). *Revision of the Early Adolescent Temperament Questionnaire.* Paper presented at the Biennial Meeting of the Society for Research in Child Development, Minneapolis, Minnesota.

Field, A., Miles, J., & Field, Z. (2012). *Discovering Statistics Using R*. London, UK: Sage Publications Limited.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1995). *Structured clinical interview for DSM–IV. Axis I disorder with psychotic screen*. New York: Psychiatric Institute.

Foa, E. B., Johnson, K. M., Feeny, N. C., & Treadwell, K. R. H. (2001). The child PTSD symptom scale: A preliminary examination of its psychometric properties. *Journal of Clinical Child Psychology, 30,* 376-384. doi:10.1207/S15374424JCCP3003_9

Friedman, N. P., & Miyake, A. (2004). The relations among inhibition and interference control functions: a latent-variable analysis. *Journal of Experimental Psychology: General*, *133*(1), 101-135.

Gathercole, S.E. (1998). The Development of Memory. *The Journal of Child Psychology and Psychiatry and Allied Disciplines, 39*, 3-27. doi: 10.1017/S0021963097001753

Gathercole, S. E. (2004). The structure of working memory from 4 to 15 years of age. *Developmental psychology, 40,* 177-190. doi:10.1037/0012-1649.40.2.177

Gelman, A., & Hill, J. (2007). *Data Analysis Using Regression and Multilevel/Heirarchical Models*. New York, NY: Cambridge University Press.

Geurts, H., van der Oord, S., & Crone, E. (2006). Hot and cool aspects of cognitive control in children with ADHD: Decision-making and inhibition. *Journal of Abnormal Child Psychology, 34*, 811-822. doi:10.1007/s10802-006-9059-2

Goodyer, I. M., Herbert, J., Tamplin, A., Secher, S. M., & Pearson, J. (1997). Short term outcome of M.D.II, life events, family dysfunction and friendship differences as predictors of persistent disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 474-480.

Griffith, J. W., Sumner, J. A., Raes, F., Barnhofer, T., Debeer, E., & Hermans, D. (2012). Current psychometric and methodological issues in the measurement of

overgeneral autobiographical memory. *Journal of Behavior Therapy and Experimental Psychiatry, 43*, S21-S31. doi:10.1016/j.jbtep.2011.05.008

Hale, S., Bronik, M. D., & Fry, A. F. (1997). Verbal and spatial working memory in school-age children: Developmental differences in susceptibility to interference. *Developmental Psychology, 33*, 364-371. doi:10.1037/0012-1649.33.2.364

Hamilton, M. (1967). Development of a rating scale for primary depressive illness. *British Journal of Social Clinical Psychology, 6*, 278-296. doi:10.1111/j.2044-8260.1967.tb00530.x

Hampel, P., & Petermann, F. (2005). Age and gender effects on coping in children and adolescents. *Journal of Youth and Adolescence, 34*, 73-83. doi:10.1007/s10964-005-3207-9

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. doi:10.1037/0022-006X.66.3.500

Hermans, D., de Decker, A., de Peuter, S., Raes, F., Eelen, P., & Williams, J. M. G. (2008). Autobiographical memory specificity and affect regulation: Coping with a negative life event. *Depression and Anxiety, 25*, 787-792. doi:10.1002/da.20326

Hermans, D., Defranc, A., Raes, F., Williams, J. M. G., & Eelen, P. (2005). Reduced autobiographical memory specificity as an avoidant coping style. *British Journal of Clinical Psychology, 44*, 583-589. doi:10.1348/014466505X53461

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. doi:10.1016/S0005-7967(03)00197-9

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*, 300-320. doi: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*, 254-265. doi:10.1080/15374416.2011.546037

Hitchcock, C., Nixon, R.D.V., & Weber, N. (in press). A longitudinal examination of overgeneral memory and psychopathology in children following recent trauma exposure. *Applied Cognitive Psychology.*

Hitchcock, C., Nixon, R. D. V., & Weber, N. (2013). A review of overgeneral memory in child psychopathology. *British Journal of Clinical Psychology*. doi:10.1111/bjc.12034

Horowitz, M., Wilner, N., & Alvarez, W. (1979). Impact of Event Scale: A measure of subjective stress. *Psychosomatic Medicine, 41*, 209-218.

Howe, M.L., Toth, S.L., & Cicchetti, D. (2006). Memory and developmental psychopathology. In D. Cicchetti & D. J. Cohen (Eds.), Developmental psychopathology: Developmental neuroscience (pp. 629-655). Hoboken: John Wiley and Sons.

Johnson, R. J., Follmer Greenhoot, A., Glisky, E., & McCloskey, L. A. (2005). The relations among abuse, depression, and adolescents' autobiographical memory. *Journal of Clinical and Adolescent Psychology, 34*, 235-247. doi:10.1207/s15374424jccp3402_3

Joormann, J., Gilbert, K., & Gotlib, I. H. (2010). Emotion identification in girls at high risk for depression. *Journal of Child Psychology and Psychiatry, 51*, 575-582. doi:10.1111/j.1469-7610.2009.02175.x

Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of Abnormal Psychology, 116*, 135-143.

Jose, P., & Brown, I. (2008). When does the gender difference in rumination begin? Gender and age differences in the use of rumination by adolescents. *Journal of Youth and Adolescence, 37*, 180-192. doi:10.1007/s10964-006-9166-y

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. doi:10.1037/0022-066X.73.2.293

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 (KSADSPL): Initial reliability and validity data. *Journal of the American Academy of Child & Adolescent Psychiatry, 36*, 980-988. doi:10.1097/00004583-199707000-00021

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. doi:10.1037/0022-006X.71.4.692

Kleim, B., & Ehlers, A. (2008). Reduced autobiographical memory specificity predicts depression and posttraumatic stress disorder after recent trauma. *Journal of*

Consulting and Clinical Psychology, 76, 231-242. doi:10.1037/0022-006X.76.2.231

Kovacs, M. (1992). *The Children's Depression Inventory (CDI)*. Tonawanda, NY: Multi-Health Systems.

Kuyken, W., & Dalgleish, T. (2011). Overgeneral autogiographical memory in adolescents at risk for depression. *Memory, 19*, 241-250. doi:10.1080/09658211.2011.554421

Kuyken, W., Howell, R., & Dalgleish, T. (2006). Overgeneral autobiographical memory in depressed adolescents with, versus without, a reported history of trauma. *Journal of Abnormal Psychology, 115*, 387-396. doi:10.1037/0021-843X.115.3.387

Levin, H. S., Culhane, K. A., Hartmann, J., Evankovich, K., Mattson, A. J., Harward, H., et al. (1991). Developmental changes in performance on tests of purported frontal lobe functioning. *Developmental Neuropsychology, 7*, 377-395. doi:10.1080/87565649109540499

Manly, T., Anderson, V., Nimmo-Smith, I., Turner, A., Watson, P., & Robertson, I. H. (2001). The differential assessment of children's attention: The Test of Everyday Attention for Children (TEA-Ch), normative sample and ADHD performance. *The Journal of Child Psychology and Psychiatry and Allied Disciplines, 42*, 1065-1081. doi:10.1017/S0021963001007909

Manly, T., Robertson, I. H., Anderson, V., & Nimmo-Smith, I. (1999). *TEA-Ch: the test of everyday attention for children*: Thames Valley Test Company Bury St. Edmunds, England.

McEvoy, P. M., Mahoney, A. E. J., & Moulds, M. L. (2010). Are worry, rumination, and post-event processing one and the same?: Development of the repetitive

thinking questionnaire. *Journal of Anxiety Disorders, 24*, 509-519. doi:10.1016/j.janxdis.2010.03.008

McKinnon, A., Nixon, R. D. V., & Brewer, N. (2008). The influence of data-driven processing on perceptions of memory quality and intrusive symptoms in children following traumatic events. *Behaviour Research and Therapy, 46*, 766-775. doi:10.1016/j.brat.2008.02.008

Meesters, C., Merckelbach, H., Muris, P., & Wessel, I. (2000). Autobiographical memory and trauma in adolescents. *Journal of Behavior Therapy and Experimental Psychiatry, 31*, 29-39. doi:10.1016/S0005-7916(00)00006-9

Meiser-Stedman, R. (2002). Towards a cognitive–behavioral model of PTSD in children and adolescents. *Clinical Child and Family Psychology Review, 5*, 217-232. doi:10.1023/A:1020982122107

Moore, S. A., & Zoellner, L. A. (2007). Overgeneral autobiographical memory and traumatic events: An evaluative review. *Psychological Bulletin, 133*, 419-437. doi:10.1037/0033-2909.133.3.419

Nader, K., Kriegler, J. A., Blake, D. D., Pynoos, R. S., Newman, E., & Weather, F. W. (1996). *Clinician Administered PTSD Scale, Child and Adolescent Version*. White River Junction, VT: National Center for PTSD.

Nelson, K., & Gruendel, J. (Eds.). (1981). *General event representations: Basic building blocks of cognitive development*. Hillsdale, NJ: Erlbaum.

Neshat-Doost, H. T., Dalgleish, T., Yule, W., Kalantari, M., Ahmadi, S. J., Dyregrov, A., et al. (2013). Enhancing autobiographical memory specificity through cognitive training: An intervention for depression translated from basic science. *Clinical Psychological Science, 1*, 84-92. doi:10.1177/2167702612454613

Nixon, R. D. V., Ball, S.-A., Sterk, J., Best, T., & Beatty, L. (2013). Autobiographical memory in children and adolescents with acute stress and chronic posttraumatic stress disorder. *Behaviour Change, 30*, 180-198. doi:10.1017/BEC.2013.17

Nixon, R. D. V., Meiser-Stedman, R., Dalgleish, T., Yule, W., Clark, D. M., Perrin, S., et al. (2013). The Child PTSD Symptom Scale: An update and replication of its psychometric properties. *Psychological Assessment*. doi:10.1037/a0033324

Nolen-Hoeksema, S., & Morrow, J. (1993). Effects of rumination and distraction on naturally occurring depressed mood. *Cognition & Emotion, 7*, 561-570. doi:10.1080/02699939308409206

Orbach, Y., Lamb, M. E., Sternberg, K. J., Williams, J. M. G., & Dawud-Noursi, S. (2001). The effect of being a victim or witness of family violence on the retrieval of autobiographical memories. *Child Abuse & Neglect, 25*, 1427-1437. doi:10.1016/S0145-2134(01)00283-6

Park, R. J., Goodyer, I., & Teasdale, J. (2002). Categoric overgeneral autobiographical memory in adolescents with major depressive disorder. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences, 32*, 267-276. doi:10.1017/S0033291701005189

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. doi:10.1111/j.1469-7610.2004.t01-1-00291.x

Philippot, P., Schaefer, A., & Herbette, G. (2003). Consequences of specific processing of emotional information: Impact of general versus specific autobiographical memory priming on emotion elicitation. *Emotion, 3*, 270-283.

Phipps, S., Fairclough, D., & Mulhern, R. K. (1995). Avoidant coping in children with cancer. *Journal of Pediatric Psychology, 20*, 217-232. doi:10.1093/jpepsy/20.2.217

Piolino, P., Coste, C., Martinelli, P., Macé, A.-L., Quinette, P., Guillery-Girard, B., et al. (2010). Reduced specificity of autobiographical memory and aging: Do the executive and feature binding functions of working memory have a role? *Neuropsychologia, 48*, 429-440. doi:10.1016/j.neuropsychologia.2009.09.035

Pynoos, R. S., Frederick, C., Nader, K., Arroyo, W., Steinberg, A., Eth, S., et al. (1987). Life threat and posttraumatic stress in school-age children. *Archives of General Psychiatry, 44*, 1057-1063. doi:10.1001/archpsyc.1987.01800240031005

R Development Core Team. (2011). *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401. doi:10.1177/014662167700100306

Raes, F., Hermans, D., de Decker, A., Eelen, P., & Williams, J. M. G. (2003). Autobiographical memory specificity and affect regulation: An experimental approach. *Emotion, 3*, 201-206. doi:10.1037/1528-3542.3.2.201

Raes, F., Hermans, D., Williams, J. M. G., & Eelen, P. (2006). Reduced autobiographical memory specificity and affect regulation. *Cognition & Emotion, 20*, 402-429. doi:10.1080/02699930500341003

Raes, F., Verstraeten, K., Bijttebier, P., Vasey, M. W., & Dalgleish, T. (2010). Inhibitory control mediates the relationship between depressed mood and overgeneral memory recall in children. *Journal of Clinical Child and Adolescent Psychology, 39*, 276-281. doi:10.1080/15374410903532684

Raes, F., Watkins, E. R., Williams, J. M. G., & Hermans, D. (2008). Non-ruminative processing reduces overgeneral autobiographical memory retrieval in students. *Behaviour Research and Therapy, 46*, 748-756. doi:10.1016/j.brat.2008.03.003

Rawal, A., & Rice, F. (2012). Examining overgeneral autobiographical memory as a risk factor for adolescent depression. *Journal of the American Academy of Child & Adolescent Psychiatry, 51*, 518-527.

Romens, S. E., & Pollak, S. D. (2012). Emotion regulation predicts attention bias in maltreated children at-risk for depression. *Journal of Child Psychology and Psychiatry, 53*, 120-127. doi:10.1111/j.1469-7610.2011.02474.x

Rosenthal, R., & Rosnow, R. (2008). *Essentials of Behavioural Research: Methods and Data Analysis* (Third ed.). New York, NY: McGraw-Hill.

Salmon, K., & Bryant, R. A. (2002). Posttraumatic stress disorder in children: The influence of developmental factors. *Clinical Psychology Review, 22*, 163-188. doi:10.1016/S0272-7358(01)00086-1

Sexton, K. A., & Dugas, M. J. (2008). The Cognitive Avoidance Questionnaire: Validation of the English translation. *Journal of Anxiety Disorders, 22*, 355-370. doi:10.1016/j.janxdis.2007.04.005

Smets, J., Griffith, J. W., Wessel, I., Walschaerts, D., & Raes, F. (2013). Depressive symptoms moderate the effects of a self-discrepancy induction on overgeneral autobiographical memory. *Memory, 21*, 751-761. doi:10.1080/09658211.2012.756039

Smith, P., Perrin, S., Dyregrov, A., & Yule, W. (2003). Principal components analysis of the impact of event scale with children in war. *Personality and Individual Differences, 34*, 315-322. doi:10.1016/S0191-8869(02)00047-8

Spence, S. H. (1998). A measure of anxiety symptoms among children. *Behaviour Research and Therapy, 36*, 545-566. doi:10.1016/S0005-7967(98)00034-5

Stange, J. P., Hamlat, E. J., Hamilton, J. L., Abramson, L. Y., & Alloy, L. B. (2013). Overgeneral autobiographical memory, emotional maltreatment, and depressive symptoms in adolescence: Evidence of a cognitive vulnerability–stress interaction. *Journal of Adolescence, 36,* 201-208. doi:10.1016/j.adolescence.2012.11.001

Stegge, H., & Terwogt, M. M. (2007). Awareness and regulation of emotion in typical and atypical development. In J. J. Gross (Ed.), *Handbook of Emotion Regulation* (pp. 269-286). New York: Guilford Press.

Stokes, D. J., Dritschel, B. H., & Bekerian, D. A. (2004). The effect of burn injury on adolescents autobiographical memory. *Behaviour Research and Therapy, 42,* 1357-1365. doi: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, 32,* 34-48. doi: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,* 614-625. doi:10.1016/j.brat.2010.03.013

Sumner, J. A., Griffith, J. W., & Mineka, S. (2011). Examining the mechanisms of overgeneral autobiographical memory: Capture and rumination, and impaired executive control. *Memory, 19,* 169-183. doi:10.1080/09658211.2010.541467

Sumner, J. A., Griffith, J. W., Mineka, S., Newcomb Rekart, K., Zinbarg, R., & Craske, M. (2011). Overgeneral autobiographical memory and chronic interpersonal stress as predictors of the course of depression in adolescents. *Cognition & Emotion, 25,* 183-192. doi:10.1080/02699931003741566

Sutherland, K., & Bryant, R. A. (2007). Rumination and overgeneral autobiographical memory. *Behaviour Research and Therapy, 45*, 2407-2416. doi:10.1016/j.brat.2007.03.018

Swales, M. A., Williams, J. M. G., & Wood, P. (2001). Specificity of autobiographical memory and mood disturbance in adolescents. *Cognition & Emotion, 15*, 321 - 331.

Taylor, L., & Ingram, R. E. (1999). Cognitive reactivity and depressotypic information processing in children of depressed mothers. *Journal of Abnormal Psychology, 108*, 202. doi:10.1037/0021-843X.108.2.202

Tombaugh, T. N., Kozak, J., & Rees, L. (1999). Normative data stratified by age and education for two measures of verbal fluency: FAS and Animal Naming. *Archives of Clinical Neuropsychology, 14*, 167-177. doi:10.1016/S0887-6177(97)00095-4

Unsworth, N., & Engle, R.W. (2007). On the division of short-term and working memory: An examination of simple and complex span and their relation to higher order abilities. *Psychological Bulletin, 133*, 1038-1066. doi: 10.1037/0033-2909.133.6.1038

Valentino, K. (2011). A developmental psychopathology model of overgeneral autobiographical memory. *Developmental Review, 31*, 32-54. doi:10.1016/j.dr.2011.05.001

Valentino, K., Bridgett, D. J., Hayden, L. C., & Nuttall, A. K. (2012). Abuse, depressive symptoms, executive functioning, and overgeneral memory among a psychiatric sample of children and adolescents. *Journal of Clinical Child & Adolescent Psychology, 41*, 491-498. doi:10.1080/15374416.2012.660689

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, 50*, 1029-1038. doi:10.1111/j.1469-7610.2009.02072

Vincken, M. J. B., Meesters, C., Engelhard, I. M., & Schouten, E. (2012). Psychometric qualities of the White Bear Suppression Inventory in a Dutch sample of children and adolescents. *Personality and Individual Differences, 52*, 301-305. doi:10.1016/j.paid.2011.10.023

Vrielynck, N., Delpus, S., & Philippot, P. (2007). Overgeneral autobiographical memory and depressive disorder in children. *Journal of Clinical and Adolescent Psychology, 36*, 95-105. doi:10.1080/15374410709336572

Vuontela, V., Steenari, M.-R., Carlson, S., Koivisto, J., Fjällberg, M., & Aronen, E. T. (2003). Audiospatial and visuospatial working memory in 6–13 year old school children. *Learning & Memory, 10*, 74-81. doi:10.1101/lm.53503

Wade, T. J., Cairney, J., & Pevalin, D. J. (2002). Emergence of gender differences in depression during adolescence: National panel results from three countries. *Journal of the American Academy of Child & Adolescent Psychiatry, 41*, 190-198. doi:10.1097/00004583-200202000-00013

Watkins, E., & Teasdale, J. (2001). Rumination and overgeneral memory in depression: Effects of self-focus and analytic thinking. *Journal of Abnormal Psychology, 110*, 353-357. doi:10.1037/0021-843X.110.2.333

Wechsler, D. (2003). *Wechsler Intelligence Scale for Children- Fourth Edition (WISC-IV) Administration and Scoring Manual*. San Antonio, TX: The Psychological Corporation.

Wegner, D. M., & Zanakos, S. (1994). Chronic thought suppression. *Journal of Personality, 62*, 615-640. doi:10.1111/j.1467-6494.1994.tb00311.x

Weiss, D. S., & Marmar, C. R. (1997). The Impact of Event Scale-Revised. In J. P. Wilson & T. M. Keane (Eds.), *Assessing Psychological Trauma and PTSD* (pp. 399-411). New York: Guilford Press.

Welsh, M. C., Pennington, B. F., & Groisser, D. B. (1991). A normative-developmental study of executive function: A window on prefrontal function in children. *Developmental Neuropsychology, 7*, 131 - 149. doi:10.1080/87565649109540483

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., Barnhofer, T., Crane, C., Herman, D., Raes, F., Watkins, E., et al. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin, 133*, 122-148. doi:10.1037/0033-2909.133.1.122

Williams, J. M. G., & Broadbent, K. (1986). Autobiographical memory in suicide attempters. *Journal of Abnormal Psychology, 95*, 144-149. doi:10.1037/0021-843X.95.2.144

Williams, J. M. G., Eelen, P., Raes, F., & Hermans, D. (2006). Reduced autobiographical memory specificity and affect regulation. *Cognition & Emotion, 20*, 402-429. doi:10.1080/02699930500341003

Ziegert, D. I., & Kistner, J. A. (2002). Response styles theory: Downward extension to children. *Journal of Clinical and Adolescent Psychology, 31*, 325-334. doi:10.1207/S15374424JCCP3103_04

Appendix A

Study 1 Cue Words for Autobiographical Memory Test

Table A1

Cue Words for Time 1, Time 2 and Time 3, in Order of Presentation

Time 1	Time 2	Time 3
Happy	Happy	Happy
Sad	Sad	Sad
Easy	Friend	Beautiful
Lonely	Stupid	Hurt
Proud	Surprised	Safe
Scared	Tears	Sorry
Brave	Smart	Lucky
Angry	Mad	Upset
Successful	Playing	Interested
Broken	Afraid	Alone

Appendix B

Study 2 Cue Words for Autobiographical Memory Test

Table B1

Cue Words for Time 1 and Time 2, in Order of Presentation

Time 1	Time 2
Happy	Happy
Sad	Sad
Easy	Friend
Lonely	Stupid
Proud	Surprised
Scared	Tears
Brave	Smart
Angry	Mad
Successful	Playing
Broken	Afraid

Appendix C

Study 3 Autobiographical Memory Test Instructions by Retrieval Condition

Avoid Condition

For each of the following words we would like you to think of a memory. The memory could have happened recently (e.g., yesterday, last week) or a long time ago. It might be something important, or not so important. But we would like you to avoid thinking about a specific event, or one particular time that something happened. So if we said the word ‘‘good’’ it would not be OK to say, ‘‘I had a good time at Jane’s party” because this is a specific event. But it would be OK to say, ‘‘I always enjoy a good party’’ because this is not a specific event. Another example is if we said the word “amazing” you could say “I always get to do amazing things on my holidays.” We don’t need to know exactly what the amazing thing is that you did, just that stuff you do on holidays is amazing. You could also write about something you do that makes you feel like the word. For example, for the word “excited” you could say “I feel excited when I have a friend sleep over at my house”. So we would like to know about things that happen lots of times, not something that happened one time.

Specific Condition

We are interested in your memory for events that have happened in your life. For each of the following words we would like you to think of an event that happened to you which the word reminds you of. The event could have happened recently (e.g., yesterday, last week) or a long time ago. It might be an important event, or a not so important event. However, the memory you recall should be a specific event. So if we said the word ‘‘good’’ it would not be OK to say, ‘‘I always enjoy a good party’’ because it does not mention a specific event. But it would be OK to say, ‘‘I had a good

time at Jane's party'' because this is a specific event. So "when I go to school" is not specific, because you go to school lots of times, but "at school last Friday" is specific because that's one time that you went to school. So we would like to know about one particular time you did something, or one specific event.

Control Condition

We are interested in your memory for events that have happened in your life. For each of the following words we would like you to think of a memory which the word reminds you of. The event could have happened recently (e.g., yesterday, last week) or a long time ago. It might be an important event, or a not so important event.

Appendix D

Affect Rating Scale for Study 3 and Study 4

We would like to know how you are feeling right now. Please circle a face that best shows how you are feeling

Appendix E

Study 4 Autobiographical Memory Test Instructions by Retrieval Condition

Avoid Condition

For each of the following words we would like you to think of a memory. The memory could have happened recently (e.g., yesterday, last week) or a long time ago. It might be something important, or not so important. But we would like you to avoid thinking about the specific event you just wrote about. Instead, we'd like you to tell us about events that happen lots of times. So if we said the word ''bad'' it would not be OK to say, ''I felt bad when my dog got put down" if you just told me about when your dog passed away. But it would be OK to say, ''I feel bad when I lose a game'' because this is of something that happens lots of times. Another example is if we said the word "pain" you could say "It's a pain when I have to get an injection." So we would like to know about things that happen lots of times, not about the one memory you just told us about.

Specific Condition

For each of the following words we would like you to tell us a memory about the event you just wrote about. Each memory should be a specific event that happened during the time you told us about. A specific event is something that happened one particular time. So if we said the word ''bad'' it would not be OK to say, ''I feel bad when I lose a game'' if you just told me about when your dog passed away, and besides, losing a game is something that happens lots of times. But it would be OK to say ''I felt bad when my dog got put down", because this is a specific memory about the time your dog passed away. Another example is if we said the word "pain" you could say "My

dog was in pain so we took him to the vet, and then the vet said he had to be put down". So we would like to know about specific memories about the event you described.

Distraction Condition

For each of the following words, we would like you to please pick a movie or TV show and briefly describe what it is about. I will read each word out, and give you a minute to write about it.