

ABSTRACT

Title of Dissertation: EXAMINING THE CO-DEVELOPMENT OF EPISODIC MEMORY AND HIPPOCAMPAL SUBFIELDS – A LONGITUDINAL STUDY

Kelsey Leigh Canada, Doctor of Philosophy,
2020

Dissertation directed by: Associate Professor, Tracy Riggins,
Department of Psychology

Episodic memory is a cornerstone ability that allows one to recall past events and the context in which they occur. Many different tasks have been used to assess the development of episodic memory during early childhood. Previous longitudinal work on individual tasks has noted accelerated changes from approximately 5 to 7 years, suggesting non-linear changes in memory ability during early childhood. However, the extent to which tasks relate to one another and are indicative of the latent construct of episodic memory is not known. Further, improvements in memory are thought to relate to underlying changes occurring in the functionally distinct subfields of the hippocampus (i.e., CA2-4/dentate gyrus (DG), CA1, and Subiculum) during this developmental period. This study examined changes in episodic memory ability, hippocampal subfield volume, and the relation between changes in episodic memory and volume of hippocampal subfields during early childhood (4 to 8 years) using longitudinal data and a structural equation modeling framework. Results suggest that

episodic memory ability improves substantially during this period, with consistent improvements between 4 to 8 years. Further, there are robust increases in subiculum, CA1, and CA2-4/DG volume between 5 to 6 years of age. Finally, within this sample, there were relations between the development of hippocampal subfields and improvements on a single source memory task commonly used to assess episodic memory. Interestingly, this relation was most robust between subiculum and source memory. Overall, these results highlight the ability to use laboratory tasks to characterize developmental changes in episodic memory, highlight 5- to 6-years as a period of developmental change in hippocampal subfields, and further support a role of the hippocampus in supporting episodic memory.

EXAMINING THE CO-DEVELOPMENT OF EPISODIC MEMORY AND
HIPPOCAMPAL SUBFIELDS – A LONGITUDINAL STUDY

by

Kelsey Leigh Canada

Dissertation submitted to the Faculty of the Graduate School of the
University of Maryland, College Park, in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
2020

Advisory Committee:

Associate Professor Tracy Riggins, Chair
Professor Gregory R. Hancock
Professor Michael R. Dougherty
Associate Professor L. Robert Slevc
Associate Professor J. Carson Smith

© Copyright by
Kelsey Leigh Canada
2020

Dedication

To my family – you have filled my life with love, support, and so many memories worth remembering. And especially to my Baba, whom I miss dearly, for encouraging me to always shoot for the moon and stars. I love and appreciate you all more than words can ever express. Thank you for being my anchors through the ups and downs of this part of my journey. I could not have completed it without all of you.

Acknowledgements

There are many people I am thankful for that have supported me throughout my graduate training and the completion of this dissertation. Completing my dissertation amid the COVID-19 pandemic has endlessly reminded me how lucky and grateful I am for my support system. First and foremost, I would like to express my appreciation for my amazing advisor, Dr. Tracy Riggins. She has gone above and beyond in her patience, kindness, guidance, and support for my research and for me as a person (including allowing me to include Harry Potter references in my talks). I am truly lucky to have a mentor as dedicated as her. I would also like to thank Dr. Gregory R. Hancock for his mentorship, patience while teaching me about Structural Equation Modeling, continual support, and good humor. Further, I want to thank my other committee members Drs. Dougherty, Slevc, and Smith for their time and helpful feedback on my dissertation research. I would also like to thank the members of the University of Maryland Neurocognitive Development Lab who helped with scheduling participants, data collection, and data processing over the years. This research would not be possible without the work of the NCDL team. I especially want to thank my labmate and friend Morgan Botdorf for the years of sharing in my coffee addiction, providing invaluable feedback on my research, and most importantly, her true kindness and friendship. I want to thank my family and friends both near and far for their encouragement and sharing in my tears and laughter throughout graduate school. My boyfriend, Marcus, my parents, Frank Canada and Nancy Miles-Canada, my sister, Jaime Canada, and my Nana, Doreen Miles, have given me the strength to persevere and believe in myself even when the going gets tough. Lastly, I would like

to thank the participants and their families for their dedication to science and of their time. Their contributions allowed me to pursue my passion for understanding how and why memory changes over development.

Table of Contents

Dedication.....	ii
Acknowledgements.....	iii
Table of Contents.....	v
List of Tables.....	vii
List of Figures.....	viii
List of Abbreviations.....	ix
Chapter 1: Introduction.....	1
Episodic Memory Development.....	3
Hippocampal Development.....	6
Relations between Hippocampal Subfields and Episodic Memory.....	11
Chapter 2: Method.....	15
Participants.....	15
Materials and Procedures.....	17
Behavioral Tasks.....	18
Primacy task.....	18
Temporal memory recall.....	19
Feature-binding task.....	20
Source memory task.....	22
General cognitive ability.....	24
Practice effects.....	24
MRI Assessment.....	25
Subfields.....	25
Analytical Framework: Structural Equation Modeling.....	27
Latent Growth Models.....	28
Preregistration of Analyses.....	30
Confirmatory Analyses.....	31
Modeling Episodic Memory Development.....	31
<i>Confirmatory factor analysis</i>	32
<i>Latent growth model</i>	34
Trajectory Convergence.....	36
Modeling Parallel Development of Episodic Memory and Hippocampal Subfields.....	36
Alternative Analytical Approach.....	37
Assessing Model Fit.....	39
Exploratory Analyses.....	41
Modeling Hippocampal Subfield Development.....	41
Specificity in the Relation Between Episodic Memory and Hippocampal Subfields.....	44
Chapter 3: Results.....	46
Preliminary Analyses – Practice Effects.....	46
Measurement Invariance of Episodic Memory Tasks.....	47
Cohort Convergence.....	48
Development of Episodic Memory.....	48

Development of Hippocampal Subfields	50
Amygdala Development	53
Parallel Development – Brain and Behavior	55
Chapter 4: Discussion	57
What is the trajectory of the development of episodic memory ability during early childhood?	57
What are the developmental trajectories of CA2-4/DG, CA1, and subiculum subfields in the hippocampal body during early- to mid-childhood?	65
Do developmental CA2-4/DG, CA1, and subiculum subfields in the hippocampal body relate to episodic memory development?	69
Assessing specificity of results related to the hippocampus	73
General Limitations	75
Conclusions	76
Appendices	78
References	99

List of Tables

Table 1. Summary of Aims and Hypotheses.

Table 2. Average Age and Number of Participants by Group Providing Any Data at Each Time Point.

Table 3. Breakdown of the Number of Children Contributing Behavioral and Neuroimaging Data at Each Wave.

Table 4. Summary of Tasks and Measures.

Table 5. Summary of Differences Between Cohorts for All Indicator Variables.

Table 6. Growth Parameters for Episodic Memory Models by Cohort.

Table 7. Fit Indices for Each Subfield Model (CA2-4/DG, CA1, and Subiculum) by Cohort.

Table 8. Growth Parameters for Each Subfield Model (CA2-4/DG, CA1, and Subiculum) by Cohort.

Table 9. Growth Parameters for Amygdala Models by Cohort.

Table 10. Fit Indices for Multi-Cohort Multidomain Second-Order Piecewise Latent Growth Models.

List of Figures

Figure 1. Schematic depiction of the timing of data collection.

Figure 2. Conceptual Diagram of episodic memory CFA model used to test the measurement model of the indicators of a latent episodic memory variable across the age-range of interest, assuming factorial invariance within each cohort separately.

Figure 3. Conceptual diagram of second-order piecewise latent growth model for episodic memory (EM).

Figure 4. Conceptual diagram of second-order piecewise latent growth model for a selected hippocampal subfield (CA2-4/DG).

Figure 5. Conceptual diagram of multidomain second-order piecewise latent growth model for episodic memory (EM) and a selected hippocampal subfield (CA2-4/DG) in the 4-year-old cohort.

Figure 6. Growth trajectory of episodic memory ability from 4 to 8 years of age.

Figure 7. Growth trajectories of hippocampal subfield volumes in body from 4 to 8 years of age.

Figure 8. Growth trajectories of amygdala volume from 4 to 8 years of age.

List of Abbreviations

CA1 = Cornu Ammonis 1

CA2-4/DG = Cornu Ammonis 2-4/Dentate Gyrus

ICV = Intracranial Volume

Chapter 1: Introduction

“You have to begin to lose your memory, if only in bits and pieces to realize that memory is what makes our lives. Life without memory is no life at all...our memory is our coherence, our reason, our feeling, even our action. Without it, we are nothing...” (Buñuel, 1983, pp. 4-5).

Memory, as eloquently stated above, makes for a rich life. However, memory is not a unitary construct, and we rely on many forms of memory. As defined by Tulving, a memory system can be thought of as a system of processes that is defined by its “brain mechanisms, the kind of information it processes, and the principles of its operations” (Schacter & Tulving, 1994, p. 13). The idea of multiple systems, versus a single system, of memory emerged in the 1950s when a patient (H.M.) with bilateral damage to the medial temporal lobes (MTL) showed catastrophic impairments in memory that required conscious awareness (i.e., declarative memory, explained below), but had relatively intact short-term memory, and was able to learn new skills that did not require conscious learning (Scoville & Milner, 1957).

At a very broad level, memory systems can be divided into a short-term memory system that can only hold a limited amount of information in conscious attention for a brief amount of time, and a long-term memory system that can retain information on the scale of minutes, days, weeks, months, or years (Schacter & Tulving, 1994). Short-term memory, also referred to at times as working memory, is often conceptualized as a memory system that can temporarily hold a limited amount of relevant information in conscious attention, with the capability to use and manipulate information in the limited store (Baddeley, 1992). Short-term memory is thought to be supported by cortical regions, specifically inferior temporal regions that can be supported by frontal and posterior-parietal networks, with the supporting networks appearing similar to the

system relied on in long-term memory at times (Eichenbaum, 2000; Nee & Jonides, 2008).

While some posit that short-term memory can be parsed into different components (Baddeley & Hitch, 1974), which process potentially different types of information that rely on different regions in the brain (Eichenbaum & Cohen, 2001; Talmi, Grady, Goshen-Gottstein, & Moscovitch, 2005), the discussion of short-term memory is beyond the scope of this dissertation.

For long-term memory, a distinction can be made between systems that support the conscious retrieval and use of stored information, called declarative memory, and systems that largely operate at the unconscious level, called non-declarative memory (Schacter & Tulving, 1994). Non-declarative memory includes: the capacity for learning skills or habits, which relies on the striatum; priming, which is thought to rely on regions of neocortex; and some forms of simple conditioning, which rely on the cerebellum and certain deep nuclei of the brainstem (Bauer, 2008; Squire, 1992). As mentioned above, patients with damage to the MTL of the brain, including the hippocampus, which supports declarative memory (expanded upon below) are still able to acquire new skills (Squire, 1992), albeit slowly. This suggests the non-declarative memory system does not critically rely on the same processes that subserve declarative memory.

Of interest to this dissertation, the declarative memory system is thought to support conscious memory that is specialized for rapid learning (Bauer, 2008). Declarative memory can be divided into the subsystems of episodic and semantic memory. Semantic memory supports memory for facts and general knowledge of the world, such as people, places, and things (e.g., I know that Michigan is shaped like a hand, but I do not remember where or when I learned this). This type of memory is relatively spared following damage to the hippocampus (Vargha-Khadem et al., 1997) and is thought to rely more on the cortices surrounding the hippocampus (Davachi, 2006; Squire, 1992). The episodic memory system is thought to support memory for

one's own experiences that encompasses information about not only the content of the experience (i.e., what) but the spatial (i.e., where) and temporal (i.e., when) context in which an event occurred (Tulving, 2002). Whereas semantic memory may recruit the hippocampus to retrieve known factual details (Vargha-Khadem et al., 1997), episodic memory is thought to crucially rely on the hippocampus for both forming and retrieving details of events (Squire & Zola-Morgan, 1991). The hippocampus is critically important in the network of regions in the MTL, including perirhinal cortex, parahippocampal cortex, entorhinal cortex (e.g., Davachi, Mitchell, & Wagner, 2003; Diana, Yonelinas, & Ranganath, 2007; Ghetti, DeMaster, Yonelinas, & Bunge, 2010), as well as prefrontal cortex (e.g., Ghetti et al., 2010; Ofen et al., 2007), and in some cases posterior parietal cortex (e.g., DeMaster & Ghetti, 2013; reviewed in Moscovitch, Cabeza, Winocur, & Nadel, 2016) that work together to support episodic memory. The focus of this dissertation was on this latter form of memory, episodic memory, and the region of the brain necessary for this ability, the hippocampus.

Episodic Memory Development

As noted above, episodic memory is the ability to encode and retrieve specific details of life-experiences, including associations between people or objects, spatial relations of objects, and temporal information in such a way that an individual can “mentally time travel” and re-experience a specific event (Rolls, 2016; Tulving, 1972, 1993), and is critically dependent on the hippocampus (e.g., Nadel & Moscovitch, 1997; Ranganath et al., 2004).

Current work examining the development of episodic memory using both cross-sectional and longitudinal research suggests significant improvements in children's memory ability occur between the ages of 5 and 7 years (Riggins, 2014). This period of early childhood is a time of rapid, significant change in many aspects of episodic memory ability including: memory for

details such as color (Cycowicz, Friedman, Snodgrass, & Duff, 2001), location (Bauer et al., 2012), memory for the source of information (e.g., Drummey & Newcombe, 2002; Riggins, 2014), memory for the temporal order of events (e.g., Friedman, 1992; Riggins, Miller, Bauer, Georgieff, & Nelson, 2009), memory for fine-grained details (e.g., Canada, Ngo, Newcombe, Geng, & Riggins, 2019; Ngo, Newcombe, & Olson, 2017), and overall increased memory of the number of details recalled (e.g., Riggins, Blankenship, Mulligan, Rice, & Redcay, 2015). For example, when tasked with recalling the source of information, whether a novel fact was learned from either a person or a puppet, young children are more likely than older children to forget the source of the information and attribute the learned information to a source outside of the experiment (i.e., extra-experimental errors; Drummey & Newcombe, 2002; Riggins, 2014; Riggins et al., 2018).

Although studies of memory development are effective in informing the field's understanding of when episodic memory develops, they lack that utilized tasks are each thought to tap *aspects* of episodic memory. As noted by Tulving (2002), it is unlikely that a specific relation between a given task and a given memory system exists. Instead, it is probable that different memory systems (e.g., episodic memory) are recruited to varying degrees depending on the features of the task. These tasks are not process-pure, and the measurements of tasks are not error-free. Given the difficulty of using single tasks to assess episodic memory, other approaches, such as utilizing latent constructs, may be useful for examining the development of episodic memory.

The use of latent constructs to assess differences in cognitive ability, including episodic memory, has been successfully applied in adult populations (e.g., Brambilla, Manenti, Ferrari, & Cotelli, 2015; Head, Allison, Lucena, Hassenstab, & Morris, 2016; MacAulay et al., 2017; Park

et al., 1996; Spaan, 2015; Zuber, Kliegel, & Ihle, 2016). For example, Head et al. (2016) utilized structural equation modeling (SEM) to assess the latent structure of a battery of standardized tests. Specifically, they confirmed the feasibility of using a logical memory task, a verbal paired associates test, and a free recall task to assess episodic memory as a construct. Another study used both standard and lab-based tests of episodic memory abilities (Spaan, 2015), specifically a 10-word free-recall test, 10-word recognition test, paired associates learning (cued-recall) test for semantic pairs, paired associates learning (cued-recall) test for non-semantic pairs, and a paired-associate-recognition (forced choice) test to indicate a latent construct of episodic memory. Although both of these studies were cross-sectional in nature, both suggest the use of both standardized and novel tasks of episodic memory to indicate a latent construct of episodic memory ability is feasible.

To my knowledge, no research to date has attempted to examine a latent construct of episodic memory using a battery of tasks during early childhood. A latent construct would be beneficial as it would allow us to move beyond the use of single tasks examined separately and independently of each other, account for measurement error of these tasks, and crucially, examine the trajectory of the underlying construct of interest, episodic memory.

Aim 1 of this dissertation was A) to examine the feasibility of capturing a latent construct of episodic memory and B) to examine how a latent construct of episodic memory ability changes across development.

Hypothesis 1_A: Tasks used individually to assess aspects of episodic memory ability will share variance that indicates a latent construct of episodic memory.

Hypothesis 1B: Episodic memory ability will show non-linear increases, with greater positive slopes between ages 4 to 5 years and between 5 to 6 years, and lesser increases in performance between 6 to 7 years and between 7 to 8 years.

Hippocampal Development

Interestingly, in addition to improvements in performance on episodic memory tasks documented in young children, early childhood is also a period of development when the hippocampus, a structure critical for memory, is proposed to mature. As noted above, the hippocampus is a heterogeneous structure within the MTL. This structure can be divided along its longitudinal axis into subregions (head, body, and tail; e.g., Poppenk, Evensmoen, Moscovitch, & Nadel, 2013) and into functional subunits (subfields; Lavenex & Banta Lavenex, 2013). Subregions are thought to have different functional relevance because of structural connectivity, as there are structural projections from each subregion to different brain regions (Amaral & Lavenex, 2006; Duvernoy, 2005; Poppenk et al., 2013; Strange, Witter, Lein, & Moser, 2014). Subregions show different developmental trajectories in cross-sectional (DeMaster, Pathman, Lee, & Ghetti, 2014; Krogsrud et al., 2014; Riggins et al., 2018) and longitudinal samples (Gogtay et al., 2006; Tamnes, Bos, van de Kamp, Peters, & Crone, 2018). Studies with young children (e.g., 4- to 5-year-olds) have shown age-related increases in head volume (Krogsrud et al., 2014; Riggins et al., 2018), increases in body volume (Krogsrud et al., 2014), and slight decreases (DeMaster et al., 2014; Gogtay et al., 2006) or little change in tail volume (Riggins et al., 2018; Tamnes et al., 2018). Other studies with older participants have shown age-related decreases in head volume (DeMaster et al., 2014; Gogtay et al., 2006), and similar developmental patterns in body and tail.

Subfields of the hippocampus (dentate gyrus, DG; cornu ammonis, CA1-CA4; subiculum) are anatomically interconnected but functionally distinct (Rolls, 2013). These subunits are delineated *along* the longitudinal axis (versus delineating the longitudinal axis, i.e., hippocampal subregions), and also are thought to have differential developmental trajectories. DG and CA3 show the most prolonged developmental trajectory and are thought to reach relative maturity between 5 to 7 years based on work in non-human primates (Lavenex & Banta Lavenex, 2013) and post-mortem humans (Seress, 2001). The extended trajectory of CA3 is proposed due to its reliance on input from DG, suggesting that development of DG is especially critical to improvements in memory (Rolls, 2013). Cross-sectional work examining structural differences in volume of CA2-4/DG combined across hippocampal head and body suggest initial increases in volume followed by decreases in a sample of 4- to 22-year-olds (Krogsrud et al., 2014) and 4- to 8-year-olds (Canada et al., 2019). CA1 also shows an extended developmental trajectory, with post-mortem work in humans suggesting relative maturity of this region between 3 to 4 years (Seress, 2001). Cross-sectional work examining structural differences in CA1 combined across hippocampal head and body suggests slight initial increases in volume followed by decreases in a sample of 4- to 22-year-olds (Krogsrud et al., 2014) and 4- to 8-year-olds (Canada et al., 2019). The subiculum is thought to mature earlier than both CA2-4/DG, and CA1 based on work in non-human primates (Jabès, Banta Lavenex, Amaral, & Lavenex, 2011; Lavenex, Banta Lavenex, & Amaral, 2004; Seress, 2001). Cross-sectional work examining structural differences in subiculum combined across hippocampal head and body suggests slight volumetric decreases in a sample of 4- to 22-year-olds (Krogsrud et al., 2014) and 4- to 8-year-olds (Canada et al., 2019).

However, additional cross-sectional work in early childhood has shown that subfields are disproportionately distributed along the anterior-posterior axis of the hippocampus (i.e., in subregions). Specifically, the proportion of the subiculum is greater in head than body while the proportions of CA1 and CA2-4/DG volume are smaller in head than body (Riggins et al., 2018). Because subregions of the hippocampus (i.e., head, body, tail) show different developmental trajectories, form connections to different areas of the brain, and are comprised of variable amounts of the functionally distinct subfields, considering the development of subfields within head and body separately may be especially informative.

Results examining age-related differences of hippocampal subfields within head and body subregions separately (reliably identifying subfields in tail is not yet possible, see Yushkevich et al., 2015a) are less consistent than results examining the development of these subfields combined across subregions. Specifically, cross-sectional work examining development differences in DG volume has suggested no age-related differences in head or body in a sample of 4- to 8-year-olds (Riggins et al., 2018), a surprising finding considering work showing differences in combined volume of these regions (e.g., Canada et al., 2019). However, in a different sample of 6- to 30-year-olds, DG volume showed age-related increases in body (Schlichting, Guarino, Schapiro, Turk-Browne, & Preston, 2017). In the sample of 4- to 8-year-olds, CA1 volume in head showed a positive quadratic association with age (Riggins et al., 2018), whereas age negatively related to volume in the sample of 6- to 30-year-olds (Schlichting et al., 2017). No significant age-related differences in CA1 volume in body were observed in either sample. Finally, age-related differences were absent in both samples for subiculum volume in head (Riggins et al., 2018; Schlichting et al., 2017) and in body for the 4- to 8-year-old sample (Riggins et al., 2018). Subiculum volume in body showed age-related linear decreases in the 6-

to 30-year-old sample (Schlichting et al., 2017). Although cross-sectional results are mixed, they suggest developmental changes in hippocampal subfields should be considered along the anterior-posterior division of the hippocampal (i.e., within head and body, separately).

Of the above-mentioned studies, none examined longitudinal changes in hippocampal subfield development during early childhood. To my knowledge, work examining developmental changes in subfield volume is limited to one longitudinal study in a sample of 8- to 26-year-olds (Tamnes et al., 2018). This study combined subfield volumes in head and body and showed age-related linear decreases in CA2-4 and DG, a quadratic effect in CA1 with initial age-related increases in volume followed by decreases, and a cubic effect in subiculum, with early age-related increases in volume and decelerating decreases. Additional longitudinal research may clarify the changes that occur in these functionally distinct subfields earlier in development (i.e., before 8 years).

A consideration when studying hippocampal subfield development in-vivo is the difficulty of measuring these regions in a way that is structurally and functionally appropriate. As such, at best, the in-vivo measurement of subfields should be considered an educated approximation (Yassa & Stark, 2011; Yushkevich et al., 2015a). Many studies have restricted the examination of subfield volume to hippocampal body (or a subset of slices within the hippocampal body; Daugherty, Yu, Flinn, & Ofen, 2015; Yushkevich et al., 2015a) because subfields are more difficult to delineate in the head and tail. Thus, research on the development of subfields requires additional longitudinal research using reliable segmentation methods in order to clarify the developmental trajectories in humans (Sankar et al., 2017). Researchers have started to address this concern by utilizing improved methods of segmentation such as the Automated Segmentation of Hippocampal Subfields software (ASHS; Yushkevich et al., 2015a),

which was recently validated in a separate pediatric sample (Schlichting, Mack, Guarino, & Preston, 2019), and latent constructs of these subfields (Daugherty, Flinn, & Ofen, 2017). For example, research in a cross-sectional sample of 8- to 25-year-olds utilized SEM to study subfield development by using measures from left and right hemispheres in hippocampal head and body (Daugherty et al., 2017). The construction of this model uses the commonality of the two hemisphere measures to indicate a latent construct of each subfield while accounting for measurement error and thereby producing hypothetically error-free estimates of the intended variable.

Cross-sectional work on the development of hippocampal subfields during early childhood suggests developmental differences in CA2-4/DG, CA1, and subiculum. However, the direction, magnitude, and presence of age-related differences varied as a function of hippocampal subregion. Only one study has examined changes in these subfields during childhood, and this study included only older participants (> 8-years-old; Tamnes et al., 2018). Unfortunately, this age is beyond the point where early crucial developmental changes are thought to occur. Additionally, this longitudinal study did not differentiate subfields between subregions and used segmentation methods less reliable than recent advances. Work using latent constructs of hippocampal subfields and subregions have improved understanding of developmental differences starting in late childhood into adulthood (Daugherty et al., 2017). However, an understanding of the *change* that occurs in hippocampal subfields during the important period of early childhood is lacking. As the majority of extant cross-sectional literature has focused on developmental differences in the hippocampal body (due to the fact boundaries are clearest in this subregion), this dissertation focuses on examining changes in subfields of the hippocampal body during early- to mid-childhood.

Aim 2: Examine the developmental changes in hippocampal subfields A) CA2-4/DG, B) CA1, and C) subiculum within the body of the hippocampus during early childhood (4-8 years) using longitudinal data and latent constructs.

Hypothesis 2A:) Development of CA2-4/DG in the hippocampal body during early childhood is non-linear, with greater increases in volume occurring between ages 5 to 7 years compared to little change between 4 to 5 years and 7 to 8 years.

Hypothesis 2B) Development of CA1 in the hippocampal body during early childhood is non-linear, with increases in volume occurring between ages 4 to 6 years and little change between 6 to 8 years, as it is thought to develop before DG.

Hypothesis 2C:) Development of subiculum in the hippocampal body is negligible between 4 to 8 years as it is thought to be early developing.

Relations between Hippocampal Subfields and Episodic Memory

Thus far, developmental differences in aspects of episodic memory ability and hippocampal subfields have been highlighted separately. Drawing connections between these domains to examine the extent to which development in brain and behavior relate is equally important in informing our understanding of typical development in early childhood. Prior cross-sectional research that included children during early- to mid-childhood has linked gains across a variety of episodic memory tasks to the hippocampus.

To be specific, in a study of 6- to 30-year-olds examining associative inference, or the ability to make connections between pairs of related items (i.e., AB and BC \rightarrow AC), smaller volumes of CA1 in hippocampal head related to better associative memory in children while larger volumes of CA1 related to better associative memory in adults (Schlichting et al., 2017). A different study examining memory for the source of information in 4- to 8-year-olds found better

source memory related to larger CA1 in the hippocampal head for younger children while smaller volumes of CA1 related to better source memory in older children. Furthermore, in the hippocampal body, smaller volumes of CA1 and larger volumes of CA2-4/DG related to better source memory (Riggins et al., 2018). Further, a study examining memory for fine-grained detail (e.g., memory for a specific piano versus a slightly different piano) in 4- to 8-year-old children found that this ability was related to larger volumes of CA2-4/DG combined across hippocampal head and body in younger children, while smaller volumes of CA2-4/DG related to better fine-grained memory in older children (Canada et al., 2019). These findings point to similar subfields related to episodic memory in older children (8+ years) and adolescents, (i.e., CA2-4/DG and CA1, Daugherty et al., 2017; Lee, Ekstrom, & Ghetti, 2014).

Current work relating hippocampal subfields is limited in scope and cross-sectional in nature. However, these studies suggest age-related differences in the volume of CA1 and CA2-4/DG relate to performance on individual tasks examining aspects of episodic memory. To my knowledge, the co-occurring developmental changes in hippocampal subfields and episodic memory have yet to be examined during early childhood.

Aim 3: Examine the relations between changes in episodic memory ability and hippocampal subfields A) CA2-4/DG, B) CA1, and C) subiculum within the body of the hippocampus during early childhood (4 to 8 years) using longitudinal data and latent variables.

Hypothesis 3_A:) CA2-4/DG in the hippocampal body will relate to the development of episodic memory.

Hypothesis 3_B:) CA1 in the hippocampal body will relate to the development of episodic memory.

Hypothesis 3c:) Subiculum in the hippocampal body will not relate to the development of episodic memory.

In sum, this dissertation seeks to address three main aims: 1) to examine developmental changes in episodic memory using a battery of episodic memory tasks; 2) to examine developmental changes in hippocampal subfields; and 3) to examine the relation between changes in episodic memory and developmental changes in the volume of hippocampal subfields to in 4- to 8-year-old children (see Table 1 for summary of hypotheses).

Table 1

Summary of Aims and Hypotheses.

Aim	Hypotheses
<p>1) What is the trajectory of development during early childhood episodic memory ability?</p>	<p>1A) Tasks used individually to assess aspects of episodic memory ability will share variance that indicates a latent construct of episodic memory.</p> <p>1B) Episodic memory ability will show non-linear increases, with steeper positive slopes between ages 4 to 6 years, and slight increases in performance between 6 to 8 years.</p>
<p>2) What is the trajectory of development during early childhood in hippocampal body for:</p> <ol style="list-style-type: none"> 1. CA2-4/DG subfield 2. CA1 subfield 3. Subiculum subfield 	<p>2A) Development of CA2-4/DG in hippocampal body during early childhood is non-linear, with increases in volume occurring between ages 5 to 7 years and little change between 4 to 5 years and 7 to 8 years.</p> <p>2B) Development of CA1 in hippocampal body during early childhood is non-linear, with increases in volume occurring between ages 4 to 6 years and little change between 6 to 8 years, as it is thought to develop before DG.</p> <p>2C) Development of subiculum in hippocampal body is negligible between 4 to 8 years as it is thought to be early developing.</p>
<p>3) Do developmental changes in hippocampal body subfields relate to episodic memory development for:</p> <ol style="list-style-type: none"> 1. CA2-4/DG 2. CA1 3. Subiculum 	<p>3A) CA2-4/DG in hippocampal body will relate to the development of episodic memory.</p> <p>3B) CA1 in hippocampal body will relate to the development of episodic memory.</p> <p>3C) Subiculum in hippocampal body will not relate to the development of episodic memory.</p>

Chapter 2: Method

Participants

The current study was part of a larger research project examining the development of the brain in relation to memory. Prior to data collection, all methods were approved by the Institutional Review Board at The University of Maryland. Hippocampal subfields have been previously examined cross-sectionally (Riggins et al., 2018), specifically in relation to the source memory task included as an indicator in the current study. However, these data have not yet been examined using the longitudinal sample, nor using a latent construct of episodic memory ability. Children were screened via caregiver self-report to ensure they were not born premature (via gestational age), had normal or corrected-to-normal vision, and had no diagnoses for any neurological conditions, developmental delays, or disabilities. Informed consent was obtained from parents, written assent was obtained for children aged 7 years or older than, and verbal assent was obtained for children younger than 7 years.

A total of 200 4- to 8-year-old children (100 reported females, 100 reported males) participated in the current study. A cohort-sequential (i.e., accelerated longitudinal) design was employed with cohorts overlapping at age 6 years in order to simulate a longer longitudinal trajectory (Duncan, Duncan, & Hops, 1996). By simulating a traditional longitudinal design, the goal was to assess the developmental change that occurs during this period. Of the 200 children who participated in the study at wave 1, 96 were invited to participate in subsequent visits and were included as the longitudinal cohort. Children who were recruited at age 4 years or age 6 years were invited back at two subsequent waves at provided data maximally at 3 waves.

Children in the cross-sectional cohorts (i.e., recruited at 5, 7, or 8 years of age) provided data only at one wave.

Younger age groups were oversampled to ensure enough usable data would be available and because participants were being followed longitudinally. For a breakdown of the average age at which data was provided at each wave and the number of subjects providing data for each group see Table 2. For a breakdown of the number of data points acquired at each wave see Table 3. The majority of the sample (76%) was Caucasian, from middle- to high-income households (median = >\$105,000, range = < \$15,000 - >\$105,000). 89% of participants had at least one parent with a 4-year college degree.

Table 2

Average Age and Number of Participants by Group Providing Any Data at Each Time Point.

	M_{AGE} , (N)				
Cross-sectional sample	4.40 (61)	5.55 (35)	6.36 (41)	7.53(32)	8.60 (31)
Longitudinal sample 1	4.40 (61)	5.46 (50)	6.44 (48)		
Longitudinal sample 2			6.36 (41)	7.32 (35)	8.47 (35)

Note. Cohort-sequential design. Red and blue color denotes same individuals. M_{AGE} = Mean age in years.

Table 3

Breakdown of the Number of Children Contributing Behavioral and Neuroimaging Data at Each Wave.

Measure	Participants providing data							Total <i>N</i>
	Wave 1 Only	Wave 2 Only	Wave 3 Only	Waves 1 & 2	Waves 1 & 3	Waves 2 & 3	Waves 1, 2, &3	
MRI	93	5	4	6	10	8	39	165
Source Memory	108	0	0	5	4	0	77	195
Primacy	113	0	0	4	2	1	80	200
Temporal Recall	112	0	0	3	3	1	79	198
Feature- Binding	112	0	0	6	2	0	79	199

Materials and Procedures

At each wave children visited the laboratory twice, approximately 7 days apart, and completed a battery of tasks¹ thought to reflect aspects of episodic memory and a structural magnetic resonance imaging (MRI) scan. A measure of global intelligence was administered only at wave 1 (Figure 1). Two groups of children, 4-year-olds and 6-year-olds were invited to participate in the longitudinal portion of the study. For information on counterbalancing of tasks across waves see Appendix A.

¹ The Stories subtest from the Children's Memory Scale (Cohen, 1997) was also administered to children at each wave. However, this task ultimately was not included in this dissertation due to multiple modifications from the standardized scoring guide and the presence of practice effects.

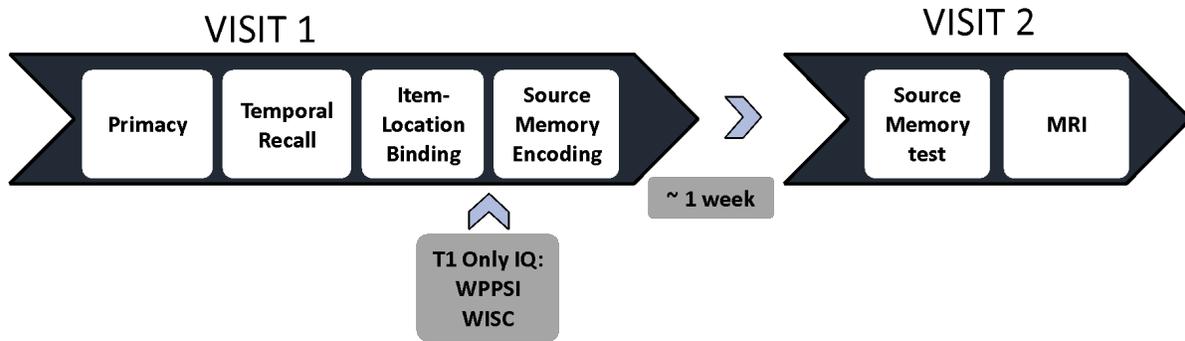


Figure 1. Schematic depiction of the timing of data collection.

Behavioral Tasks

For a summary of behavioral tasks and measures used see Table 4.

Table 4

Summary of Tasks and Measures.

Aspect of Episodic Memory	Measures	Dependent Variables
Recall for source details	Source Memory	Proportion of facts with both correct fact memory and correct source memory
Recognition of temporal order	Primacy	Proportion of correct primacy judgments
Recall of temporal order	Temporal Recall	Proportion of adjacent pairs recalled for two sequences
Recognition for item and location	Feature-Binding	$d' = Z(\text{Proportion of Hits}) - Z(\text{Proportion of FAs})$

Primacy task. The primacy task was included as a measure of recognition memory for temporal order. It consisted of a modified version of a task used in two previous investigations of primacy discrimination in early to middle childhood (Alden, 1994; Mathews & Fozard, 1970).

Specifically, children were presented with four different lists of pictures (two 8-item lists and two 12-item lists). Each child was also given a 4-item practice list to ensure task understanding. Item lists were composed of simple line-drawings of common objects (e.g., button, paper bag). Each item was presented individually with a verbal label (e.g., “button,”

“paper bag”) at a rate of approximately 1 picture every 2 seconds and placed face up in a pile on the table to eliminate spatial cues. Children were instructed to remember the order of the pictures. After each list was presented, the child was presented with two 2-alternative forced-choice questions immediately, or with an age-appropriate distractor task before answering the forced-choice questions. For the distractor task, the child either played tic-tac-toe or was given instructions to draw a house if he/she did not know how to play tic-tac-toe. Children were asked which of the two pictures was presented earlier in the sequence (a primacy judgment).

Each pair of items used for primacy judgments was presented an equal distance apart within the lists (with one picture in between). One pair of items was drawn from the first half of the list and the second pair from the second half of the list. Thus, for both 8-items lists, items 2 and 4 were paired and items 5 and 7 were paired for primacy judgments. For 12-item lists, one tested judgments for items 3 and 5, and items 7 and 9, the other tested judgments for items 4 and 6, and items 8 and 10. This list design was used in order to ensure 1) the first and last items presented were not used for judgments, and 2) there was no overlap between pairs of items. List presentation order and lists assigned immediate judgments or a distractor task before judgments were counterbalanced across participants. Preliminary analyses investigated whether any of the minor experimental manipulations impacted memory performance (i.e., 8 versus 12 items per list, immediate versus delayed judgments, and section of the list). While slight differences were found, both 8- and 12-item lists showed statistically significant improvements over this period. Thus, the proportion of correct primacy judgments across all lists was used as the dependent variable and included as an indicator of the latent construct of episodic memory.

Temporal memory recall. To assess recall memory for temporal order, an ordered sequence recall task (Bauer et al., 2013) was used. Children were shown one 4-item practice

picture sequence (Yard) to ensure understanding, and then two of three possible 9-item picture sequences (Pet Shop, Park, Fair) using laminated index cards for each picture. The order of the two 9-item sequence presentations was counter-balanced and pairs of lists randomly assigned across participants. The Experimenter introduced the sequences with a verbal label (e.g., “I’m going to show you how I work in the yard.”). Child the watched the Experimenter demonstrate the sequence by showing each picture in the sequence (e.g., “mow the lawn”) accompanied with a verbal label and placing the pictures on the table in an upside down “V” shape (from child’s left to right). No causal or temporal language cues (e.g., “next” or “then”) were given in the verbal labels. When the sequence was finished, the event label was provided again, (e.g., “That’s how I work in the yard”). Upon completion, the pictures were shuffled and children attempted to reconstruct the sequence.

Participants were randomly assigned to an age-appropriate distractor task (a game of tic-tac-toe or instructions to draw a picture of a house) between the presentation of one of the 9-item picture sequences and its reconstruction. Once the child reconstructed the sequence to the best of their ability, the child’s reconstructed sequence order was recorded. Children’s reconstructions were scored on the number of adjacent pairs (two items in the exact correct order, one after another, such as 6 and 7), with 8 possible adjacent pairs for each sequence (16 total adjacent pairs possible). Whether immediate or delayed reconstructions differed in accuracy was assessed, and no differences between these reconstructions were found. Thus, the proportion of adjacent pairs recalled across sequences was included as an indicator of the latent construct of episodic memory.

Feature-binding task. To assess children’s ability to bind items and locations in memory, they completed a feature-binding task (Lorsbach & Reimer, 2005). Participants were

first shown a booklet that introduced them to a set of eight black-and-white line drawings of common objects (pumpkin, fish, balloon, kite, snowman, lion, frog, heart), and a 3×3 grid that was approximately 15.5 cm × 15.5 cm. Children were told that on each trial, three different pictures would be displayed one at a time in different grid squares. Children then saw a booklet with an example of a sequence that might occur. After completing training, children completed task using the presentation software, E-Prime (Psychology Software Tools, Pittsburgh, PA).

Participants completed two practice trials on the computer to familiarize them with the task and to ensure they understood the directions. After successful completion of the practice trials, the presentation software randomly assigned each participant one of two possible versions. For longitudinal participants, at each wave, a participant was equally likely to be randomly assigned to version 1 or version 2 of the task by E-Prime.

The task consisted of 32 trials testing children on their memory for both objects and their respective locations on the grid. The 32 trials were split between 16 target trials and 16 lure trials. The presentation order of target and lure trials was random within participant. Target trials consisted of a test item showing the object in its studied location on the grid for that trial and lure trials consisted of test items showing objects re-paired to their respective locations. Participants were instructed to verbally respond "Yes" if the test item displayed an object in its studied location on the grid and to respond "No" if it did not. All responses were recorded by the Experimenter using a keyboard.

For each trial, the word "Ready?" was displayed on screen followed by a 1000 ms blank screen delay before the 3×3 grid was displayed. The grid remained on the screen for 3000 ms, with three different pictures shown successively for 1000 ms each in three different locations on the grid. A 4000 ms delay interval followed, and a question mark was shown in the center of the

screen. Next, a blank screen was displayed for 1000 ms, followed by the test item for that trial. The test item remained visible until the participant verbally responded. Once recorded, the response was followed by a 2000 ms inter-trial interval.

The measure d' was calculated (Snodgrass & Corwin, 1988) to provide a measure of children's memory for combined features (objects and locations) and included as an indicator of the latent construct of episodic memory.

Source memory task. To assess children's memory for novel items and the contextual details surrounding these items, a source memory task was used (Drumme & Newcombe, 2002; Riggins, 2014). The source memory task was administered across 2 visits in the lab that were separated by approximately 7 days. At Visit 1, children were taught new facts (e.g., "A group of rhinos is called a crash") from two different sources, a female adult ("Abby") and a male-voiced puppet ("Henry"), via digital videos. The children learned 6 facts from each source for a total of 12 facts. Three lists of facts were created, consisting of unique facts that were similar across lists (e.g., "A group of kangaroos is called a mob" or "A group of goats is called a tribe"). Lists were randomly assigned across participants. For each list, sources had 8 possible facts. Consequently, if a child knew 3 or more facts from a source, the total number of facts the child was tested on was reduced (but this was a rare case, $n = 16$ across all waves). Presentation of facts was blocked by source, with 6 facts from the first source (e.g., Henry) followed by 6 facts from the second source (e.g., Abby). Order of the blocks was randomly assigned across participants. To ensure that longitudinal participants did not receive the same facts in subsequent years, participants who received facts from List 1 at wave 1 received List 2 at wave 2 and List 3 at wave 3, and so on. Children were instructed to pay attention to the facts as they would be tested on the facts the following week. Children were not told they would be tested on the source of the facts. Before

the answer to each fact question was given, children were asked if they knew the fact (e.g., “Do you know what a group of rhinos is called?”). If they answered correctly, that fact was excluded at testing and an additional novel fact from the same list and source was presented.

At Visit 2, children were tested on their memory for the facts and their sources. Children were asked to answer 22 trivia questions and to tell the Experimenter where they had learned the answers to those trivia questions. Each test list of 22 facts had two random presentation orders, with orders counterbalanced across participants. Children were told that they had learned some of the questions the week before from either “Abby” or “Henry,” some they might have learned outside the laboratory (e.g., from a teacher or parent), and some they may not know. Of the 22 facts presented, 6 were from “Abby,” 6 were from “Henry,” 5 were commonly known by children (e.g., “What color is the sky?”), and 5 were facts that children typically would not know (e.g., “What is the colored part of your eye called?”). Children were instructed to ask the Experimenter for “hints” (i.e., multiple-choice options) if they did not know an answer to a question.

Each question was asked (e.g., “What is a group of rhinos called?”) and the child was given the opportunity to answer freely. If the child indicated they did not know the answer, they were given four pre-determined multiple-choice options (“hints”; e.g., Mob, Crash, Herd, or School). Once the child gave an answer during free recall or multiple-choice options, the Experimenter asked where the child had learned the information. Children could again answer freely or were given five multiple-choice options if they indicated they did not know the source (i.e., parent, teacher, girl in the video, puppet in the video, or just knew). Answers given during free response for fact or source questions were considered recall. This included when the child initially indicated they “just knew” or guessed the answer, as these were reasonable answers for

commonly known or fairly difficult facts. Answers given in response to the multiple-choice options were considered recognition.

Source memory was calculated as the proportion of questions where the child provided an accurate answer for both the fact and the source of the fact and included as an indicator for a latent construct of episodic memory.

General cognitive ability. Indices of intelligence were obtained using subtests from age-appropriate standardized intelligence tests to account for differences in general intelligence. The Wechsler Preschool and Primary Scale of Intelligence (WPPSI) is standardized for children aged 2 years 6 months to 7 years 7 months and was administered to 4- and 5-year-olds. The Wechsler Intelligence Scale for Children (WISC) is standardized for children and adolescents aged 6 to 16 years and was administered to 6-, 7-, and 8-year-olds. Specifically, scaled scores from the block design subtest, which reflects visual-spatial intelligence, were obtained for use as covariates to control for general differences in intelligence. One child was not administered the IQ test.

Practice effects. Practice effects are important to consider in the current study because both longitudinal cohorts (i.e., those enrolled at age 4 or 6 years) had repeated experience with the tasks, although the contents of these tasks varied at each measurement occasion. As a result, although the source memory task was incidental at Wave 1, it was not incidental at the subsequent waves, and familiarity with all tasks was greater at subsequent waves. Consequently, practice effects could have modified performance as a result of repeated experience with the task or increased familiarity/knowledge of the task or both. To assess this possibility, pairwise comparisons were examined between performance of 4-year-olds enrolled in a longitudinal cohort at ages 5-years and 6-years to initial performance of the cross-sectional 5-year-old cohort, and the initial performance for the longitudinal 6-year-old cohort. Comparisons were also made

for performance of 6-year-olds enrolled in a longitudinal cohort at ages 7-years and 8-years to initial performance of the cross-sectional 7-year-old cohort and cross-sectional 8-year-old cohort. Practice effects would be indicated if longitudinal subjects' scores are higher than those of subjects completing the task for the first time. This method was used in a similar study examining the longitudinal development of source memory in a separate cohort of 4- to 10-year-olds (Riggins, 2014).

MRI Assessment

All participants completed training in a mock scanner before MR data acquisition in order to become acclimated to the scanner environment and receive motion feedback. Participants were scanned in a Siemens 3.0-T scanner (MAGNETOM Trio Tim System, Siemens Medical Solutions, Erlangen, Germany) using a 32-channel coil. An initial structural scan was acquired using a high-resolution T1 magnetization-prepared rapid gradient-echo (MPRAGE) sequence consisting of 176 contiguous sagittal slices (0.9 mm isotropic; 1900 ms TR; 2.32 ms TE; 900 ms inversion time; 9° flip angle; pixel matrix= 256 x 256). This was used to measure intracranial volume and isolate the hippocampus for a subsequent ultra-high-resolution structural scan using a T2-weighted fast spin echo sequence (TR=4120 ms, TE=41 ms, 24 slices, 149° flip angle, voxel size .4 mm x .4 mm x 2 mm).

Subfields. Hippocampal subfield volumes were identified in the head and body of the hippocampus in both left and right hemispheres using an existing protocol (La Joie et al., 2010) based on Duvernoy (1998) and Harding, Halliday, and Kril (1998). The protocol was selected after existing protocols for manual tracing of hippocampal subfields were reviewed ($n = 21$, see Yushkevich et al., 2015b). Protocols developed for T2-weighted images with resolution similar

to data in this study and collected from 3T scanners were compared. Although several exist, we selected a protocol (La Joie et al., 2010) that yielded the subfields of interest in both the head and body subregions of the hippocampus at the desired resolution (.4mm x .4mm x 2mm) on a 3T scanner (but see also Berron et al., 2017; Winterburn et al., 2013). This protocol was selected because previous research in children has suggested developmental effects may be present in both the hippocampal head and body (DeMaster et al., 2014; Riggins et al., 2015, 2018). Similar to La Joie et al. (2010), seven different slice types were identified from coronal slices and used for manual segmentation (see La Joie et al., 2010; Riggins et al., 2018 for details). Three subfields were identified: subiculum, CA1, and a combination region of CA2-4/dentate gyrus (CA2-4/DG). Details regarding identification of internal and external boundaries are reported in Riggins et al. (2018).

Two raters blinded to the age and sex of the subjects independently traced 10 cases (2 from each of the 5 age groups) bilaterally. Dice Similarity Coefficients (DSC) were calculated to determine overlap between raters and are as follows for each subfield: Subiculum = .74, CA1 = .73, CA2-4/DG = .85. DSC values above 0.7 are typically considered acceptable for agreement (Zijdenbos, Dawant, Margolin, & Palmer, 1994), as such, overlap between the two raters indicated agreement. Intra-class correlations (ICC (2,1); Shrout & Fleiss 1979) were also calculated to determine reliability of the volume measurement and are as follows for each subfield: Subiculum = .93, CA1 = .98, CA2-4/DG = .90. ICC values above .90 are typically considered highly reliable, indicating consistency in the volume measurements.

One rater then traced an additional 10 cases (again, 2 from each age group). These segmentations were combined with the 10 cases used for manual reliability (i.e., 20 total) and input into ASHS (Yushkevich et al., 2015a) to create a study-specific template. This study-

specific template was used to generate hippocampal subfield volumes for the entire sample. All resulting segmentations were checked visually for quality. No manual edits were made, and only data from subjects yielding high-quality segmentations were included in the present study ($n = 165$).

Analytical Framework: Structural Equation Modeling

Structural equation modeling (SEM) is a powerful method to address longitudinal questions. SEM is theory based and largely confirmatory (Little, 2013). By specifying specific developmental hypotheses about individuals and groups into the structural model (i.e., the relations between the constructs), one can compare the means, variances, and covariances of interest estimated by the hypothesized model to the observed data (McArdle, 2009). Crucially, the flexible framework provided by SEM can allow researchers to ask questions about both intra- and inter-individual changes, which is at the heart of most developmental questions. The greatest strength of SEM is that it can accommodate both measured variables and latent variables (i.e., unobserved theoretical constructs), which allows for a hypothetically error-free underlying construct. It is important to note, however, that a latent construct is only as informative as the quality of the measured variables used to indicate the underlying factor (Little, 2013). Other major strengths of SEM include the explicit specification of the model to be tested, and the flexibility to model different developmental questions such as the nature of a developmental trajectory versus the impact of change in one variable on another (Little, 2013). This framework also affords flexibility in specifying error structures, including errors that are correlated between measurement occasions (Tisak & Tisak, 1996). The process of SEM includes conceptualizing the model, identifying the parameters to be estimated, estimating the parameters, assessing the fit of the model, and modifying the model, if necessary (Little, 2013).

Latent Growth Models. A class of SEM, Latent Growth Modeling, is particularly well suited to analyze longitudinal data in the interest of examining developmental change over time (McArdle, 2009). Specifically, the goal of a latent growth model (LGM) is to describe the trajectory of change (Duncan & Duncan, 2009; Ghisletta & McArdle, 2001). In the simplest form, this model estimates a latent intercept and a latent slope, and the variance of these factors are interpreted as individual differences in the construct over time (Duncan & Duncan, 2009).

To address questions of developmental change and individual differences in change over time, LGMs use both covariance and means structures. The covariance structure contains the information that can inform questions about individual differences in how children develop while the means structure contains information at the group level (Kievit et al., 2018). The means of the latent growth factors (latent intercept and slopes) can be measured by introducing a pseudovisible that assumes a constant score of 1 for all participants (Hancock, Harring, & Lawrence, 2013). This variable has no variance, and consequently, does not impact the rest of the model (Thompson & Green, 2013). The models in this study are second-order latent growth models (Hancock, Kuo, & Lawrence, 2001). As such, the growth factors will capture information about change in the latent constructs of interest, namely episodic memory and hippocampal subfields.

Traditional methods such as ANOVA or MANOVA require data to be both balanced (participants measured at the same time points) and complete (all data collected per participant). The present study, a cohort-sequential design, includes planned missingness and is therefore unbalanced and incomplete as subjects did not provide all data. LGMs are capable of handling both unbalanced and incomplete data and are well suited for the analyses used in this study (Hancock et al., 2013). Although missingness of data was planned in the design of the current

study (i.e., cohort-sequential; Duncan et al., 1996), attrition and data loss due to poor quality and experimenter error also contribute to missing data. Acknowledging the potential impact of missing data is important, as missing data may still have important implications for longitudinal analyses. The loss of information can lead to a decrease in the precision with which growth factors and latent constructs of the hippocampus and episodic memory are estimated. The amount of precision lost is tied to the amount of data missing. Noted by Hancock et al. (2013), under certain conditions, missing data can introduce bias into the parameter estimates. If the estimation of the parameters is biased, it limits the ability to draw connections between the conclusions implied from the model and the real world. Concerns of missing data can be addressed, to an extent, using recent methodological advances such as full information maximum likelihood (FIML) or multiple imputation (MI; e.g., Enders, 2013; Hancock et al., 2013). The current study utilized FIML in handling missing data when possible. The problem of missing data is addressed in further detail below.

Theoretically, the trajectories of the constructs of interest of this study may be best captured by a non-linear growth trajectory. Several approaches exist to model such trajectories in LGMs, such as changing the loading of the latent slope factors, including both a linear slope factor and quadratic slope factor (McArdle, 2009), or relevant to the current study, splicing together separate lines (i.e., spline) to examine if growth patterns differ across developmental time periods (Hancock et al., 2013). In the current study, the ability to detect different rates of change between developmental time points was of interest. Consequently, piecewise (i.e., spline) latent growth models (Hancock et al., 2013; Meredith & Tisak, 1990) were used. Piecewise models can accommodate variations that best suit the developmental question at hand because of their sensitivity to detect different rates of change across time. For example, these models can

include one transition point (e.g., age 6, where 4-6 and 6-8 are separate slopes) or transitions at each time point (e.g., 4-5, 5-6, 6-7, 7-8).

Piecewise LGMs come in two general forms, discontinuous and additive designs. Piecewise LGMs viewing developmental trajectories between transitions as discontinuous treat the slope factor between each time point as a new linear growth function. Piecewise LGMs viewing developmental trajectories as additive view transition points as a continuation from the first trajectory with some additional growth (Hancock et al., 2013).

As described above, an advantage of SEM is the ability to use *latent variables* in order to examine the intended construct of interest (versus measured variables). Multi-group latent growth modeling (Ghisletta & McArdle, 2001; Hancock et al., 2013) was used in *Mplus* 8.2 (Muthén & Muthén, 1998-2017) in order to address the following aims: 1) to examine developmental changes in episodic memory using a battery of episodic memory tasks; 2) to examine developmental changes in hippocampal subfields; and 3) to examine how changes in episodic memory relate to developmental changes in the volume of hippocampal subfields in 4- to 8-year-old children. Two cohorts, one starting at age 4 and the other at age 6 and overlapping at one time point, were measured at one-year intervals over a three-year period. Using this overlap, the longitudinal trajectory between age 4 to age 8 was estimated.

Preregistration of Analyses

Analyses to assess hypotheses from Aim 1 and Aim 3 of this dissertation were preregistered (<https://osf.io/s8kuq>). For Aim 1, analyses examining the ability to use lab-based tasks to indicate a latent construct, and to assess changes in this construct were preregistered. A deviation from the proposed analyses was the exclusion of the Stories task (see footnote 1). Additionally, while the inclusion of IQ as a control variable was proposed, this inclusion

ultimately worsened model fit, and the reported models exclude IQ as a covariate. Finally, practice effects were not included in the preregistered plan but were added to assess the possibility that repeated testing lead to a benefit in children's performance.

For Aim 3, the preregistered analyses, which aimed at examining the co-development of episodic memory and hippocampal subfields, included multi-cohort multidomain second-order piecewise latent growth models and an alternative analytical plan of modeling a source memory task and averaged subfield volumes over time. The multi-cohort multidomain second-order piecewise latent growth models analyses conducted deviated from the proposed approach by excluding the Stories task. The alternative analytical plan using source memory as a measure of episodic memory and composite measures of subfield volumes was utilized as proposed. A deviation of the analyses including hippocampal subfields was the inclusion of ICV as a covariate. In order to better align with the current practices in the literature and due the magnitude of differences between subfield volumes and intracranial volumes, hippocampal subfield volumes were adjusted for ICV and analyses rerun with adjusted volumes versus rerun with ICV as a covariate in the raw volume model. Across all models, preregistered proposed fit indices included CFI. However, because this metric is often not helpful in assessing model fit in LGMs, it was not considered.

Confirmatory Analyses

Modeling Episodic Memory Development. As noted above, four tasks that have been used independently in previous studies examining the development of memory during early- to middle-childhood were used as indicators of a latent construct of episodic memory (measures to be included are listed in Table 4). Each task was administered, maximally, at three different

waves for each cohort. Due to the cohort-sequential design, planned missingness is present in the data. Additional missingness in behavioral data was due to attrition caused by families leaving the area, and in rare cases, experimental error during data collection. Thus, I argue that the data meet the assumption of missing at random required for the estimation procedure of FIML, and robust maximum likelihood estimation was used (Enders, 2013). In *Mplus* 8.2, robust maximum likelihood estimation for non-complete data can be estimated with standard errors and chi-squared statistics that are robust to potential non-normality of the data (Byrne, 2012).

Confirmatory factor analysis. Confirmatory factor analysis (CFA) was utilized to test if independent episodic memory measures indicate a latent construct of episodic memory, and the extent to which the construct is invariant across time (Figure 2). Longitudinal measurement invariance was tested for configural invariance, and both partial and full weak and strong factorial invariance (Kline, 2011; Putnick & Bornstein, 2016). Configural invariance is the least stringent assumption, where the indicators of a construct are the same across measurement occasions. Weak factorial invariance makes the assumption that constructs have the same meaning across measurement occasions (Kline, 2011). This assumption can be tested by constraining the loadings of indicators of a construct to be equal (Byrne, 2012). If the model assuming weak factorial invariance does not fit statistically significantly worse than the model assuming configural invariance, one can assume constructs hold the same meaning across time (e.g., measured variables at 4-years-old and 6-years-old indicate a common episodic memory construct).

Strong factorial invariance makes the assumption that constructs have the same meaning across time and that the scale of this construct holds the same meaning across time (Putnick & Bornstein, 2016). This assumption can be tested by constraining both the factor loadings and

intercepts of indicators of a construct to be equal across time (Byrne, 2012). If the model assuming strong factorial invariance does not fit statistically significantly worse than the model assuming weak factorial invariance, one can assume constructs hold the same meaning across time and differences in indicators across time are due to developmental changes in the latent construct (e.g., a 4-year-old and 6-year-old with the same estimated latent score will have the same scores on the indicators).

In some instances, indicator parameters are invariant for some measures but variant for other measures (Millsap, 2011). While there is some debate regarding the ability to draw comparisons between latent constructs when partial, versus full, measurement invariance is supported (Kang, McNeish, & Hancock, 2016), comparisons can be made so long as the loading and intercept of one measure is invariant across measurement occasions (Hancock, Stapleton, & Arnold-Berkovits, 2009; Thompson & Green, 2013). While changes in the underlying latent construct are based on items for which partial invariance is supported, indicators that are noninvariant facilitate the identification of the latent construct of each measurement occasion (Grimm, Ram, & Estabrook, 2016). Factor structure was compared between 6-year-olds from the 4-year-old cohort and the 6-year-old cohort by constraining loadings to be equal across groups (see Trajectory Convergence below). Latent means and variances were tested for statistically significant differences to assess whether similar constructs were estimated between the cohorts.

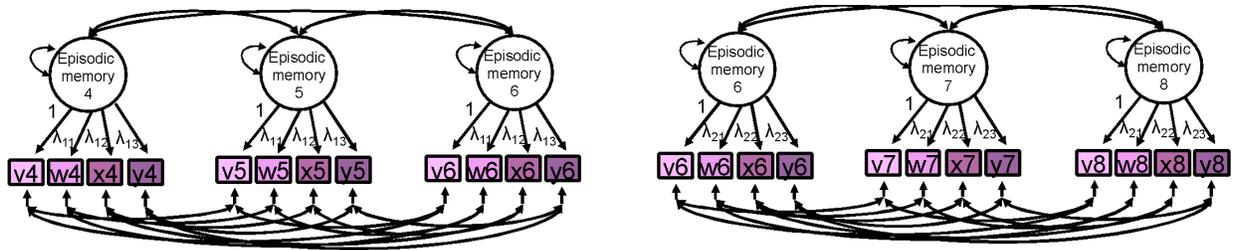


Figure 2. Conceptual Diagram of episodic memory CFA model used to test the measurement model of the indicators of a latent episodic memory variable across the age-range of interest, assuming factorial invariance within each cohort separately. *Note.* V= Temporal Order , W= Source Memory, X= Feature-Binding, Y=Primacy.

Latent growth model. Multi-cohort second-order piecewise latent growth models were used. This approach is flexible enough to accommodate non-linear change in episodic memory across time by estimating the change between each age-point, and to estimate the extent to which change differs among individuals. Previous longitudinal work on individual tasks has noted accelerated changes from approximately 5 to 7 years (e.g., Riggins, 2014), suggesting a non-linear growth function. To estimate a model that is sensitive to the possible non-linearity of episodic memory development, piecewise latent growth models were used. The first-order episodic memory factors were estimated using the final measurement model for each cohort, respectively (see Results for discussion of factorial invariance).

To estimate the latent intercepts at 4- and 6-years, loadings of the first-order episodic memory factors were constrained to 1. The loadings of episodic memory constructs on the first slope factor (e.g., change between 4- to 5-years) were constrained to 0 at wave 1, 1 at wave 2, and 1 at wave 3 to estimate the growth occurring between the initial measurement occasion and the change one year later. The loadings on the second slope factor (e.g., change between 5- to 6-

years) were constrained to 0 at wave 1, 0 at wave 2, and 1 at wave 3 to estimate the growth occurring between the second measurement occasion and the change one year later (the third measurement occasion). Intercepts of indicator variables were constrained to be equal across time points for variables established as invariant across time, reflecting that change in indicator variables starts at the same point (Hancock et al., 2001). Error variance of each measured variable (i.e., indicator) was freely estimated for episodic memory indicator variables. Error covariance parameters were estimated for each indicator across measurement waves as it is likely that other aspects of the task not explained by the latent construct of episodic memory related to each other similarly across time.

Two models were tested, one for each cohort examining the developmental trajectory of episodic memory (Figure 3). Parameters of interest included means and variances for the intercept (differences in initial episodic memory ability), and the means and variances of the slope between each age transition (e.g., the slope between age 4 and age 5, which represents the change in episodic memory ability). For example Mplus code see Appendices C and D

To ensure that changes observed were not simply the result of changes in overall cognitive ability, measures of IQ were entered as time invariant covariates to models of episodic memory development to account for global cognitive ability. Interestingly, the addition of IQ worsened the fit of the models. However, the pattern of results for the latent measure of episodic memory ability remained the same. Given the poor model fit when including IQ, and the similarity of results, only models excluding IQ are reported.

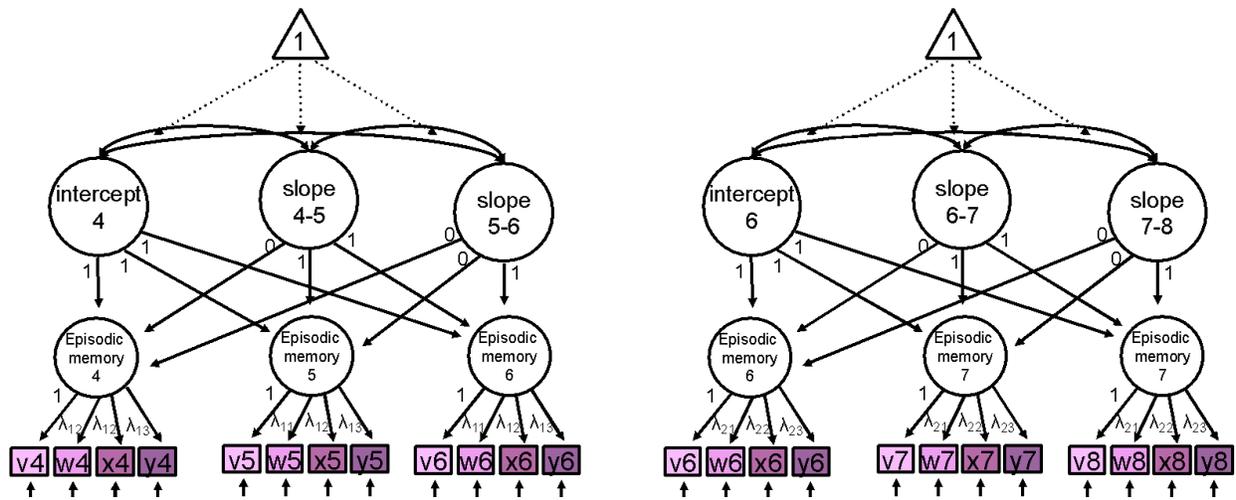


Figure 3. Conceptual diagram of second-order piecewise latent growth model for episodic memory (EM). Primary hypothesis models tested age-related changes in episodic memory ability within each cohort separately, with convergence tested at age 6 years between cohorts. Note. V= Temporal Order , W= Source Memory, X= Feature-Binding, Y=Primacy. Error covariances omitted from diagram for clarity.

Trajectory Convergence. To explore whether trajectories in each cohort converge at the 6-year knot, latent means and variances of the latent factor intercept (i.e., episodic memory or hippocampal subfields) were tested for equivalence. Specifically, intercept convergence between cohorts was tested by grouping subjects by cohort (i.e., 4-year and 6-year) and specifying model constraints to assess the average estimated intercept for both cohorts, the difference between the estimated intercept between cohorts, and the difference between the estimated variance of the intercept between cohorts. For example Mplus code see Appendix E.

Modeling Parallel Development of Episodic Memory and Hippocampal Subfields. In the current study, the extent to which one domain relates to change in the other domain is of interest was examined in addition to examining the developmental trajectories of episodic

memory and hippocampal subfields. To address this question, multi-cohort multidomain second-order piecewise latent growth models (Willet & Sayer, 1996) were first used. Specifically, both developmental trajectories of episodic memory and each hippocampal subfield within the hippocampal body were modeled to determine whether the intercepts and growth factors of brain and behavior related over time and if these relations differed between individuals (Figure 5). This model can answer questions about the relations within each domain (i.e., brain and behavior) of development, and questions about the relations *between* brain and behavior. Correlations were estimated within (e.g., the intercept of episodic memory to the slope of episodic memory), and across the domains (e.g., the intercept of episodic memory ability correlated with the intercept of a specific subfield). Error variance of each measured variable (i.e., indicator) was freely estimated. Error covariance parameters were estimated for each indicator across measurement waves. Modeling relations between intercepts and slopes both within and between domains allowed the opportunity to examine the relative strength of these relations. This resulted in 6 total models examining the relations between episodic memory ability and the hippocampus: 3 per subfield (CA2-4/DG, CA1, subiculum) in hippocampal body for each cohort. Both raw and adjusted results are reported for the cross-domain models (using the alternative approach described below).

Alternative Analytical Approach. As laid out in the preregistered analysis plan, when models converged, the second order spline models examining relations between latent measures of episodic memory and hippocampal subfields had inadmissible fit (see Table 10), possibly due to lack of fit in the measurement model or lack of fit in the structural model and were not admissible. Consequently, composite measures of hippocampal subfields and a single task thought to assess episodic memory were modeled across time by cohort. Specifically, measures

of volume in left and right hemisphere were averaged to form a variable for each subfield (i.e., CA2-4/DG, CA1, and subiculum). For episodic memory ability, a composite variable was not thought to be entirely suitable, due to the fact that these tasks are measured on different scales, administered in different formats and on different time scales, have not previously validated for use in this way, and may be unreliable. Consequently, the source memory was selected given its well-documented use in the literature as a measure of children's episodic memory ability (e.g., Drumme & Newcombe, 2002; Riggins, 2014; Riggins et al., 2018). Both cross-sectional (Riggins et al., 2018) and longitudinal (Riggins, 2014) studies employing this source memory task show age-related improvements in performance on this task during early childhood. Although it has been associated with age-related differences in CA2-4/DG and CA1 hippocampal subfields cross-sectionally (Riggins et al., 2018), this task has not previously been examined in relation to hippocampal development over time during this developmental period. To assess specificity in relations between hippocampal subfields and memory ability, composite measures of amygdala volume were also modeled by cohort in relation to the source memory task.

Because all these models used a single indicator variable, error variances of the indicator variables were constrained to 0 to allow for model identification. The resulting models were just-identified or saturated (i.e., no degrees of freedom). Consequently, fit indices for these models are perfect by default and specific hypothesis cannot be tested about the model as a whole. However, just-identified models still provide the ability to test hypotheses about relations within the model, which should be based on theory because of the inability to test the hypothesized model as a whole. Specifically, these models allowed for testing the extent to which development of hippocampal subfields relates to changes in episodic memory ability. Although these models

provide cross-domain correlations for all intercept and slope factors, to address the hypotheses of whether the extent to which changes in the hippocampus and episodic memory co-occur, only parameters for cross-domain relations between intercepts and slopes, and slopes at corresponding time points were examined (e.g., change in source memory between 4 to 5 years and change in volume between 4 to 5 years). For example Mplus code see Appendix F.

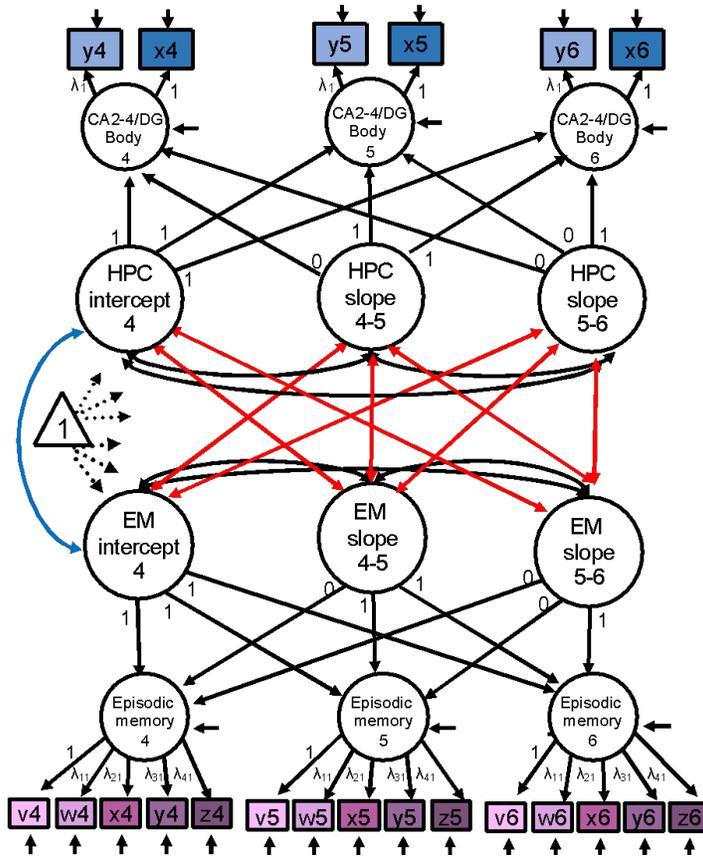


Figure 5. Conceptual diagram of multidomain second-order piecewise latent growth model for episodic memory (EM) and a selected hippocampal subfield (CA2-4/DG) in the 4-year-old cohort. Primary hypothesis models attempted to test relations between age-related changes in subfields and EM, separately for each cohort. *Note.* Error covariances omitted for clarity.

Assessing Model Fit. Although there is widespread agreement that assessing model fit is a vitally important aspect to SEM, the choice of which inferential statistic and descriptive index

to use and which values are indicative of good fit has been a highly contentious issue (see, e.g., Barrett, 2007; Hayduk, Cummings, Boadu, Pazderka-Robinson, & Boulianne, 2007; Hu & Bentler, 1998, 1999; Millsap, 2007). As noted by Preacher (2010, p. 197), “latent growth models are notoriously poor-fitting by traditional criteria,” likely because such models are highly restrictive compared to other applications of SEM that often have many free parameters, leading to unrealistically good fit. However, a lack of fit between models and measured data could be due to misspecification of the functional form of the developmental trajectory (e.g., a linear function when the trajectory is non-linear). If models do not fit to an acceptable degree, it indicates that further theoretical or explanatory work is likely needed or that the data are not well suited for second-order latent growth models. However, it is important to acknowledge that all models are inherently wrong (Hancock & Mueller, 2011). Thus, models with acceptable fit are not necessarily “truth” but those that are tenable can still provide valuable information to advance our understanding. Specifically, in this study, such models can advance our understanding of the development of episodic memory and the hippocampus.

The following measures typical of the field were included: root-mean square error of approximation ($RMSEA \leq 0.08$ supports acceptable fit; Browne & Cudeck, 1993; MacCallum, Browne, & Sugawara, 1996), and standardized root mean square residual ($SRMR \leq 0.08$ supports good fit; Hu & Bentler, 1999). For a discussion on the subjective nature of recommended cutoff criteria for fit indices in latent variable analyses, see Browne and Cudeck (1993). For a discussion of the limitations and potential limitations when considering both fit indices and measurement quality, see McNeish, An, and Hancock (2018). Parameters of interests (i.e., latent means, latent variances, latent covariances, latent and directional relations between intercept and slope factors) are reported for models with satisfactory fit.

Exploratory Analyses

Analyses related to Aim 2 of this dissertation were not preregistered as they had been conducted previously to examine the development of hippocampal subfield volumes during the period of early- to mid-childhood. While not preregistered, hypotheses for those analyses were proposed a priori based on existing cross-sectional and longitudinal research. Additionally, examination of amygdala development and its relation to episodic memory were exploratory and added to assess specificity of any possible relations observed between the hippocampal subfields and episodic memory. Results of the exploratory analyses should be taken with caution, as the possibility of false positives increases with multiple exploratory analyses. However, results still provide useful information about patterns of change that can be confirmed with future research.

Modeling Hippocampal Subfield Development. Multi-cohort second-order piecewise latent growth models were used to estimate the developmental trajectories of each hippocampal subfield within hippocampal body. Subfields in the body were modeled separately based on work showing age-related differences in the contribution of subfields to each subregion and differences in the proposed functional significance between subfields in head and body. Each subject provided neuroimaging data, maximally, at three different waves for each cohort. As noted above, planned missingness is present in the data. Additional missingness was due to attrition caused by families leaving the area, and lack of data quality due to excessive motion, a common occurrence in young participants. Consequently, I argue that the data is missing at random and FIML was utilized.

Latent constructs of hippocampal subfields in the body were identified by left and right hemisphere measures. This model construction uses the commonality of the two measures to indicate the latent construct while removing measurement error and thereby produces error-free

estimates of the hypothesized effects. At least two measures are required to indicate a latent construct apart from measurement error (i.e., left and right hemisphere volumes), and thus laterality (i.e., left versus right hemisphere) of effects cannot be tested.

To indicate latent constructs of hippocampal subfields, loadings of right hemisphere indicators (e.g., CA2-4/DG in right body at age 4, 5, and 6) were constrained to 1 at each wave to scale the construct. The loadings of left hemisphere indicators were constrained to be equal across waves (e.g., CA2-4/DG in left body age 4, 5, and 6) within cohorts. To estimate latent intercepts at 4- and 6-years, loadings of the first-order hippocampal subfield factors were constrained to 1. The loadings on the first slope factor (either between ages 4-5 or between ages 6-7) were constrained to 0 at wave 1, 1 at wave 2, and 1 at wave 3 to estimate the growth occurring between the initial measurement occasion and the change one year later. The loadings on the second slope factor (either between ages 4-5 or between ages 6-7) were constrained to 0 at wave 1, 0 at wave 2, and 1 at wave 3 to estimate the growth occurring between the second measurement occasion and the change one year later (the third measurement occasion). Intercepts of indicator variables will also be constrained to be equal across time points in order to reflect that change in indicator variables should start at the same point (Hancock et al., 2001). Error variance of each measured variable (i.e., indicator) were freely estimated except in instances where constraints facilitated model convergence (i.e., subiculum in the 4-year-old cohort). Error covariance parameters were estimated for each indicator across measurement waves, as it is likely that other aspects of the measurement tool (i.e., ASHS) not explained by the latent construct of hippocampal subfields will relate to each other.

Six models were tested, 3 per subfield (CA2-4/DG, CA1, subiculum) in hippocampal body for each cohort (Figure 4). Parameters of interest included means and variances for the

intercept (i.e., differences in initial subfield volume) and slope between each age transition (e.g., change in subfield volume between age 4 and age 5). For example Mplus code see Appendices G and H.

To ensure that changes observed were not simply the result of changes in overall head size, raw hippocampal subfield volumes were adjusted for ICV and models rerun for comparison. Only results that were significant in raw volume models were examined in ICV-adjusted volume results, as different adjustment methods can lead to spurious results (Perlaki et al., 2014; Pintzka, Hansen, Evensmoen, & Håberg, 2015). The adjustment was done using an analysis of covariance approach (Raz et al., 2005). Age and sex were used to estimate ICV values using the following formula (adjusted volume = raw volume – $b * (ICV - \text{predicted ICV})$, see Keresztes et al., 2017). Separate adjustments were performed for each wave of data collection. Results were examined with raw volumes first and then with ICV-adjusted volumes. For adjusted results, both statistical significance and patterns of results compared to raw volumes were examined. Given the high similarity between results for raw versus ICV-adjusted volumes, only the raw results are reported unless otherwise indicated.

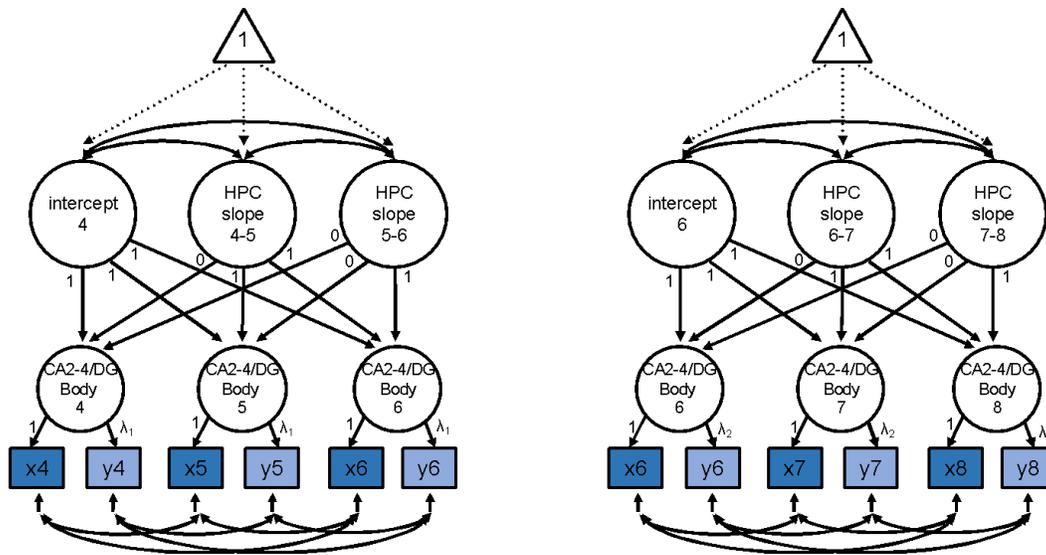


Figure 4. Conceptual diagram of second-order piecewise latent growth model for a selected hippocampal subfield (CA2-4/DG). Primary hypothesis models tested age-related changes in hippocampal subfield volume within each cohort separately with convergence tested at age 6 years between cohorts. *Note.* x= region of interest (ROI) in right hemisphere, y= ROI in left hemisphere; HPC = hippocampus.

Specificity in the Relation Between Episodic Memory and Hippocampal Subfields.

To examine the specificity of results, additional second-order latent growth models modeling development of a region outside of the hippocampus proper, the amygdala, in 4-year-old and 6-year-old cohorts were examined. To ensure that changes observed were not simply the result of changes in overall head size, raw amygdala volumes were adjusted for ICV and models rerun for comparison. The adjustment was done using an analysis of covariance approach (Raz et al., 2005). Sex was unrelated to amygdala volume; thus age was used to estimate ICV values using the same formula for hippocampal volume adjustment. Separate adjustments were performed for each wave of data collection. Results were examined with raw volumes first and then with ICV-adjusted volumes. No a priori hypotheses were made regarding the trajectory of development,

although prior work has noted greater volume in adults versus 6-year-olds, and marginally greater volume in 6-year-old versus 4-year-old children (Rice, Viscomi, Riggins, & Redcay, 2014), and increases that extend into adolescence (Herting et al., 2018). Given the high similarity between results for raw versus ICV-adjusted volumes for growth models, only the raw results are reported. Further, models examining the relation between change in amygdala volume and change in episodic memory were examined. Both raw results and the examined adjusted results are reported for the cross-domain models.

Chapter 3: Results

Preliminary Analyses – Practice Effects

Analyses examining practice effects were exploratory. Practice effects were not observed for any of the measured variables. Specifically, children of the same age tested multiple times and children tested once performed similarly on the task (e.g., 6-year-olds tested 3 times vs. 6-year-olds tested once, see Table 5).

Table 5

Summary of Differences Between Cohorts for All Indicator Variables.

Wave	Cohort	Age (years)	Wave	Cohort	Age (years)	Source Memory	Temporal Recall	Feature-binding	Primacy
2	4-year	5	1	5-year	5	0.06	-0.11	-0.18	-0.01
3	4-year	6	1	6-year	6	-0.002	0.05	0.02	-0.04
2	6-year	7	1	7-year	7	-0.01	0.004	-0.15	-0.03
3	6-year	8	1	8-year	8	-0.05	-0.02	0.02	-0.10

Note. Bold denotes significant effects at $p < .05$ for paired two-tailed t-test. Values displayed

represent the average difference between cohorts on a given task in the task metric. Positive values indicate performance in a longitudinal cohort was greater than a cross sectional cohort (e.g., 5-year-olds who had prior experience with the task at age 4 years performed better than 5-year-olds completing the task for the first time). Negative values indicate performance in a cross cohort was greater than a longitudinal cohort (e.g., 5-year-olds completing the task for the first time performed better than 5-year-olds who had prior experience with the task at age 4 years).

Measurement Invariance of Episodic Memory Tasks

Analyses examining the ability to indicate a latent construct of episodic memory were confirmatory. Configural factorial invariance of the episodic memory factor was supported such that the same factor structure was specified at each age (4, 5, 6, 7, 8) with acceptable fit (4-year-old cohort: SRMR = .079, RMSEA = .00 (90% CI = .00 - .059); 6-year-old cohort: SRMR = .067, RMSEA = .00 (90% CI = .00 - .048)).²

Strong factorial invariance was assessed. Variable loadings and intercepts were constrained to be equal across measurement occasions within each cohort. Fit indices of the final measurement model with the above mentioned constraints were acceptable (4-year-old cohort: SRMR = .107, RMSEA = .00 (90% CI = .00 - .065); 6-year-old cohort: SRMR = .103, RMSEA = .00 (90% CI = .00 - .044)). Model fit was compared using both the scaled chi-square difference test (Satorra, 2000; Satorra & Bentler, 2001) and Δ McDonald's Non-Centrality Index (NCI; for a discussion see Kang et al., 2016) and the cut-off value of 0.0085 empirically derived by Meade, Johnson, and Braddy (2008). The final measurement models did not fit worse than the configural models (4-year-old cohort Δ McDonald's NCI = -0.02, scaled $\Delta\chi^2(12) = 16.19, p = .18$; 6-year-old cohort Δ McDonald's NCI = .006, scaled $\Delta\chi^2(12) = 19.60, p = .07$). In summary, strong factorial invariance was supported, allowing for the assessment of change of a latent measure of episodic memory.

²An alternative approach to examining the similarity of the episodic memory construct between cohorts would be the use of multi-group modeling. However, convergence issues arose with this approach and, consequently, convergence at the 6-year time point was tested to provide evidence of similar constructs indicated across cohorts.

Cohort Convergence

Analyses examining convergence in the models of episodic memory were confirmatory, while analyses examining convergence of hippocampal subfield volume were exploratory. The episodic memory factor did not significantly differ between cohorts at the knot point of age 6 years ($ps > .66$). That is, the factor structure (i.e., all loadings and all intercepts constrained to be equal), mean, and variance of the estimated latent episodic memory factor did not differ between participants in the 4- and 6-year-old cohorts at 6-years-old, suggesting similar episodic memory constructs indicated within each cohort. Latent subfield factors also did not significantly differ between cohorts at the knot point of age 6 years (CA2-4/DG $ps > .39$, CA1 $ps > .38$, subiculum $ps > .07$). That is, the mean and variance of the estimated latent volume did not differ between participants in the 4- and 6-year-old cohorts at 6-years-old.

Development of Episodic Memory

Analyses examining episodic memory development were confirmatory. Fit indicators for spline models of episodic memory for each cohort were satisfactory (4-year: SRMR = .107, RMSEA = .00 (90% CI = .00 - .065); 6-year: SRMR = 0.103, RMSEA = .00 (90% CI = .00 - .044)). The developmental trajectory of episodic memory showed positive rates of change between time points (see path coefficients in Table 6). Contrary to predictions, increases were not greatest between 4-6 years. Instead, significant increases were observed across this developmental period. Although increases appeared greater between 4 to 5 years of age in the 4-year-old cohort, and between 7 to 8 years of age in the 6-year-old cohort (Figure 6), change did not statistically significantly differ between ages 4 to 5 and 5 to 6 years ($p = .40$) and between 6 to 7 years and 7 to 8 years ($p = .36$).

Regarding tests of intercept variance, children differed in initial episodic memory ability at age 4 years ($p = .047$) but did not significantly differ in initial ability at 6 years ($p = .34$). Regarding tests of slope variance, children did not significantly differ in their growth between 4 to 5 or 5 to 6 years of age ($ps > .06$), nor growth between 6 to 7 or 7 to 8 years of age ($ps > .23$). Together, these results suggest that there is significant variability in where 4-year-olds start, but that growth after that time is similar across children (both from 4 to 6 years and 6 to 8 years).

Table 6

Growth Parameters for Episodic Memory Models by Cohort.

Growth Parameter	Intercept	Slope 4-5	Slope 5-6	Slope 6-7	Slope 6-8
4-year-old cohort	.306 (.019)**	.121 (.020)**	.098 (.022)**	-	-
6-year-old cohort	.469 (.030)**	-	-	.09 (.026)**	.121 (.023)**

Note: Unstandardized path coefficients scaled to temporal recall performance (i.e., scores range from 0-1). ** $p < .01$, * $p < .05$. Coefficients were estimated within each cohort separately (i.e., 4-year-old and 6-year-old cohorts)

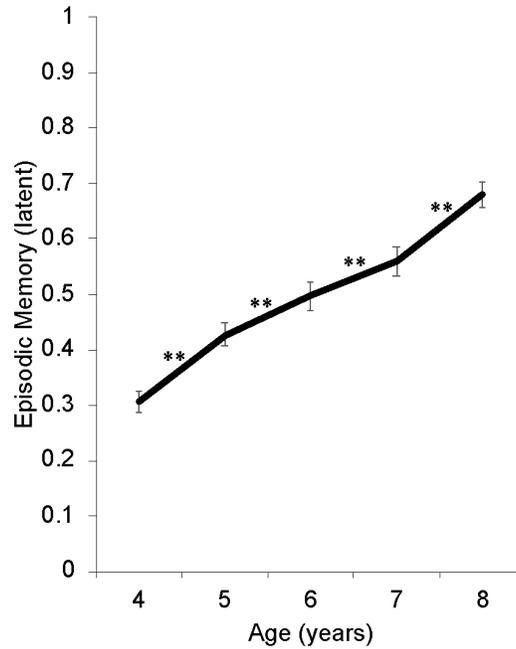


Figure 6. Growth trajectory of episodic memory ability from 4 to 8 years of age. *Note.* ** $p < .01$. Error bars represent the estimated standard error. Estimated performance at age 6 was averaged between cohorts for illustrative purposes, and analytically performance at age 6 years did not significantly differ between cohorts.

Development of Hippocampal Subfields

Analyses examining hippocampal subfield development were exploratory. Fit indicators for the spline models for each subfield and cohort were acceptable (See Table 7).

Table 7

Fit Indices for Each Subfield Model (CA2-4/DG, CA1, and Subiculum) by Cohort.

Subfield	Cohort			
	4-year-old		6-year-old	
	RMSEA (90% CI)	SRMR	RMSEA (90% CI)	SRMR
CA2-4/DG	.022 (.00-.181)	.054	.00 (.00-.146)	.051
CA1	.00 (.00-.162)	.057	.037 (.00-.165)	.041
Subiculum	.037 (.00-.185)	.120	.096 (.00-.183)	.093

Note. RMSEA = Root Mean-Square Error of Approximation; SRMR = Standardized Root Mean-Square Residual.

As noted previously in the Methods, the overall developmental patterns for subfields did not differ from models of ICV-adjusted subfield volumes. Thus, only models examining raw hippocampal volumes are reported. Of interest, change in hippocampal subfield volume was significant only between age 5 to 6 years for all subfields (see path coefficients in Table 8, Figure 7). Consistent with predictions, CA2-4/DG and CA1 volume increased during this developmental period, with robust increases in volume between 5 to 6 years. However, this effect was less robust in CA1 as it was no longer statistically significant in the ICV-adjusted model ($p > .23$). Subiculum volume also increased between 5 to 6 years. Regarding tests of intercept variance, children did not significantly differ in initial CA2-4/DG volume at 4 years ($p = .29$) but did differ in initial CA1 ($p = .042$) and subiculum volume ($p = .001$). Further, children did not differ in their initial CA2-4/DG, CA1, or subiculum volume at 6 years of age ($ps > .14$). Regarding tests of slope variance, children did not differ in their growth between 4 to 5 or 5 to 6 years of age ($ps > .18$) or in growth between 6 to 7 or 7 to 8 years of age ($ps > .16$) for any subfield in hippocampal body. Together these results suggest that children differ in initial

volume for CA1 and subiculum early in childhood but are similar in growth of CA2-4/DG, CA1, and subiculum over time.

Table 8

Growth Parameters for Each Subfield Model (CA2-4/DG, CA1, and Subiculum) by Cohort.

Growth Parameter	Intercept	Slope 4-5	Slope 5-6	Slope 6-7	Slope 6-8
<i>4-year-old cohort</i>					
CA2-4/DG	421.59 (13.60)**	-9.88 (12.97)	18.01 (9.07)*	-	-
CA1	273.39 (5.26)**	-2.37 (3.61)	7.65 (3.20)*	-	-
Subiculum	227.88 (5.51)**	-3.73 (6.29)	21.80 (6.52)*	-	-
<i>6-year-old cohort</i>					
CA2-4/DG	432.59 (8.87)**	-	-	9.96 (6.61)	.81 (6.03)
CA1	287.35 (5.41)**	-	-	1.56 (3.35)	.18 (1.08)
Subiculum	235.32 (5.82)**	-	-	-4.39 (6.20)	5.82 (13.06)

Note: Unstandardized path coefficients in mm³. **p < .01, *p < .05. Coefficients were estimated

within each cohort separately (i.e., 4-year-old and 6-year-old cohorts).

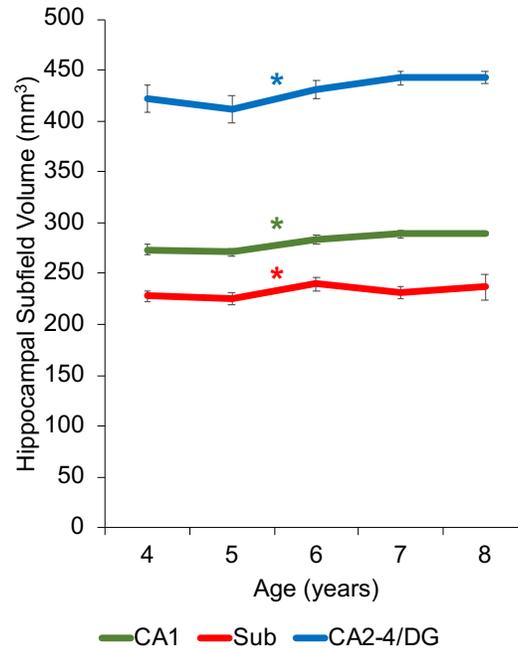


Figure 7. Growth trajectories of hippocampal subfield volumes in body from 4 to 8 years of age. *Note.* Sub = subiculum. * $p < .05$. Error bars represent the estimated standard error. Estimated volumes at age 6 were averaged between cohorts for illustrative purposes. Analytically, volumes at age 6 years did not significantly differ between cohorts for any subfield.

Amygdala Development

Analyses examining amygdala development were exploratory. Fit indices were satisfactory for spline models of amygdala development for the 6-year-old, but not the 4-year-old, cohort (4-year: RMSEA= 0.137 (90% CI = .00-.27), SRMR = 0.230; 6-year: RMSEA= 0.00 (90% CI = .00-.12), SRMR = 0.058). Consequently, the estimated parameters for the 4-year-old cohort are reported for completeness but should be interpreted with caution. The developmental trajectory of amygdala volume showed positive change between 6 to 8 years (see path coefficients in Table 9).

Table 9

Growth Parameters for Amygdala Models by Cohort.

Growth Parameter	Intercept	Slope 4-5	Slope 5-6	Slope 6-7	Slope 6-8
4-year-old cohort	1528.76 (25.19)**	46.97 (29.26)	16.09 (27.31)	-	-
6-year-old cohort	1555.82 (25.01)**	-	-	86.89 (25.17)**	78.84 (26.21)**

Note. Unstandardized path coefficients in mm³. ** $p < .01$, * $p < .05$. Coefficients were estimated within each cohort separately (i.e., 4-year-old and 6-year-old cohorts).

Although no a priori predictions regarding development of the amygdala were made, this region showed non-linear developmental growth during this period. Raw amygdala volume did not robustly change between 4 to 5 years of age nor between age 5 and 6 years. However, volume increased between 6 to 7 and 7 to 8 years of age (Figure 8). Regarding individual differences, children differed in initial raw amygdala volume at age 4 years ($p = .04$) and 6 years ($p = .01$), and growth between 6 to 7 years ($p = .04$). Children did not significantly differ in growth measuring in raw volumes between 4 to 5 ($p = .08$) or 5 to 6 years of age ($p = .09$), nor in growth between 7 to 8 years of age ($p = .74$). The mean and variance of the estimated latent volumes did not differ between participants in the 4- and 6-year-old cohorts at 6-years-old ($ps > .58$). Together these results suggest, children differ in initial amygdala volume early in childhood, but are similar in growth overtime except between 6 to 7 years.

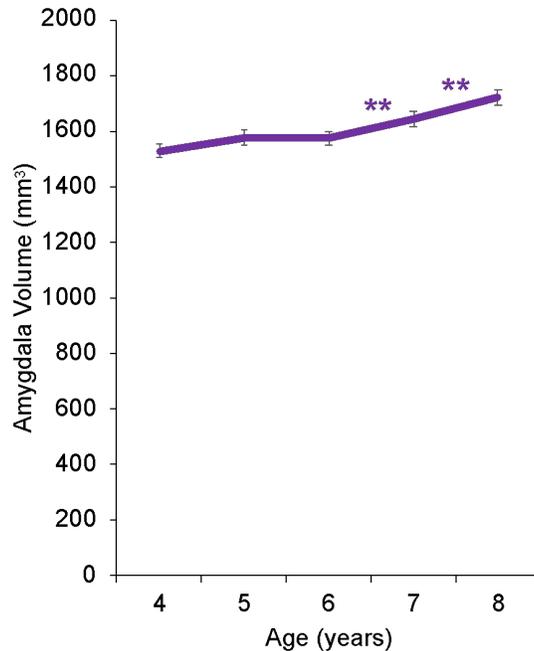


Figure 8. Growth trajectories of amygdala volume from 4 to 8 years of age. *Note.* ** $p < .01$, * $p < .05$. Error bars represent the estimated standard error. Estimated volume at age 6 was averaged between cohorts for illustrative purposes. Analytically, volumes at age 6 years did not significantly differ between cohorts for any subfield.

Parallel Development – Brain and Behavior

Analyses examining hippocampal subfield development were confirmatory. Model fit for the multi-cohort multidomain second-order piecewise latent growth models examining co-occurring development in episodic memory, and both hippocampal subfields and amygdala was inadmissible for all models (see Results below). Consequently, the preregistered proposed alternative analytical approach was used to address Aim 3 of this dissertation: examining the relation between changes in the brain and changes in behavior. Cross-domain relations between intercepts and slopes, and slopes at corresponding time points were examined (e.g., change in source memory between 4 to 5 years and change in volume between 4 to 5 years). Composite

measures of left and right hippocampal subfield and amygdala volume were modeled by cohort in relation to a single task thought to assess episodic memory: source memory. For hippocampal subfields in the 4-year-old cohort, no relations were observed between raw CA2-4/DG volume and source memory. Changes in raw CA1 volume between 4-5 years positively related to change in source memory ability between 4-5 years ($p = .02$). This positive relation was not statistically significant in the model of ICV-adjusted CA1 volume ($p = .11$), but the pattern of relations was similar to analyses using raw volumes. Changes in raw subiculum volume between 4-5 years positively related to change in source memory ability between 4-5 years ($p = .003$). This relation was also observed in the model of ICV-adjusted subiculum volume ($p = .01$). In the 6-year-old cohort, none of the examined relations between raw CA2-4/DG, CA1, or subiculum volume related to source memory. Overall, this suggests relations between CA1 and subiculum volume and source memory during early to mid-childhood.

These relations during early childhood were specific to the hippocampus. In the 4-year-old cohort, difference in initial raw amygdala volume at age 4 positively related to source memory ability at age 4 ($p = .005$) and negatively related to change in source memory between 4-5 ($p = .003$). However, these relations did not hold after adjusting volume for ICV ($ps > .8$). In the 6-year-old cohort, none of the examined relations were significant between raw amygdala volumes and source memory ($ps > .23$). In sum, changes in amygdala volume and changes in source memory volume did not relate during this developmental period. Further, given the variation between raw and ICV-adjusted volumes, this overall pattern suggests observed relations between initial amygdala volume and source memory are not especially robust, and observed relations should be noted with caution.

Chapter 4: Discussion

This dissertation examined 1) the utility of using a battery of lab-based tasks to indicate a latent construct of episodic memory ability and the developmental trajectory of episodic memory as a latent construct, 2) developmental trajectories of memory-related brain structures within the hippocampus: CA2-4/DG, CA1, and subiculum subfields, and 3) the co-development of episodic memory and hippocampal subfields in a longitudinal sample of 4- to 8-year-old children. Results revealed that lab-based tasks can be used to indicate a latent measure of episodic memory, and that this construct shows consistent developmental growth between 4 to 8 years. In addition, CA2-4/DG, CA1, and subiculum subfields of the hippocampal increased in volume between 5- to 6-years. Finally, developmental improvements in a single measure of episodic memory related to development of CA1 and subiculum hippocampal subfield volumes. Below I expand on the findings and discuss how they relate to previous developmental work. I also address the limitations of this study and suggest potential future directions to address them. I end by describing the implications of this work, which highlight early- to mid-childhood as a particularly important period for both hippocampal and memory development.

What is the trajectory of the development of episodic memory ability during early childhood?

The first aim of this dissertation sought to 1) assess episodic memory using multiple lab-based tasks and 2) examine developmental improvements in this ability over the period of early- to mid-childhood. Overall, results support the hypothesis that lab-based tasks can be used to indicate a latent construct of episodic memory. Further, measurement invariance was supported

across time points within each cohort, and similar episodic memory constructs were indicated at 6 years of age for both the 4-year-old and 6-year-old cohort, suggesting a similar latent episodic memory construct was measured across ages and cohorts. This is exciting because no prior research has examined the feasibility of using lab-based tasks longitudinally to assess the development of episodic memory as a latent construct in young children, nor characterized the developmental trajectory of episodic memory as a construct during this developmental period.

Although it was possible to indicate a latent construct from these measures, model fit (i.e., the estimated SRMR for each cohort) for most models was “satisfactory” as opposed to “good.” There are a few possibilities to explain why model fit was satisfactory with the current battery of tasks. First, bivariate correlations (see Appendix B) between tasks at a given age did not exceed .346 in the 4-year-old cohort and did not exceed .263 in the 6-year-old cohort. As noted by Little (2013, *p.* 12), a correlation of approximately .30 is at “the lower end of encouragement” when examining the indicators intended to estimate the hypothetical construct. Although low correlations are not inherently bad, one can be more certain that indicators reflect a construct when correlations between measured indicator variables are high (Little, 2013).

One possible explanation for the low correlations between tasks is the different methodological features of each task. Given that the tasks varied widely along a number of dimensions, each task potentially relied on associated cognitive resources (e.g., attention, inhibitory control) to varying degrees. More specifically, the tasks used in the current study varied in the number of stimuli included (e.g., 12 facts, 8-item and 12-item lists, 9-item sequences, 32 trials), the delay between encoding and retrieval (e.g., seconds versus minutes versus a week), and mode of administration (e.g., computer game versus physical cards versus videos). Previous cross-sectional work in 3- to 6-year-old children (Cheke & Clayton, 2015) and

adults (Cheke & Clayton, 2013) using different batteries of tasks that vary in surface features and mnemonic demands have reported low or few correlations between tasks after accounting for age. Further, while tasks used in the study by Cheke and Clayton (2015) indicated a single episodic memory construct, the authors noted that individual task performance, if considered separately, lead to different conclusions about age-related variability.

Another possible explanation of low correlations between tasks is low reliability in tests of memory ability. Very little research has considered the psychometric properties of tasks often used in episodic memory research. This prior work has argued for the possibility that low (or absent) correlations between tasks may result from low reliability, or consistency, within memory tests (Cheke & Clayton, 2015). For example, it is possible that certain questions in the utilized tasks (e.g., first half of the feature-binding versus second half) differentially measure aspects of episodic memory as a construct. Additionally, while the test-retest reliability of episodic memory tasks has not been the focus of research, possibly due to the difficulty of teasing apart practice effects, some research does suggest low reliability in memory measures in adults (Dikmen, Heaton, Grant, & Temkin, 1999). Overall, it remains an open question if the tasks used in the present study, or others focused on episodic memory, differentially measure aspects of single episodic memory construct, or measure different constructs. This is an important avenue of inquiry for the episodic memory literature, not only in developmental research, but across the lifespan.

An additional possibility is that low correlations stem from the fact that the tasks utilized in the current study were designed to focus on different “subcomponents” of episodic memory (i.e., relations between items and time, location, source of information; or recall vs recognition). Consequently, it is possible that if measures used in the present study were used in combination

with other tasks examining the same subcomponents, correlations would have been higher and model fit would have been better. This idea aligns with work in 7- to 11-year-old children and young adults showing developmental differences in subcomponents (i.e., item-item, item-space, item-time) that support children's episodic memory ability, as measured by performance on a source memory task (Lee, Wendelken, Bunge, & Ghetti, 2016). Specifically, Lee et al. (2016) showed that children reached adult-like levels of item-space memory by 9.5 years of ages, yet item-time and item-item memory improved into adulthood. They suggested that while all subcomponents support the binding of details into episodic memories, the extent to which each plays a role differs across development. Further, recent studies including younger children (< 5 years) have shown that separate tasks thought to assess different mnemonic processes that rely on the hippocampus in supporting episodic memory (relational binding and pattern separation) are unrelated (Hassevoort, Khan, Hillman, & Cohen, 2019; Ngo, Lin, Newcombe, & Olson, 2019). For example, Ngo et al. (2019) showed that 4-year-olds, 6-year-olds, and adults' performance on both a relational memory (akin to the source memory task in the current study) and a mnemonic similarity task, which assesses the ability to distinguish between highly similar memories, was not statistically significantly related. Thus, it is possible that the tasks used in the present study differentially reflect subcomponents of episodic memory. Consequently, future work would benefit from examining these considerations under a theoretical framework that includes a multi-faceted construct of episodic memory (Cheke & Clayton, 2015; Chen, Gilmore, Nelson, & McDermott, 2017).

A related limitation is that while latent factors of episodic memory were indicated at each age, not all factors were entirely distinguishable from each other due to Heywood cases observed in the models (i.e., configural and final measurement models). Specifically, within the 4-year-old

cohort, the estimated correlation between the episodic memory factor at age 4 years and 5 years exceeded 1; within the 6-year-old cohort, the estimated correlation between the episodic memory factor at age 6 years and 7 years exceeded 1. These Heywood cases remained even after scaling the variables to be of similar magnitudes. Although not surprising, as episodic memory improves into adolescence (Lee et al., 2014; Lee et al., 2020) and developmental similarities are evident in children's performance between these time points, reported results should be considered with caution.

An important direction for future research is to assess the reliability of the tasks used in the current study and/or other tasks commonly used to assess the development of episodic memory. As noted above, reliability was not explored/assessed in this dissertation. Although this approach is consistent with standards in the field, it is a notable limitation since models with indicators that have relatively poor psychometric characteristics also tend to have low standardized loadings, and are more susceptible to Heywood cases (Wothke, 1993). Further, low task reliability can lead to the underestimation of correlations between tasks due to measurement error. Knowledge of the reliability of tasks used to assess episodic memory would facilitate the ability to measure attenuated correlations of episodic memory tasks (Spearman, 1904) and to better understand the extent to which these tasks are related (i.e., address whether low correlations between tasks are due to measurement error or lack of a relation). Taken with previous discussions of the reliability and correlations of tasks thought to assess episodic memory, results from this dissertation add support for the argument that has been made previously in the literature, but has yet to be addressed, to better characterize the psychometrics of selected episodic memory tasks, improve the psychometric properties of these tasks, and/or use existing measures with good psychometric characteristics. To improve the tasks used to

assess the tasks used in episodic memory research, an approach similar to that used in recent work addressing issues of reliability of tasks used to assess attention could be taken (Draheim et al., 2019). For example, future research can work to assess reliability using 1) internal consistency of tasks when possible and 2) test-retest reliability of episodic memory tasks. Internal consistency can be measured using split-half reliability (e.g., odd and even trials). Test-retest reliability can be measured by administering the same task to individuals within a relatively short period of time (e.g., within a week or month) as the test-retest correlation presumes no true change. Although ideally the delay between assessments would be consistent, potential differences in the duration of time between assessments between individuals could be examined by including the delay as a moderating effect.

Results examining developmental change in episodic memory ability did not support the hypothesis that this ability shows non-linear increases in development in early childhood. Instead, change was similar across this developmental period. Considered in context, this work suggests that young children's episodic memory ability shows improvements that continue across this developmental period. Although the amount of change in episodic memory ability between timepoints was similar (i.e., did not statistically differ), it is possible that the cognitive and neural mechanisms that support this improvement, as well as the role of the environment, differ across this period. For example, while the consistent increases observed do not fully align with work examining the "5- to 7-year" shift in children's cognitive abilities as they enter formal schooling (i.e., ~5 years of age, Brod, Bunge, & Shing, 2017), it is important to consider the possibility that improvements in memory relate to changes in children's environment. Specifically, a study examining changes in children's cognitive control and brain function showed that 5-year-old children who entered first grade exhibited greater improvements in

executive function and greater activation in frontal regions thought to support this ability compared to 5-year-olds who remained in kindergarten (Brod et al., 2017). It could be the case that the increased demands of formalized schooling contribute to a broad array of processes beyond cognitive control, including improvements in children's ability to form detailed memories as children progress from preschool to more formal schooling. Although the current study did not specifically examine the effects of more formal (e.g., first-grade) versus play-based (e.g., kindergarten) schooling on development, an interesting future study could compare memory ability in children of the same age in different grades.

Additionally, although improvements have been observed in tasks of episodic memory beyond the "5- to 7-year shift" (e.g., source memory; Riggins, 2014), and it is well-documented that episodic memory continues to improve into adolescence and adulthood (e.g., Cycowicz et al., 2001; Lee et al., 2020), an explanation for the similar developmental change across this period is less evident based on the current episodic memory literature. However, there are numerous possibilities for the increases observed, including: 1) developmental differences in subcomponents of episodic memory, 2) developments in attention, 3) development in other brain regions and/or connections between the hippocampus and other regions, and 4) environmental changes. First, it is possible that it is better to consider episodic memory ability as consisting of multiple subcomponents that differ in their developmental trajectories, as suggested above (for review in older children see Ghetti, 2017; Lee et al., 2020; Lee et al., 2016). Second, additional facets of children's cognitive development might support children's episodic memory ability differentially during middle childhood (e.g., 7 years; for review see Schneider & Ornstein, 2019). For example, work in 6- to 8-year-old children suggests middle childhood as a transitional period for the development of episodic memory and an increased role of children's attention (Diaz,

Blankenship, & Bell, 2018). Third, improvements in episodic memory might be supported by maturation of different brain regions and/or their connections to the hippocampus. For example, a variety of research supports a role of the prefrontal cortex in strategic memory processes and monitoring processes that facilitate episodic memory encoding (for review, see Ghetti & Lee, 2011). One such study examined differences in eye-movements thought to reflect prefrontal contributions in 7- to 8-year-old children and adults and proposed that control processes and underlying neural mechanisms that support memory formation may be less developed in older children than in adults (Edgin, Liu, Hughes, Spanò, & Clark, 2019). It is likely that these processes are even less mature in younger children. Additionally, the maturation of functional connections between the hippocampus and cortex have been related to memory performance in older (6-year-old) but not younger (4-year-old) children (Riggins, Geng, Blankenship, & Redcay, 2016). Thus, adult-like connections observed in older children might differentially facilitate this ability as children enter middle childhood. Finally, it is possible that environmental impacts also play a role in the substantial improvements observed during the transition between second and third grade. For example, given that children are tasked with increasingly demanding coursework during their education (e.g., more advanced mathematical calculations), the proposed use of strategies (for review see Bjorklund, Dukes, & Brown, 2009; Schneider & Ornstein, 2019), attentional behavior, and executive functions that aid in academic success (Fuchs, Geary, Fuchs, Compton, & Hamlett, 2016) may play a role in the improvements observed in episodic memory ability across this developmental period. Additional work is needed to address and disentangle these, and other, possibilities.

Overall, results related to Aim 1 of this dissertation suggest lab-based tasks *can* be used to indicate a latent construct of episodic memory, and that age-related changes are consistent

across early to mid-childhood. However, given the variability in the battery of tasks included in the current study, future research can improve on this latent approach by selecting tasks that are similar in terms of attention, executive function, retrieval delays, and multiple subcomponents of episodic memory. In order to facilitate the use of similar tasks, it is important for the field to consider the measurement properties of tasks used in the episodic memory literature to ensure they are well designed and reliable. Furthermore, future studies assessing children's attention and/or executive functioning are needed to examine the role of additional processes that support children's ability to remember during this important developmental period. Finally, longitudinal work is also needed to draw connections to later periods of development (e.g., adolescence, early adulthood, late adulthood) using conceptually similar assessments of episodic memory in order to characterize changes in this critical ability across the lifespan.

What are the developmental trajectories of CA2-4/DG, CA1, and subiculum subfields in the hippocampal body during early- to mid-childhood?

The second aim of this dissertation sought to examine developmental trajectories of 1) CA2-4/DG, 2) CA1, and 3) subiculum hippocampal subfields over the period of early- to mid-childhood. First, results provided partial support for the hypothesis that the development of CA2-4/DG in the hippocampal body would show increases between 5 to 7 years, but not between 4 to 5 or 7 to 8 years. Specifically, increases in volume occurred between 5 to 6 years, but change in volume was non-significant between 4 to 5 years, 6 to 7 years, and 7 to 8 years. These results align with cross-sectional research showing greater volumes in older children (Keresztes et al., 2017; Schlichting et al., 2017). However, they differ from the cross-sectional subsample of the current report that showed no age-related differences in CA2-4/DG volume (Riggins et al., 2018) and from a longitudinal study showing volumetric decreases (Tamnes et al., 2018).

Second, results provided partial support for the hypothesis that the development of CA1 in the hippocampal body would show increases between 4 to 6 years, but not between 6 to 8 years. Specifically, increases in volume occurred between 5 to 6 years, but change in volume was non-significant between 4 to 5 years, 6 to 7 years, and 7 to 8 years. Though less robust, as this effect was no longer statistically significant after accounting for ICV, these results align with longitudinal research showing increases in CA1 volume earlier in development (Tamnes et al., 2018) and cross-sectional research showing greater CA1 volumes in older children (Keresztes et al., 2017). However, they differ from cross-sectional research, including the cross-sectional sample of the current report, that showed no age-related differences in CA1 volume (Riggins et al., 2018; Schlichting et al., 2017).

Third, while change in subiculum was hypothesized to be negligible based on previous cross-sectional work during this developmental period, increases were also observed in this subfield between 5 to 6 years of age. These results align with longitudinal research showing increases in subiculum volume earlier in development (Tamnes et al., 2018) and cross-sectional research showing greater subiculum volumes in older children (Keresztes et al., 2017). However, they differ from cross-sectional research, including the cross-sectional subsample of the current report, that showed smaller subiculum volumes in older children (Schlichting et al., 2017) or no age-related differences in subiculum volume (Lee et al., 2014; Riggins et al., 2018).

A limitation of these results is that, while latent measures of volume were estimatable for all subfields, Heywood cases were observed. Specifically, in the 4-year-old cohort, the correlation between latent slopes (slope 1 and slope 2) exceeded 1 in the CA2-4/DG model and the correlation exceeded 1 between volumes at age 4 and age 6 in the CA1 model. Additionally, negative residual error variances were observed, but non-significant, in the 4-year-old cohort

CA2-4/DG and subiculum models. In the 6-year-old cohort, the correlation exceeded 1 between volumes at age 6 and age 8 in the subiculum model. Additionally, negative residual error variances were observed, but non-significant, in the CA1 and CA2-4/DG models. Residual error variances were constrained in order to facilitate model convergence for the 6-year-old cohort subiculum model. These cases remained even after scaling the variables to be smaller in magnitude, and by modifying models with negative residual error variances by constraining the variance to be equal across measurement occasions. Additionally, while model fit for subiculum was satisfactory, the estimated SRMR for each cohort fell outside of the range considered as “good” fit ($\leq .08$). As estimates of development were taken approximately a year apart, and subfields continue to show developmental changes across the lifespan, it is possible that these difficulties in the model estimation arose from the similarity of measures taken so closely in time.

Interestingly, the period of time in which significant changes were observed in all subfields (5 to 6 years) aligns with the period of time during which subfields are thought to be functionally mature based on work in non-human primates (Lavenex & Banta Lavenex, 2013) and postmortem humans (Seress, 2001). Although this previous work has proposed that CA1 and subiculum mature earlier than CA2-4/DG, the present work, which is the first to focus on this developmental period using an in-vivo investigation, shows that all subfields continue to change during early- to mid-childhood. Age-related changes in subfields may be associated with structural maturation, such as synaptic growth and pruning, dendritic arborization, and vascularization (Benes, 1998; Huttenlocher & Dabholkar, 1997; Lenroot & Giedd, 2006; Utsunomiya, Takano, Okazaki, & Mitsudome, 1999). In addition, postnatal neurogenesis in the dentate gyrus of the hippocampus may also contribute to volumetric changes, as animal models

propose that, until at least 5 years of age, immature granule cells accumulate and there are higher rates of dendritic development and synaptic formation (Eckenhoff & Rakic, 1988; Josselyn & Frankland, 2012; Lavenex & Banta Lavenex, 2013; Seress, 2001). However, current methodological limitations restrict the ability to assess which underlying processes contribute to the developmental changes observed.

Overall, results related to Aim 2 of this dissertation highlights the importance of examining change in each of the examined subfields during early- to mid-childhood, specifically between 5 to 6 years of age. The most interesting comparison of these results (as noted above) is that they differ from findings reported in Riggins et al. (2018) that examined the cross-sectional subsample of children from the current study. Specifically, whereas no age-related differences in CA2-4/DG, CA1, or subiculum in hippocampal body were observed in the cross-sectional subsample of participants from the current report, changes were observed in all of these subfields in the full accelerated longitudinal sample. This highlights the importance of longitudinal studies for detecting slight, but significant, developmental changes in hippocampal volume while also assessing the amount of change between periods of time in order to characterize trajectories of change. While developmentally trajectories visually appeared to differ (Figure 6), these differences were not statistically tested as it was not possible to examine development of all subfields in the same model. However, critically, all examined subfields showed statistically significant change during a developmentally important period of time, 5 to 6 years.

Unlike Riggins et al. (2018), this dissertation did not examine subfields in hippocampal head. Given age-related variations were observed in the cross-sectional subsample, this is an important area for future work in order to characterize potential differences in subfield development along the longitudinal axis of the hippocampus (i.e., subregions), which shows

differential developmental trajectories likely due to distribution of subfields (Gogtay et al., 2006).

Do developmental CA2-4/DG, CA1, and subiculum subfields in the hippocampal body relate to episodic memory development?

The final aim of this dissertation sought to assess the relation between episodic memory development and 1) CA2-4/DG, 2) CA1, and 3) subiculum hippocampal subfield development over the period of early- to mid-childhood.

Results did not support the hypothesis that CA2-4/DG would relate to episodic memory (using source memory as a measure), as relations were not observed in either the 4-year-old or 6-year-old cohort between change in volume or source memory performance nor in initial volume and source memory performance. Although this finding was surprising based on findings from Riggins et al. (2018) and other cross-sectional reports (Daugherty et al., 2017; Lee et al., 2014), it does align with other developmental cross-sectional literature. Specifically, Schlichting et al. (2017) did not show a relation between volume of CA3/DG and either a statistical learning or associative memory task in a cross-sectional sample of 6- to 30-year-olds. Further, Keresztes et al. (2017) utilized a maturational index of hippocampal subfield development, with a focus on CA3/DG, and did not observe a relation between subfield maturation and performance on an associative memory task but did observe a relation to performance on a mnemonic discrimination task. Interestingly, mnemonic discrimination tasks are proposed to reflect the neurocomputational process of pattern separation, and subsequently thought to rely on the CA3/DG subfields (Rolls, 2013). Additionally, research in children (Canada et al., 2019) and adults (for review see Yassa & Stark, 2011) has shown that performance on mnemonic discrimination tasks relate to CA2-4/DG subfields. Keresztes et al. (2017) also discussed the

possibility that successful performance on their source memory task also requires frontal control, as source memory related to their maturational index of frontal cortices but not the index of hippocampal maturity. Further, they proposed that the process of pattern completion, thought to be facilitated by connections between CA1 and CA3 (Rolls, 2013), may more be implicated in supporting performance on a source memory task. Therefore, it is possible that tasks designed to assess the formation of finely grained detailed memories relate to change in CA2-4/DG subfields over time.

Findings partially support the hypothesis that developmental changes in CA1 would relate to episodic memory (using source memory as a measure). Specifically, although changes in raw CA1 volume between age 4 to 5 years related to changes in source memory performance between age 4 to 5 years, this result was not robust, as this effect was marginal after adjusting raw subfield volume by ICV. Additional relations were not observed in either the 4-year-old or 6-year-old cohort between change in volume or source memory performance nor initial volume and source memory performance. While not as robust as the relation between source memory and the subiculum, this finding aligns with previous cross-sectional work that has noted a relation between CA1 volume and tasks thought to assess episodic memory (Lee et al., 2014; Schlichting et al., 2017; Riggins et al., 2018; Tamnes et al., 2014). Although the current study differs in its approach to examining change co-occurring at each time point, versus across a developmental period, the finding a similar pattern of results in raw and adjusted volume results suggest a role of changes in CA1 in supporting children's changing episodic memory ability. Additional longitudinal work would serve to strengthen support for this claim.

The hypothesis that developmental changes in subiculum would not show relations to episodic memory (using source memory as a measure) was not supported. Instead, changes in

subiculum volume between age 4 to 5 years were related to changes in source memory performance between age 4 to 5 years. Interestingly, the relation between source memory and subiculum was the most robust, as it held both for raw and ICV-adjusted subfield volumes. Although this finding differed from the cross-sectional sample in a previous report (Riggins et al., 2018), it does align with other measures of memory ability in the developmental cross-sectional literature. For example, in Riggins et al., (2018), subiculum volume related to a measure of the number of extra-experimental errors children made (e.g., attributing a fact learned within the task to an outside source, such as a parent). Additionally, differences in subiculum volume have been related to a measure of source memory corrected for false alarms (item-color discrimination; Lee et al., 2014) and performance on a statistical learning task, also thought to tax the hippocampus (Schlichting et al., 2017).

Although findings of a relation between memory and the subiculum were not expected, the role of subiculum in memory remains poorly understood (Ketz, Morkonda, & O'Reilly, 2013; Liang & Preston, 2015). Additional work is needed to better characterize the structural and functional relations between changes in children's episodic memory ability and the subiculum over time. However, a reasonable explanation for relations between changes in source memory performance and both CA1 and subiculum volume during the period of 4-5 years is that this relation reflects a role of the monosynaptic pathway of the hippocampus. Within the hippocampus, CA1 and subiculum comprise a tightly coupled monosynaptic pathway, with CA1 serving as the input of information and subiculum serving as the major output to the cortex. Further, CA1 and the subiculum serve as the major outputs to cortical and subcortical regions that work with the hippocampus to support episodic memory (Ledergerber & Moser, 2017). Work examining hippocampal function in both animals (for review see O'Mara, 2005) and adult

humans (Suthana, Ekstrom, Moshirvaziri, Knowlton, & Bookheimer, 2011) has implicated subiculum (specifically dorsal subiculum, akin to subiculum in the body) in the retrieval of information and CA1 in the encoding of that information. Overall, it is possible that observed relations between CA1 and subiculum and source memory during this period are reflective of a reliance on the monosynaptic pathway and its outputs to support young children's episodic memory. Specifically, it has been previously argued that subiculum subfields support episodic memory earlier in development followed by development of CA1, with these subfields proposed to support memory more via generalizations and pattern completion (Keresztes, Ngo, Lindenberger, Werkle-Bergner, & Newcombe, 2018). Conversely, CA2-4/DG is thought to be functionally mature later in development and is implicated in the trisynaptic pathway that facilitates the neurocomputational process of pattern separation (described in Discussion above).

Finally, although relations were observed between source memory and CA1 and subiculum volume in hippocampal body, future work is needed to expand upon and better characterize the functional implications of these findings. First, it is possible that relations between subfields and episodic memory differ within the head of hippocampus. This is especially true given research in older children and adults examining relations between both the structure and function of hippocampal subregions (i.e., head, body, tail) have reported relations between performance on episodic memory tasks and variability in hippocampal head and tail, but not hippocampal body (DeMaster et al., 2014; Sastre III, Wendelken, Lee, Bunge, & Ghetti, 2016). These findings may result from variability in specific subfields that are disproportionately distributed along the longitudinal axis and further support the need to examine development of subfields of the hippocampus during earlier periods of development. While relations between hippocampal structure (i.e., volume) and performance on memory tasks are thought to reflect

functional changes in these regions, future work using functional magnetic resonance imaging is needed to confirm this.

Overall, this dissertation highlights that, within the hippocampal body, CA1 and subiculum relate to improvements in children's episodic memory (as measured by source memory) and supports the conclusion that the hippocampus and episodic memory co-develop during early childhood. While different from results of the cross-sectional report (Riggins et al., 2018), the approach in the current study examined relations of change between domains for each time-period. It is possible that this approach more accurately reflects the relation between the hippocampus and episodic memory ability by considering the amount of change occurring in each domain between time point, versus across the entire period.

Assessing specificity of results related to the hippocampus

Both the developmental trajectory of the amygdala and relations between changes in source memory performance and the amygdala were examined to assess the extent to which the findings detailed above are specific to subfields of the hippocampus. Although no a priori predictions were made regarding amygdala development, significant increases in volume were observed between 6 to 7 and 7 to 8 years of age. It is worth noting that the fit of the spline model in the 4-year-old cohort was relatively poor. I propose two possibilities for lack of acceptable model fit for amygdala development in this cohort. First, the measure of amygdala volumes in the current study are coarser than those of the hippocampus. To expand, this means that although, like the hippocampus, the amygdala *can* be examined as a homogenous structure, it is better considered as heterogeneous because it consists of different nuclei that vary in their developmental trajectories (Campbell et al., 2019). It is possible that differences in the developmental trajectories of the nuclei are present during the period of early childhood,

something not considered in the current study. Another possibility relates to the quality of the total amygdala segmentations used in the current study, especially in younger children. No existing quality control protocol currently within the lab for amygdala segmentations, and additional measurement error may be present in these segmentations (i.e., it is closely situated near the hippocampus, and could include voxels from this, or other, adjacent structures). Although all scans from which segmentations were extracted passed quality control checks, segmentation specific checks do not exist in current protocols, and it is possible that amygdala volumes are noisier, especially in younger children, resulting in worse model fit. Although some caution should be noted, overall, these findings showing increases in total amygdala volume between 6 to 8 years suggests that the significant developmental changes observed between 5 to 6 years in hippocampal subfields are not reflective of general changes across the brain during this time. These results are similar to past work comparing amygdala volumes in 4-year-olds, 6-year-olds, and adults that shows greater volume in both left and right amygdala volumes in adults compared to 6-year-olds, but only marginally larger left amygdala volume in 6-year-olds compared to 4-year-olds (Rice et al., 2014).

In regard to associations between amygdala development and change in source memory performance, although relations were observed with raw amygdala volumes and source memory performance, this pattern of relations was no longer observed when accounting for changes in the size of participants' heads (i.e., adjusting volume for ICV). Adjusted volumes are used to assess the robustness of relations observed using raw volumes. Consequently, only relations that appeared in the raw volumes were compared to results from adjusted amygdala volumes and source memory. As a whole, these findings suggest specificity in the relation between

developmental changes in hippocampal subfield volumes and changes in performance on the source memory task thought to assess episodic memory ability during early childhood.

General Limitations

Although this dissertation advances our understanding of measures used to assess episodic memory, the development of episodic memory, the development of hippocampal subfields, and the co-development of brain and behavior forward, it has several limitations. First, the volumetric measure used to assess hippocampal maturity is not a direct measure of hippocampal function. While structural variability is thought to relate to individual differences in function and memory ability (e.g., Carr et al., 2017), the current work does not provide direct evidence of whether the observed volumetric changes in subfields have functional implications during this period of development. The reason function was not directly assessed was due to the difficulty of collecting neuroimaging data within a population for whom staying still is a demanding task. However, with improvements in scanning methods (e.g., multiband and compressed sense), future work may be able to examine hippocampal subfield structure and function within the same developmental population. Additionally, although the accelerated longitudinal design allows for the assessment of change overtime, the current study was limited in only including one overlapping time point between cohorts. In order to better assess change over time, future research might consider implementing planned missingness that incorporates multiple overlapping time points, and potentially additional cohorts to expand the period of time under-investigation. Further, given the disproportional distribution of subfields along the longitudinal axis (e.g., CA1 and CA3; Duvernoy, 2005), it is likely that subfields of the hippocampus contribute to the different developmental trajectories of hippocampal subregions (e.g., Gogtay et al., 2006). As the current study limited investigation to subfields within the

hippocampal body, future work is needed to examine trajectories along the longitudinal axis (i.e., in the hippocampal head) and relations with episodic memory. Finally, the protocol adapted and used to segment hippocampal subfields in the present study (La Joie et al., 2010; Riggins et al., 2018), while well-suited to the neuroimaging measures collected (e.g., T2 image), combines the DG subfield with several other smaller subfields (CA2, CA3, and CA4) that are not always considered together in the literature. As such, while CA subfields make up a smaller proportion of the combined CA2-4/DG region, the developmental trajectory of DG may be impacted by the inclusion of these regions. Ongoing efforts by the Hippocampal Subfields Group (<http://hippocampalsubfields.com>) to create a reliable and harmonized subfield tracing protocol in both the head and the body of the hippocampus are underway (Wisse et al., 2017). Future work examining the development of these critically important subunits of the hippocampus should strive to adopt a protocol best suited to the questions at hand that also facilitates comparison with research conducted by other groups.

Conclusions

In conclusion, this dissertation demonstrates the ability to use tasks in the laboratory to characterize developmental changes in episodic memory as a latent construct, highlights the developmental changes in hippocampal subfields occurring between 5 to 6 years, and suggests specificity of the role of the hippocampus in supporting episodic memory. Future work can build upon this foundation by designing and incorporating psychometrically sound tasks in memory research and expanding examinations of the brain and memory to gain insight into how this critical ability and its neural correlates change over the human lifespan.

Data Availability Statement

Data used for this research are available upon request. Code for implementing the analyses in MPlus is available in the appendices and on the osf website corresponding to this project (<https://osf.io/s8kuq>).

Appendices

Appendix A

Table A1.

Counterbalancing Across Participants and Waves of Data Collections

	Wave 1	Wave 2	Wave 3
Primacy	Presentation of 4 lists CB for presentation.	Lists reordered (offset by 2) and CB as in wave 1.	Lists reordered (offset by 3) and CB as in wave 1.
Ordered Recall	Pet Shop → Fair → Park sequences CB for sequence use and run order.	Lists reordered (run order offset by 1) and CB as in wave 1.	Lists reordered (run order offset by 1) and CB as in wave 1.
Feature- Binding	No CB. Randomly assigned version E or F.	No CB. Randomly assigned version E or F.	No CB. Randomly assigned version E or F.
Source Memory	Novel fact lists CB (1,2,3) for version (A/B) and (Puppet/Person).	Novel fact lists (1→2→3→1) CB for version (A/B) and (Puppet/Person).	Novel fact lists (1→2→3→1) CB for version (A/B) and (Puppet/Person).
IQ	No CB. Age appropriate task administered only at wave 1.	N/A	N/A

Note. CB = Counterbalancing.

Appendix B

Table B1.

Four-year-old Cohort Correlation Matrix of Indicator Variables.

	AP4	AP5	AP6	SOURCE 4	SOURCE 5	SOURCE 6	LORS 4	LORS 5	LORS 6	PRIM 4	PRIM 5	PRIM 6
AP4	1	-	-	-	-	-	-	-	-	-	-	-
AP5	0.202	1	-	-	-	-	-	-	-	-	-	-
AP6	0.151	0.284	1	-	-	-	-	-	-	-	-	-
SOURCE 4	0.199	0.064	0.263	1	-	-	-	-	-	-	-	-
SOURCE 5	0.366	0.091	0.215	0.613	1	-	-	-	-	-	-	-
SOURCE 6	0.139	-0.026	0.062	0.232	0.290	1	-	-	-	-	-	-
LORS4	0.310	0.218	0.281	0.214	0.305	-0.181	1	-	-	-	-	-
LORS5	0.158	0.230	0.220	0.068	0.168	-0.091	0.246	1	-	-	-	-
LORS6	0.033	0.346	0.275	0.14	0.366	0.075	0.217	0.267	1	-	-	-
PRIM4	0.346	0.210	0.098	0.287	0.260	0.159	0.167	0.091	0.130	1	-	-
PRIM5	0.215	0.100	0.011	0.333	0.328	0.155	0.119	0.128	0.201	0.052	1	-
PRIM6	0.174	0.323	0.255	0.147	0.108	0.242	0.077	0.201	0.238	0.246	0.273	1

Note. AP = Temporal Recall, SOURCE = Source Memory, LORS = Feature-Binding task, PRIM = Primacy.

Table B2.

Six-year-old Cohort Correlation Matrix of Indicator Variables.

	AP6	AP7	AP8	SOURCE 6	SOURCE 7	SOURCE 8	LORS 6	LORS 7	LORS 8	PRIM 6	PRIM 7	PRIM 8
AP6	1	-	-	-	-	-	-	-	-	-	-	-
AP7	0.338	1	-	-	-	-	-	-	-	-	-	-
AP8	0.286	0.298	1	-	-	-	-	-	-	-	-	-
SOURCE 6	0.230	0.370	0.055	1	-	-	-	-	-	-	-	-
SOURCE 7	0.024	0.255	0.212	0.473	1	-	-	-	-	-	-	-
SOURCE 8	0.317	0.215	0.134	0.257	0.287	1	-	-	-	-	-	-
LORS6	0.029	0.246	0.034	0.068	0.166	0.144	1	-	-	-	-	-
LORS7	0.090	0.166	0.048	0.202	-0.012	-0.066	0.428	1	-	-	-	-
LORS8	0.145	0.232	0.151	0.063	-0.050	0.129	0.492	0.440	1	-	-	-
PRIM6	0.204	-0.016	0.047	0.263	0.204	0.263	0.123	0.050	0.254	1	-	-
PRIM7	0.231	0.259	0.208	0.025	0.125	0.057	0.162	0.100	0.166	0.055	1	-
PRIM8	0.190	-0.005	0.111	0.196	0.009	0.048	0.049	0.065	0.036	0.200	0.131	1

Note. AP = Temporal Recall, SOURCE = Source Memory, LORS = Feature-Binding task, PRIM = Primacy.

Appendix C

Episodic Memory Final Model Mplus code – 4-year-old cohort

TITLE:

Behavioral Latent 4-yo cohort

DATA:

FILE IS Younger.csv;

VARIABLE:

NAMES ARE Sub_ID Cohort Gender BlockIQ VocabIQ

AP4 AP5 AP6 AP7 AP8

Source4 Source5 Source6 Source7 Source8

Lors4 Lors5 Lors6 Lors7 Lors8

LP84 LP85 LP86 LP87 LP88

LP124 LP125 LP126 LP127 LP128

Prim4 Prim5 Prim6 Prim7 Prim8

CMS4 CMS5 CMS6 CMS7 CMS8

ICV4 ICV5 ICV6 ICV7 ICV8

DGhdr4_adj DGhdr5_adj DGhdr6_adj DGhdr7_adj DGhdr8_adj

DGhdl4_adj DGhdl5_adj DGhdl6_adj DGhdl7_adj DGhdl8_adj

DGbodr4_adj DGbodr5_adj DGbodr6_adj DGbodr7_adj DGbodr8_adj

DGbodl4_adj DGbodl5_adj DGbodl6_adj DGbodl7_adj DGbodl8_adj

CA1hdr4_adj CA1hdr5_adj CA1hdr6_adj CA1hdr7_adj CA1hdr8_adj

CA1hdl4_adj CA1hdl5_adj CA1hdl6_adj CA1hdl7_adj CA1hdl8_adj

CA1bodr4_adj CA1bodr5_adj CA1bodr6_adj CA1bodr7_adj CA1bodr8_adj

CA1bodl4_adj CA1bodl5_adj CA1bodl6_adj CA1bodl7_adj CA1bodl8_adj

Subhdr4_adj Subhdr5_adj Subhdr6_adj Subhdr7_adj Subhdr8_adj

Subhdl4_adj Subhdl5_adj Subhdl6_adj Subhdl7_adj Subhdl8_adj

Subbodr4_adj Subbodr5_adj Subbodr6_adj Subbodr7_adj Subbodr8_adj

Subbodl4_adj Subbodl5_adj Subbodl6_adj Subbodl7_adj Subbodl8_adj

DGhdr4 DGhdr5 DGhdr6 DGhdr7 DGhdr8

DGhdl4 DGhdl5 DGhdl6 DGhdl7 DGhdl8

DGbodr4 DGbodr5 DGbodr6 DGbodr7 DGbodr8

DGbodl4 DGbodl5 DGbodl6 DGbodl7 DGbodl8

CA1hdr4 CA1hdr5 CA1hdr6 CA1hdr7 CA1hdr8

CA1hdl4 CA1hdl5 CA1hdl6 CA1hdl7 CA1hdl8

CA1bodr4 CA1bodr5 CA1bodr6 CA1bodr7 CA1bodr8

CA1bodl4 CA1bodl5 CA1bodl6 CA1bodl7 CA1bodl8

Subhdr4 Subhdr5 Subhdr6 Subhdr7 Subhdr8

Subhdl4 Subhdl5 Subhdl6 Subhdl7 Subhdl8

Subbodr4 Subbodr5 Subbodr6 Subbodr7 Subbodr8

Subbodl4 Subbodl5 Subbodl6 Subbodl7 Subbodl8;

USEVARIABLES ARE AP4-AP6 Source4-Source6 Lors4-Lors6 Prim4-Prim6;
MISSING IS ALL (-999);

ANALYSIS:

ESTIMATOR IS MLR;
ITERATIONS = 100000;
SDITERATIONS = 10000;
HIITERATIONS = 10000;
CONVERGENCE = .001;
H1CONVERGENCE = .001;

MODEL:

EPISOD4 BY AP4@1
 Source4 (aa)
 Lors4 (bb)
 Prim4 (cc);
EPISOD5 BY AP5@1
 Source5 (aa)
 Lors5 (bb)
 Prim5 (cc);
EPISOD6 BY AP6@1
 Source6 (aa)
 Lors6 (bb)
 Prim6 (cc);

interc BY EPISOD4-EPISOD6@1;
slope1 BY EPISOD4@0 EPISOD5@1 EPISOD6@1;
slope2 BY EPISOD4@0 EPISOD5@0 EPISOD6@1;
[EPISOD4-EPISOD6@0];
EPISOD4-EPISOD6@0;
[interc-slope2];

[AP4@0];
[AP5@0];
[AP6@0];

[Source4] (ee);
[Source5] (ee);
[Source6] (ee);

[Lors4] (ff);
[Lors5] (ff);
[Lors6] (ff);

[Prim4] (gg);
[Prim5] (gg);

[Prim6] (gg);

AP4;
AP5;
AP6;

Source4;
Source5;
Source6;

Lors4;
Lors5;
Lors6;

Prim4;
Prim5;
Prim6;

Source4 with Source5 Source6;
Source5 with Source6;
AP4 with AP5 AP6;
AP5 with AP6;
Lors4 with Lors5 Lors6;
Lors5 with Lors6;
Prim4 with Prim5 Prim6;
Prim5 with Prim6;

OUTPUT:
SAMPSTAT TECH4 MODINDICES(3.641);

Appendix D

Episodic Memory Final Model Mplus code – 6-year-old cohort

TITLE:

Behavioral Latent 6-yo cohort

DATA:

FILE IS Older.csv;

VARIABLE:

NAMES ARE Sub_ID Cohort Gender BlockIQ VocabIQ

AP4 AP5 AP6 AP7 AP8

Source4 Source5 Source6 Source7 Source8

Lors4 Lors5 Lors6 Lors7 Lors8

LP84 LP85 LP86 LP87 LP88

LP124 LP125 LP126 LP127 LP128

Prim4 Prim5 Prim6 Prim7 Prim8

CMS4 CMS5 CMS6 CMS7 CMS8

ICV4 ICV5 ICV6 ICV7 ICV8

DGhdr4_adj DGhdr5_adj DGhdr6_adj DGhdr7_adj DGhdr8_adj

DGhdl4_adj DGhdl5_adj DGhdl6_adj DGhdl7_adj DGhdl8_adj

DGbodr4_adj DGbodr5_adj DGbodr6_adj DGbodr7_adj DGbodr8_adj

DGbodl4_adj DGbodl5_adj DGbodl6_adj DGbodl7_adj DGbodl8_adj

CA1hdr4_adj CA1hdr5_adj CA1hdr6_adj CA1hdr7_adj CA1hdr8_adj

CA1hdl4_adj CA1hdl5_adj CA1hdl6_adj CA1hdl7_adj CA1hdl8_adj

CA1bodr4_adj CA1bodr5_adj CA1bodr6_adj CA1bodr7_adj CA1bodr8_adj

CA1bodl4_adj CA1bodl5_adj CA1bodl6_adj CA1bodl7_adj CA1bodl8_adj

Subhdr4_adj Subhdr5_adj Subhdr6_adj Subhdr7_adj Subhdr8_adj

Subhdl4_adj Subhdl5_adj Subhdl6_adj Subhdl7_adj Subhdl8_adj

Subbodr4_adj Subbodr5_adj Subbodr6_adj Subbodr7_adj Subbodr8_adj

Subbodl4_adj Subbodl5_adj Subbodl6_adj Subbodl7_adj Subbodl8_adj

DGhdr4 DGhdr5 DGhdr6 DGhdr7 DGhdr8

DGhdl4 DGhdl5 DGhdl6 DGhdl7 DGhdl8

DGbodr4 DGbodr5 DGbodr6 DGbodr7 DGbodr8

DGbodl4 DGbodl5 DGbodl6 DGbodl7 DGbodl8

CA1hdr4 CA1hdr5 CA1hdr6 CA1hdr7 CA1hdr8

CA1hdl4 CA1hdl5 CA1hdl6 CA1hdl7 CA1hdl8

CA1bodr4 CA1bodr5 CA1bodr6 CA1bodr7 CA1bodr8

CA1bodl4 CA1bodl5 CA1bodl6 CA1bodl7 CA1bodl8

Subhdr4 Subhdr5 Subhdr6 Subhdr7 Subhdr8

Subhdl4 Subhdl5 Subhdl6 Subhdl7 Subhdl8

Subbodr4 Subbodr5 Subbodr6 Subbodr7 Subbodr8

Subbodl4 Subbodl5 Subbodl6 Subbodl7 Subbodl8;

USEVARIABLES ARE AP6-AP8 Source6-Source8 Lors6-Lors8 Prim6-Prim8;
MISSING IS ALL (-999);

ANALYSIS:
ESTIMATOR IS MLR;
ITERATIONS = 100000;
SDITERATIONS = 10000;
HIITERATIONS = 10000;
CONVERGENCE = .001;
H1CONVERGENCE = .001;

MODEL:
EPISOD6 BY AP6@1
 Source6 (aa)
 Lors6 (bb)
 Prim6(cc);
EPISOD7 BY AP7@1
 Source7 (aa)
 Lors7 (bb)
 Prim7(cc);
EPISOD8 BY AP8@1
 Source8 (aa)
 Lors8 (bb)
 Prim8!(cc);

interc BY EPISOD6-EPISOD8@1;
slope1 BY EPISOD6@0 EPISOD7@1 EPISOD8@1;
slope2 BY EPISOD6@0 EPISOD7@0 EPISOD8@1;
[EPISOD6-EPISOD8@0];
EPISOD6-EPISOD8@0;
[interc-slope2];

[AP6@0];
[AP7@0];
[AP8@0];

[Source6] (ee);
[Source7] (ee);
[Source8] (ee);

[Lors6] (ff);
[Lors7] (ff);
[Lors8] (ff);

[Prim6] (gg);
[Prim7] (gg);

[Prim8] (gg);

AP6;

AP7;

AP8;

Source6;

Source7;

Source8;

Lors6;

Lors7;

Lors8;

Prim6;

Prim7;

Prim8;

Source6 with Source7 Source8;

Source7 with Source8;

AP6 with AP7 AP8;

AP7 with AP8;

Lors6 with Lors7 Lors8;

Lors7 with Lors8;

Prim6 with Prim7 Prim8;

Prim7 with Prim8;

OUTPUT:

SAMPSTAT TECH4 MODINDICES(3.841);

Appendix E

Example convergence code (Episodic Memory)

TITLE:

Behavioral Convergence

DATA:

FILE IS CONVERGE CHECK.csv;

VARIABLE:

NAMES ARE Sub_ID Cohort AgeGroup

Gender BlockIQ VocabIQ

AP4 AP5 AP6 AP7 AP8

Source4 Source5 Source6 Source7 Source8

Lors4 Lors5 Lors6 Lors7 Lors8

LP84 LP85 LP86 LP87 LP88

LP124 LP125 LP126 LP127 LP128

Prim4 Prim5 Prim6 Prim7 Prim8

CMS4 CMS5 CMS6 CMS7 CMS8

ICV4 ICV5 ICV6 ICV7 ICV8

DGhdr4_adj DGhdr5_adj DGhdr6_adj DGhdr7_adj DGhdr8_adj

DGhdl4_adj DGhdl5_adj DGhdl6_adj DGhdl7_adj DGhdl8_adj

DGbodr4_adj DGbodr5_adj DGbodr6_adj DGbodr7_adj DGbodr8_adj

DGbodl4_adj DGbodl5_adj DGbodl6_adj DGbodl7_adj DGbodl8_adj

CA1hdr4_adj CA1hdr5_adj CA1hdr6_adj CA1hdr7_adj CA1hdr8_adj

CA1hdl4_adj CA1hdl5_adj CA1hdl6_adj CA1hdl7_adj CA1hdl8_adj

CA1bodr4_adj CA1bodr5_adj CA1bodr6_adj CA1bodr7_adj CA1bodr8_adj

CA1bodl4_adj CA1bodl5_adj CA1bodl6_adj CA1bodl7_adj CA1bodl8_adj

Subhdr4_adj Subhdr5_adj Subhdr6_adj Subhdr7_adj Subhdr8_adj

Subhdl4_adj Subhdl5_adj Subhdl6_adj Subhdl7_adj Subhdl8_adj

Subbodr4_adj Subbodr5_adj Subbodr6_adj Subbodr7_adj Subbodr8_adj

Subbodl4_adj Subbodl5_adj Subbodl6_adj Subbodl7_adj Subbodl8_adj

DGhdr4 DGhdr5 DGhdr6 DGhdr7 DGhdr8

DGhdl4 DGhdl5 DGhdl6 DGhdl7 DGhdl8

DGbodr4 DGbodr5 DGbodr6 DGbodr7 DGbodr8

DGbodl4 DGbodl5 DGbodl6 DGbodl7 DGbodl8

CA1hdr4 CA1hdr5 CA1hdr6 CA1hdr7 CA1hdr8

CA1hdl4 CA1hdl5 CA1hdl6 CA1hdl7 CA1hdl8

CA1bodr4 CA1bodr5 CA1bodr6 CA1bodr7 CA1bodr8

CA1bodl4 CA1bodl5 CA1bodl6 CA1bodl7 CA1bodl8

Subhdr4 Subhdr5 Subhdr6 Subhdr7 Subhdr8

Subhdl4 Subhdl5 Subhdl6 Subhdl7 Subhdl8

Subbodr4 Subbodr5 Subbodr6 Subbodr7 Subbodr8

Subbodl4 Subbodl5 Subbodl6 Subbodl7 Subbodl8;

USEVARIABLES ARE AP6 Source6 Lors6 Prim6;
Grouping is AgeGroup(1=young 2=old);
MISSING IS ALL (-999);

ANALYSIS:
ESTIMATOR IS ML;
ITERATIONS = 100000;
SDITERATIONS = 10000;
H1ITERATIONS = 10000;
CONVERGENCE = .001;
H1CONVERGENCE = .001;

MODEL:
EPISOD6 BY AP6@1
 Source6 (aa)
 Lors6 (bb)
 Prim6 (cc);

intEM BY EPISOD6@1;
[EPISOD6@0];
EPISOD6@0;
[intEM];
[AP6@0];
[Source6](a);
[Lors6](b);
[Prim6](c);

AP6;
Source6;
Lors6;
Prim6;

intEM;

MODEL young:

EPISOD6 BY AP6@1
 Source6 (aa)
 Lors6 (bb)
 Prim6 (cc);

intEM BY EPISOD6@1;
[EPISOD6@0];

EPISOD6@0;
[intEM](yintercEM);
[AP6@0];
[Source6](a);
[Lors6](b);
[Prim6](c);

AP6;
Source6;
Lors6;
Prim6;

intEM (yvarintEM);

MODEL old:

EPISOD6 BY AP6@1
 Source6 (aa)
 Lors6 (bb)
 Prim6 (cc);

intEM BY EPISOD6@1;
[EPISOD6@0];
EPISOD6@0;
[intEM](ointercEM);
[AP6@0];
[Source6](a);
[Lors6](b);
[Prim6](c);

AP6;
Source6;
Lors6;
Prim6;

intEM (ovarintEM);

MODEL CONSTRAINT:
NEW(avgintEM diffintEM diffvariEM);

avgintEM = (yintercEM + ointercEM)/2;
diffintEM = (yintercEM - ointercEM);
diffvariEM = (yvarintEM - ovarintEM);

OUTPUT: SAMPSTAT;

Appendix F

Example parallel development code (source memory and subfield volume)

TITLE:

Behavioral Parallel CA1 Source

DATA:

FILE IS YOUNGER.csv;

VARIABLE:

NAMES ARE Sub_ID Cohort AgeGroup

Gender BlockIQ VocabIQ

AP4 AP5 AP6 AP7 AP8

Source4 Source5 Source6 Source7 Source8

Lors4 Lors5 Lors6 Lors7 Lors8

LP84 LP85 LP86 LP87 LP88

LP124 LP125 LP126 LP127 LP128

Prim4 Prim5 Prim6 Prim7 Prim8

CMS4 CMS5 CMS6 CMS7 CMS8

ICV4 ICV5 ICV6 ICV7 ICV8

DGhdr4_adj DGhdr5_adj DGhdr6_adj DGhdr7_adj DGhdr8_adj

DGhdl4_adj DGhdl5_adj DGhdl6_adj DGhdl7_adj DGhdl8_adj

DGbodr4_adj DGbodr5_adj DGbodr6_adj DGbodr7_adj DGbodr8_adj

DGbodl4_adj DGbodl5_adj DGbodl6_adj DGbodl7_adj DGbodl8_adj

CA1hdr4_adj CA1hdr5_adj CA1hdr6_adj CA1hdr7_adj CA1hdr8_adj

CA1hdl4_adj CA1hdl5_adj CA1hdl6_adj CA1hdl7_adj CA1hdl8_adj

CA1bodr4_adj CA1bodr5_adj CA1bodr6_adj CA1bodr7_adj CA1bodr8_adj

CA1bodl4_adj CA1bodl5_adj CA1bodl6_adj CA1bodl7_adj CA1bodl8_adj

Subhdr4_adj Subhdr5_adj Subhdr6_adj Subhdr7_adj Subhdr8_adj

Subhdl4_adj Subhdl5_adj Subhdl6_adj Subhdl7_adj Subhdl8_adj

Subbodr4_adj Subbodr5_adj Subbodr6_adj Subbodr7_adj Subbodr8_adj

Subbodl4_adj Subbodl5_adj Subbodl6_adj Subbodl7_adj Subbodl8_adj

DGhdr4 DGhdr5 DGhdr6 DGhdr7 DGhdr8

DGhdl4 DGhdl5 DGhdl6 DGhdl7 DGhdl8

DGbodr4 DGbodr5 DGbodr6 DGbodr7 DGbodr8

DGbodl4 DGbodl5 DGbodl6 DGbodl7 DGbodl8

CA1hdr4 CA1hdr5 CA1hdr6 CA1hdr7 CA1hdr8

CA1hdl4 CA1hdl5 CA1hdl6 CA1hdl7 CA1hdl8

CA1bodr4 CA1bodr5 CA1bodr6 CA1bodr7 CA1bodr8

CA1bodl4 CA1bodl5 CA1bodl6 CA1bodl7 CA1bodl8

Subhdr4 Subhdr5 Subhdr6 Subhdr7 Subhdr8

Subhdl4 Subhdl5 Subhdl6 Subhdl7 Subhdl8

Subbodr4 Subbodr5 Subbodr6 Subbodr7 Subbodr8

Subbodl4 Subbodl5 Subbodl6 Subbodl7 Subbodl8;

USEVARIABLES ARE SOURCE4-SOURCE6 AP4-AP6 CA1body4 CA1body5
CA1body6;
AUXILIARY ARE CA1bodr4-CA1bodr6 CA1bodl4-CA1bodl6;
MISSING IS ALL (-999);

DEFINE:

CA1bodr4 = CA1bodr4/100;
CA1bodr5 = CA1bodr5/100;
CA1bodr6 = CA1bodr6/100;

CA1bodl4 = CA1bodl4/100;
CA1bodl5 =CA1bodl5/100;
CA1bodl6 =CA1bodl6/100;

CA1body4 = (CA1bodr4+CA1bodl4)/2;
CA1body5 = (CA1bodr5+CA1bodl5)/2;
CA1body6 = (CA1bodr6+CA1bodl6)/2;

ANALYSIS:

ESTIMATOR IS MLR;
ITERATIONS = 10000;
SDITERATIONS = 10000;
HIITERATIONS = 10000;
CONVERGENCE = .001;
H1CONVERGENCE = .001;

MODEL:

interc BY CA1BODY4-CA1BODY6@1;
slope1 BY CA1BODY4@0 CA1BODY5@1 CA1BODY6@1;
slope2 BY CA1BODY4@0 CA1BODY5@0 CA1BODY6@1;
[CA1BODY4-CA1BODY6@0];
CA1BODY4-CA1BODY6@0;
[interc-slope2];

intercb BY SOURCE4-SOURCE6@1;
slope1b BY SOURCE4@0 SOURCE5@1 SOURCE6@2;
slope2b BY SOURCE4@0 SOURCE5@0 SOURCE6@1;

[intercb slope1b slope2b];

[SOURCE4@0];

[SOURCE5@0];
[SOURCE6@0];

SOURCE4@0;
SOURCE5@0;
SOURCE6@0;

interc; slope1; slope2; intercb; slope1b; slope2b

intercb WITH interc;
intercb WITH slope1;
slope1b WITH slope1;
slope1b WITH interc;

OUTPUT:
SAMPSTAT; MODINDICES(ALL); TECH4;

Appendix H

Example hippocampal subfield Mplus code – 4-year-old cohort

TITLE:

DGbody spline 4-6

DATA:

FILE IS Younger.csv;

VARIABLE:

NAMES ARE Sub_ID Cohort Gender BlockIQ VocabIQ

AP4 AP5 AP6 AP7 AP8

Source4 Source5 Source6 Source7 Source8

Lors4 Lors5 Lors6 Lors7 Lors8

LP84 LP85 LP86 LP87 LP88

LP124 LP125 LP126 LP127 LP128

Prim4 Prim5 Prim6 Prim7 Prim8

CMS4 CMS5 CMS6 CMS7 CMS8

ICV4 ICV5 ICV6 ICV7 ICV8

DGhdr4_adj DGhdr5_adj DGhdr6_adj DGhdr7_adj DGhdr8_adj

DGhdl4_adj DGhdl5_adj DGhdl6_adj DGhdl7_adj DGhdl8_adj

DGbodr4_adj DGbodr5_adj DGbodr6_adj DGbodr7_adj DGbodr8_adj

DGbodl4_adj DGbodl5_adj DGbodl6_adj DGbodl7_adj DGbodl8_adj

CA1hdr4_adj CA1hdr5_adj CA1hdr6_adj CA1hdr7_adj CA1hdr8_adj

CA1hdl4_adj CA1hdl5_adj CA1hdl6_adj CA1hdl7_adj CA1hdl8_adj

CA1bodr4_adj CA1bodr5_adj CA1bodr6_adj CA1bodr7_adj CA1bodr8_adj

CA1bodl4_adj CA1bodl5_adj CA1bodl6_adj CA1bodl7_adj CA1bodl8_adj

Subhdr4_adj Subhdr5_adj Subhdr6_adj Subhdr7_adj Subhdr8_adj

Subhdl4_adj Subhdl5_adj Subhdl6_adj Subhdl7_adj Subhdl8_adj

Subbodr4_adj Subbodr5_adj Subbodr6_adj Subbodr7_adj Subbodr8_adj

Subbodl4_adj Subbodl5_adj Subbodl6_adj Subbodl7_adj Subbodl8_adj

DGhdr4 DGhdr5 DGhdr6 DGhdr7 DGhdr8

DGhdl4 DGhdl5 DGhdl6 DGhdl7 DGhdl8

DGbodr4 DGbodr5 DGbodr6 DGbodr7 DGbodr8

DGbodl4 DGbodl5 DGbodl6 DGbodl7 DGbodl8

CA1hdr4 CA1hdr5 CA1hdr6 CA1hdr7 CA1hdr8

CA1hdl4 CA1hdl5 CA1hdl6 CA1hdl7 CA1hdl8

CA1bodr4 CA1bodr5 CA1bodr6 CA1bodr7 CA1bodr8

CA1bodl4 CA1bodl5 CA1bodl6 CA1bodl7 CA1bodl8

Subhdr4 Subhdr5 Subhdr6 Subhdr7 Subhdr8

Subhdl4 Subhdl5 Subhdl6 Subhdl7 Subhdl8

Subbodr4 Subbodr5 Subbodr6 Subbodr7 Subbodr8

Subbodl4 Subbodl5 Subbodl6 Subbodl7 Subbodl8;

USEVARIABLES ARE DGbodr4-DGbodr6 DGbodl4-DGbodl6;
MISSING IS ALL (-999);

DEFINE:

DGbodr4 = DGbodr4/10;

DGbodr5 = DGbodr5/10;

DGbodr6 = DGbodr6/10;

DGbodl4 = DGbodl4/10;

DGbodl5 = DGbodl5/10;

DGbodl6 = DGbodl6/10;

ANALYSIS:

ESTIMATOR IS MLR;

ITERATIONS = 100000;

SDITERATIONS = 10000;

HIITERATIONS = 10000;

CONVERGENCE = .001;

H1CONVERGENCE = .001;

MODEL:

DGBODY4 BY DGbodr4@1

DGbodl4 (a);

DGBODY5 BY DGbodr5@1

DGbodl5 (a);

DGBODY6 BY DGbodr6@1

DGbodl6 (a);

interc BY DGBODY4-DGBODY6@1;

slope1 BY DGBODY4@0 DGBODY5@1 DGBODY6@1;

slope2 BY DGBODY4@0 DGBODY5@0 DGBODY6@1;

[DGBODY4-DGBODY6@0];

DGBODY4-DGBODY6@0;

[interc-slope2];

[DGbodr4@0];

[DGbodr5@0];

[DGbodr6@0];

[DGbodl4] (b);

[DGbodl5] (b);

[DGbodl6] (b);

DGbodl4-DGbodl6 ;

DGbodr4-DGbodr6 ;

DGbodl4 WITH DGbodl5 DGbodl6;

DGbodl5 WITH DGbodl6;

DGbodr4 WITH DGbodr5 DGbodr6;
DGbodr5 WITH DGbodr6;

interc; slope1; slope2;
interc WITH slope1 slope2;
slope1 WITH slope2;

OUTPUT:
SAMPSTAT; MODINDICES(ALL); TECH4;

Appendix G

Example hippocampal subfield Mplus code – 6-year-old cohort

TITLE:

DGbody spline 6-8

DATA:

FILE IS Older.csv;

VARIABLE:

NAMES ARE Sub_ID Cohort Gender BlockIQ VocabIQ
AP4 AP5 AP6 AP7 AP8
Source4 Source5 Source6 Source7 Source8
Lors4 Lors5 Lors6 Lors7 Lors8
LP84 LP85 LP86 LP87 LP88
LP124 LP125 LP126 LP127 LP128
Prim4 Prim5 Prim6 Prim7 Prim8
CMS4 CMS5 CMS6 CMS7 CMS8

ICV4 ICV5 ICV6 ICV7 ICV8

DGhdr4_adj DGhdr5_adj DGhdr6_adj DGhdr7_adj DGhdr8_adj
DGhdl4_adj DGhdl5_adj DGhdl6_adj DGhdl7_adj DGhdl8_adj
DGbodr4_adj DGbodr5_adj DGbodr6_adj DGbodr7_adj DGbodr8_adj
DGBodl4_adj DGBodl5_adj DGBodl6_adj DGBodl7_adj DGBodl8_adj
CA1hdr4_adj CA1hdr5_adj CA1hdr6_adj CA1hdr7_adj CA1hdr8_adj
CA1hdl4_adj CA1hdl5_adj CA1hdl6_adj CA1hdl7_adj CA1hdl8_adj
CA1bodr4_adj CA1bodr5_adj CA1bodr6_adj CA1bodr7_adj CA1bodr8_adj
CA1bodl4_adj CA1bodl5_adj CA1bodl6_adj CA1bodl7_adj CA1bodl8_adj
Subhdr4_adj Subhdr5_adj Subhdr6_adj Subhdr7_adj Subhdr8_adj
Subhdl4_adj Subhdl5_adj Subhdl6_adj Subhdl7_adj Subhdl8_adj
Subbodr4_adj Subbodr5_adj Subbodr6_adj Subbodr7_adj Subbodr8_adj
Subbodl4_adj Subbodl5_adj Subbodl6_adj Subbodl7_adj Subbodl8_adj
DGhdr4 DGhdr5 DGhdr6 DGhdr7 DGhdr8
DGhdl4 DGhdl5 DGhdl6 DGhdl7 DGhdl8
DGbodr4 DGbodr5 DGbodr6 DGbodr7 DGbodr8
DGBodl4 DGBodl5 DGBodl6 DGBodl7 DGBodl8
CA1hdr4 CA1hdr5 CA1hdr6 CA1hdr7 CA1hdr8
CA1hdl4 CA1hdl5 CA1hdl6 CA1hdl7 CA1hdl8
CA1bodr4 CA1bodr5 CA1bodr6 CA1bodr7 CA1bodr8
CA1bodl4 CA1bodl5 CA1bodl6 CA1bodl7 CA1bodl8
Subhdr4 Subhdr5 Subhdr6 Subhdr7 Subhdr8
Subhdl4 Subhdl5 Subhdl6 Subhdl7 Subhdl8
Subbodr4 Subbodr5 Subbodr6 Subbodr7 Subbodr8
Subbodl4 Subbodl5 Subbodl6 Subbodl7 Subbodl8;

USEVARIABLES ARE DGbodr6-DGbodr8 DGbodl6-DGbodl8;
MISSING IS ALL (-999);

DEFINE:

DGbodr6 = DGbodr6/10;

DGbodr7 = DGbodr7/10;

DGbodr8 = DGbodr8/10;

DGbodl6 = DGbodl6/10;

DGbodl7 = DGbodl7/10;

DGbodl8 = DGbodl8/10;

ANALYSIS:

ESTIMATOR IS MLR;

ITERATIONS = 100000;

SDITERATIONS = 10000;

HIITERATIONS = 10000;

CONVERGENCE = .001;

H1CONVERGENCE = .001;

MODEL:

DGBODY6 BY DGbodr6@1

DGbodl6 (a);

DGBODY7 BY DGbodr7@1

DGbodl7 (a);

DGBODY8 BY DGbodr8@1

DGbodl8 (a);

interc BY DGBODY6-DGBODY8@1;

slope1 BY DGBODY6@0 DGBODY7@1 DGBODY8@1;

slope2 BY DGBODY6@0 DGBODY7@0 DGBODY8@1;

[DGBODY6-DGBODY8@0];

DGBODY6-DGBODY8@0;

[interc-slope2];

[DGbodr6@0];

[DGbodr7@0];

[DGbodr8@0];

[DGbodl6] (b);

[DGbodl7] (b);

[DGbodl8] (b);

DGbodl6-DGbodl8 ;

DGbodr6-DGbodr8 ;

DGbodl6 WITH DGbodl7 DGbodl8;

DGbodl7 WITH DGbodl8;

DGboDr6 WITH DGboDr7 DGboDr8;
DGboDr7 WITH DGboDr8;

interc; slope1; slope2;
interc WITH slope1 slope2;
slope1 WITH slope2;

OUTPUT:
SAMPSTAT; MODINDICES(ALL); TECH6;

References

- Alden, J. T. (1994). *Development of memory for temporal order*. (Unpublished doctoral dissertation). University of Minnesota, Minneapolis, MN.
- Amaral, D., & Lavenex, P. (2006). Hippocampal Neuroanatomy. In P. Andersen, R. Morris, D. Amaral, T. Bliss, & J. O'Keefe (Eds.), *The hippocampus book* (pp. 37–114). New York, NY: Oxford University Press.
- Baddeley, A. (1992). Working memory. *Science*, *255*(5044), 556–559.
<https://doi.org/10.1126/science.1736359>
- Baddeley, A. D., & Hitch, G. (1974). Working memory. *Psychology of Learning and Motivation*, *8*, 47–89. [https://doi.org/10.1016/S0079-7421\(08\)60452-1](https://doi.org/10.1016/S0079-7421(08)60452-1)
- Barrett, P. (2007). Structural equation modelling: Adjudging model fit. *Personality and Individual Differences*, *42*(5), 815–824. <https://doi.org/10.1016/j.paid.2006.09.018>
- Bauer, P. J. (2008). Toward a neuro-developmental account of the development of declarative memory. *Developmental Psychobiology*, *50*(1), 19–31. <https://doi.org/10.1002/dev.20265>
- Bauer, P. J., Dikmen, S., Heaton, R., Mungas, D., Slotkin, J., & Beaumont, J. (2013). III. NIH Toolbox Cognition Battery (CB): Measuring episodic memory. *Monographs of the Society for Research in Child Development*, *78*(4), 34–48.
<https://doi.org/10.1111/mono.12033>
- Bauer, P. J., Doydum, A. O., Pathman, T., Larkina, M., Güler, O. E., & Burch, M. (2012). It's all about location, location, location: Children's memory for the "where" of personally experienced events. *Journal of Experimental Child Psychology*, *113*(4), 510–522.
<https://doi.org/10.1016/j.jecp.2012.06.007>
- Benes, F. M. (1998). Brain development, VII: Human brain growth spans decades. *The American Journal of Psychiatry*, *155*(11), 1489. <https://doi.org/10.1176/ajp.155.11.1489>

- Berron, D., Vieweg, P., Hochkeppler, A., Pluta, J. B., Ding, S. L., Maass, A., ... & Wolbers, T. (2017). A protocol for manual segmentation of medial temporal lobe subregions in 7 Tesla MRI. *NeuroImage: Clinical*, *15*, 466–482.
<https://doi.org/10.1016/j.nicl.2017.05.022>
- Bjorklund, D. F., Dukes, C., & Brown, R. D. (2009). The development of memory strategies. In M. L. Courage & N. Cowan (Eds.), *Studies in developmental psychology. The development of memory in infancy and childhood*. (pp. 145–175). Hove, East Sussex, UK: Psychology Press.
- Brambilla, M., Manenti, R., Ferrari, C., & Cotelli, M. (2015). Better together: Left and right hemisphere engagement to reduce age-related memory loss. *Behavioural Brain Research*, *293*, 125–133. <https://doi.org/10.1016/j.bbr.2015.07.037>
- Brod, G., Bunge, S. A., & Shing, Y. L. (2017). Does one year of schooling improve children's cognitive control and alter associated brain activation?. *Psychological Science*, *28*(7), 967-978. <https://doi.org/10.1177/0956797617699838>
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.
- Buñuel, L. (1983). *My Last Sigh*. New York, NY: Knopf.
- Byrne, B. M. (2012). *Structural equation modeling with Mplus: Basic concepts, applications, and programming*. New York, NY: Routledge.
- Campbell, C. E., Mezher, A. F., Eckel, S. P., Tyszka, J. M., Pauli, W. M., Nagel, B. J., & Herting, M. M. (2019). Restructuring of amygdala subregion apportion across adolescence. *bioRxiv* 690875. <https://doi.org/10.1101/690875>

- Canada, K. L., Ngo, C. T., Newcombe, N. S., Geng, F., & Riggins, T. (2019). It's all in the details: Relations between young children's developing pattern separation abilities and hippocampal subfield volumes. *Cerebral Cortex*, *29*(8), 3427-3433.
<https://doi.org/10.1093/cercor/bhy211>
- Carr, V. A., Bernstein, J. D., Favila, S. E., Rutt, B. K., Kerchner, G. A., & Wagner, A. D. (2017). Individual differences in associative memory among older adults explained by hippocampal subfield structure and function. *Proceedings of the National Academy of Sciences*, *114*(45), 12075-12080. <https://doi.org/10.1073/pnas.1713308114>
- Cheke, L. G., & Clayton, N. S. (2013). Do different tests of episodic memory produce consistent results in human adults?. *Learning & Memory*, *20*(9), 491-498.
<https://doi.org/10.1101/lm.030502.113>
- Cheke, L. G., & Clayton, N. S. (2015). The six blind men and the elephant: Are episodic memory tasks tests of different things or different tests of the same thing?. *Journal of Experimental Child Psychology*, *137*, 164-171. <https://doi.org/10.1016/j.jecp.2015.03.006>
- Chen, H. Y., Gilmore, A. W., Nelson, S. M., & McDermott, K. B. (2017). Are there multiple kinds of episodic memory? An fMRI investigation comparing autobiographical and recognition memory tasks. *Journal of Neuroscience*, *37*(10), 2764-2775.
<https://doi.org/10.1523/jneurosci.1534-16.2017>
- Cohen, M. J. (1997). *Children's Memory Scale Manual*. San Antonio, Texas: The Psychological Corporation Harcourt Brace and Company.
- Cycowicz, Y. M., Friedman, D., Snodgrass, J. G., & Duff, M. (2001). Recognition and source memory for pictures in children and adults. *Neuropsychologia*, *39*(3), 255-267.
[https://doi.org/10.1016/S0028-3932\(00\)00108-1](https://doi.org/10.1016/S0028-3932(00)00108-1)

- Daugherty, A. M., Flinn, R., & Ofen, N. (2017). Hippocampal CA3-dentate gyrus volume uniquely linked to improvement in associative memory from childhood to adulthood. *NeuroImage, 153*, 75–85. <https://doi.org/10.1016/j.neuroimage.2017.03.047>
- Daugherty, A. M., Yu, Q., Flinn, R., & Ofen, N. (2015). A reliable and valid method for manual demarcation of hippocampal head, body, and tail. *International Journal of Developmental Neuroscience, 41*, 115–122. <https://doi.org/10.1016/j.ijdevneu.2015.02.001>
- Davachi, L. (2006). Item, context and relational episodic encoding in humans. *Current Opinion in Neurobiology, 16*(6), 693–700. <https://doi.org/10.1016/j.conb.2006.10.012>
- Davachi, L., Mitchell, J. P., & Wagner, A. D. (2003). Multiple routes to memory: distinct medial temporal lobe processes build item and source memories. *Proceedings of the National Academy of Science on the United States of America, 100*(4), 2157–2162. <http://doi.org/10.1073/pnas.0337195100>
- DeMaster, D. M., & Ghetti, S. (2013). Developmental differences in hippocampal and cortical contributions to episodic retrieval. *Cortex, 49*(6), 1482–1493. <https://doi.org/10.1016/j.cortex.2012.08.004>
- DeMaster, D., Pathman, T., Lee, J. K., & Ghetti, S. (2014). Structural development of the hippocampus and episodic memory: Developmental differences along the anterior/posterior axis. *Cerebral Cortex, 24*(11), 3036–3045. <https://doi.org/10.1093/cercor/bht160>
- Diana, R. A., Yonelinas, A. P., & Ranganath, C. (2007). Imaging recollection and familiarity in the medial temporal lobe: a three-component model. *Trends in Cognitive Sciences, 11*(9), 379–386. <https://doi.org/10.1016/j.tics.2007.08.001>

- Diaz, A., Blankenship, T. L., & Bell, M. A. (2018). Episodic memory in middle childhood: Age, brain electrical activity, and self-reported attention. *Cognitive Development, 47*, 63-70. <https://doi.org/10.1016/j.cogdev.2018.03.003>
- Dikmen, S. S., Heaton, R. K., Grant, I., & Temkin, N. R. (1999). Test-retest reliability and practice effects of expanded Halstead-Reitan Neuropsychological Test Battery. *Journal of the International Neuropsychological Society : JINS, 5*(4), 346–356.
- Draheim, C., Tsukahara, J. S., Martin, J., Mashburn, C., & Engle, R. W. (2019). A toolbox approach to improving the measurement of attention control. *PsyArXiv*. <https://doi.org/10.31234/osf.io/q985d>
- Drumme, A. B., & Newcombe, N. S. (2002). Developmental changes in source memory. *Developmental Science, 5*(4), 502–513. <https://doi.org/10.1111/1467-7687.00243>
- Duncan, S. C., Duncan, T. E., & Hops, H. (1996). Analysis of longitudinal data within accelerated longitudinal designs. *Psychological Methods, 1*(3), 236–248. <https://doi.org/10.1037/1082-989X.1.3.236>
- Duncan, T. E., & Duncan, S. C. (2009). The ABC's of LGM: an introductory guide to latent variable growth curve modeling. *Social and Personality Psychology Compass, 3*(6), 979–991. <https://doi.org/10.1111/j.1751-9004.2009.00224.x>
- Duvernoy, H. (1998). *The hippocampus book: Functional anatomy, vascularization and serial sections with MRI* (2nd ed.). Berlin: Springer-Verlag.
- Duvernoy, H. (2005). Sectional Anatomy and Magnetic Resonance Imaging. In, *The hippocampus book: Functional anatomy, vascularization and serial sections with MRI* (3rd ed., pp. 129–217). Berlin: Springer-Verlag.

- Eckenhoff, M. F., & Rakic, P. (1988). Nature and fate of proliferative cells in the hippocampal dentate gyrus during the life span of the rhesus monkey. *Journal of Neuroscience*, 8(8), 2729-2747. <https://doi.org/10.1523/JNEUROSCI.08-08-02729.1988>
- Edgin, J. O., Liu, Y., Hughes, K., Spanò, G., & Clark, C. A. (2019). The “eyes have it,” but when in development?: The importance of a developmental perspective in our understanding of behavioral memory formation and the hippocampus. *Hippocampus*. <https://doi.org/10.1002/hipo.23149>
- Eichenbaum, H. (2000). A cortical–hippocampal system for declarative memory. *Nature Reviews Neuroscience*, 1(1), 41–50. <https://doi.org/10.1038/35036213>
- Eichenbaum, H., & Cohen, N. J. (2001). *From conditioning to conscious recollection: memory systems of the brain*. New York, NY: Oxford University Press.
- Enders, C. K. (2013). Analyzing structural equation models with missing data. In G. R. Hancock & R. O. Mueller, (Eds.), *Structural equation modeling. A second course* (2nd ed., pp. 493–519). Charlotte, NC: Information Age Publishing, Inc.
- Friedman, W. J. (1992). Children's time memory: The development of a differentiated past. *Cognitive Development*, 7(2), 171–187. [https://doi.org/10.1016/0885-2014\(92\)90010-O](https://doi.org/10.1016/0885-2014(92)90010-O)
- Fuchs, L. S., Geary, D. C., Fuchs, D., Compton, D. L., & Hamlett, C. L. (2016). Pathways to third-grade calculation versus word-reading competence: Are they more alike or different?. *Child Development*, 87(2), 558-567. <https://doi.org/10.1111/cdev.1247>
- Ghetti, S. (2017). Development of item–space and item–time binding. *Current Opinion in Behavioral Sciences*, 17, 211-216. <https://doi.org/10.1016/j.cobeha.2017.09.002>

- Ghetti, S., DeMaster, D. M., Yonelinas, A. P., & Bunge, S. A. (2010). Developmental differences in medial temporal lobe function during memory encoding. *Journal of Neuroscience*, *30*(28), 9548–9556. <https://doi.org/10.1523/JNEUROSCI.3500-09.2010>
- Ghetti, S., & Lee, J. (2011). Children's episodic memory. *Wiley Interdisciplinary Reviews: Cognitive Science*, *2*(4), 365–373. <https://doi.org/10.1002/wcs.114>
- Ghisletta, P., & McArdle, J. J. (2001). Latent growth curve analyses of the development of height. *Structural Equation Modeling: A Multidisciplinary Journal*, *8*(4), 531–555. <https://doi.org/10.1207/S15328007SEM0804>
- Gogtay, N., Nugent, T. F., Herman, D. H., Ordonez, A., Greenstein, D., Hayashi, K. M., ... Thompson, P. M. (2006). Dynamic mapping of normal human hippocampal development. *Hippocampus*, *16*(8), 664–672. <http://doi.org/10.1002/hipo.20193><http://doi.org/10.1002/hipo.20193>
- Grimm, K. J., Ram, N., & Estabrook, R. (2016). *Growth modeling: Structural equation and multilevel modeling approaches*. New York, NY: Guilford Press.
- Hancock, G. R., Haring, J. R., & Lawrence, F. R. (2013). Using latent growth modeling to evaluate longitudinal change. In G. R. Hancock & R. O. Mueller, (Eds.), *Structural equation modeling. A second course* (2nd ed., pp. 309–341). Charlotte, NC: Information Age Publishing, Inc.
- Hancock, G. R., Kuo, W., & Lawrence, F. R. (2001). An Illustration of Second-Order Latent Growth Models. *Structural Equation Modeling: A Multidisciplinary Journal*, *8*(3), 470–489. https://doi.org/10.1207/S15328007SEM0803_7

- Hancock, G. R., & Mueller, R. O. (2011). The reliability paradox in assessing structural relations within covariance structure models. *Educational and Psychological Measurement, 71*(2), 306-324. <https://doi.org/10.1177/0013164410384856>
- Hancock, G. R., Stapleton, L. M., & Arnold-Berkovits, I. (2009). The tenuousness of invariance tests within multisample covariance and mean structure models. In T. Teo & M. S. Khine (Eds.), *Structural equation modeling: Concepts and applications in educational research* (pp. 137-174). Rotterdam, Netherlands: Sense Publishers.
- Harding, A. J., Halliday, G. M., & Kril, J. J. (1998). Variation in hippocampal neuron number with age and brain volume. *Cerebral Cortex, 8*(8), 710–718. <https://doi.org/10.1093/cercor/8.8.710>
- Hassevoort, K. M., Khan, N. A., Hillman, C. H., & Cohen, N. J. (2019). Differential development of relational memory and pattern separation. *Hippocampus*. <https://doi.org/10.1002/hipo.23146>
- Hayduk, L., Cummings, G., Boadu, K., Pazderka-Robinson, H., & Boulianne, S. (2007). Testing! testing! one, two, three—Testing the theory in structural equation models!. *Personality and Individual Differences, 42*(5), 841-850. <https://doi.org/10.1016/j.paid.2006.10.001>
- Head, D., Allison, S., Lucena, N., Hassenstab, J., & Morris, J. C. (2016). Latent structure of cognitive performance in the adult children study. *Journal of Clinical and Experimental Neuropsychology, 39*(7), 621–635. <https://doi.org/10.1080/13803395.2016.1252725>
- Herting, M. M., Johnson, C., Mills, K. L., Vijayakumar, N., Dennison, M., Liu, C., ... & Allen, N. B. (2018). Development of subcortical volumes across adolescence in males and females: A multisample study of longitudinal changes. *NeuroImage, 172*, 194-205. <https://doi.org/10.1016/j.neuroimage.2018.01.020>

- Hu, L. T., & Bentler, P. M. (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods*, 3(4), 424.
<https://doi.org/10.1037/1082-989X.3.4.424>
- Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling: A Multidisciplinary Journal*, 6(1), 1-55. <https://doi.org/10.1080/10705519909540118>
- Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *Journal of Comparative Neurology*, 387(2), 167-178.
[https://doi.org/10.1002/\(SICI\)1096-9861\(19971020\)387:2<167::AID-CNE1>3.0.CO;2-Z](https://doi.org/10.1002/(SICI)1096-9861(19971020)387:2<167::AID-CNE1>3.0.CO;2-Z)
- Jabès, A., Banta Lavenex, P., Amaral, D. G., & Lavenex, P. (2011). Postnatal development of the hippocampal formation: A stereological study in macaque monkeys. *The Journal of Comparative Neurology*, 519(6), 1051–1070. <https://doi.org/10.1002/cne.22549>
- Josselyn, S. A., & Frankland, P. W. (2012). Infantile amnesia: A neurogenic hypothesis. *Learning & Memory*, 19(9), 423–433. <https://doi.org/10.1101/lm.021311.110>
- Kang, Y., McNeish, D. M., & Hancock, G. R. (2016). The role of measurement quality on practical guidelines for assessing measurement and structural invariance. *Educational and Psychological Measurement*, 76(4), 533-561.
<https://doi.org/10.1177/0013164415603764>
- Keresztes, A., Bender, A. R., Bodammer, N. C., Lindenberger, U., Shing, Y. L., & Werkle-Bergner, M. (2017). Hippocampal maturity promotes memory distinctiveness in childhood and adolescence. *Proceedings of the National Academy of Sciences*, 114(34), 9212-9217. <https://doi.org/10.1073/pnas.1710654114>

- Keresztes, A., Ngo, C. T., Lindenberger, U., Werkle-Bergner, M., & Newcombe, N. S. (2018). Hippocampal maturation drives memory from generalization to specificity. *Trends in Cognitive Sciences*, 22(8), 676-686. <https://doi.org/10.1016/j.tics.2018.05.004>
- Ketz, N., Morkonda, S. G., & O'Reilly, R. C. (2013). Theta coordinated error-driven learning in the hippocampus. *PLoS Computational Biology*, 9(6). <https://doi.org/10.1371/journal.pcbi.1003067>
- Kievit, R. A., Brandmaier, A. M., Ziegler, G., van Harmelen, A. L., de Mooij, S. M. M., Moutoussis, M., ... Dolan, R. J. (2018). Developmental cognitive neuroscience using latent change score models: A tutorial and applications. *Developmental Cognitive Neuroscience*, 33,1–19. <https://doi.org/10.1016/j.dcn.2017.11.007>
- Kline, R. B. (2011). *Principles and practice of structural equation modeling* (3rd ed.). New York, NY: Guilford Press.
- Krogsrud, S. K., Tamnes, C. K., Fjell, A. M., Amlien, I., Grydeland, H., Sulutvedt, U., ... Walhovd, K. B. (2014). Development of hippocampal subfield volumes from 4 to 22 years. *Human Brain Mapping*, 35(11), 5646–5657. <https://doi.org/10.1002/hbm.22576>
- La Joie, R., Fouquet, M., Mézenge, F., Landeau, B., Villain, N., Mevel, K., ... & Chételat, G. (2010). Differential effect of age on hippocampal subfields assessed using a new high-resolution 3T MR sequence. *Neuroimage*, 53(2), 506–514. <https://doi.org/10.1016/j.neuroimage.2010.06.024>
- Lavenex, P., & Banta Lavenex, P. (2013). Building hippocampal circuits to learn and remember: Insights into the development of human memory. *Behavioural Brain Research*, 254, 8–21. <https://doi.org/10.1016/j.bbr.2013.02.007>

- Lavenex, P., Banta Lavenex, P., & Amaral, D. G. (2004). Nonphosphorylated high-molecular-weight neurofilament expression suggests early maturation of the monkey subiculum. *Hippocampus*, *14*(7), 797–801. <https://doi.org/10.1002/hipo.20028>
- Ledergerber, D., & Moser, E. I. (2017). Memory retrieval: taking the route via subiculum. *Current Biology*, *27*(22), R1225-R1227. <https://doi.org/10.1016/j.cub.2017.09.042>
- Lee, J. K., Ekstrom, A. D., & Ghetti, S. (2014). Volume of hippocampal subfields and episodic memory in childhood and adolescence. *Neuroimage*, *94*, 162-171. <https://doi.org/10.1016/j.neuroimage.2014.03.019>
- Lee, J. K., Fandakova, Y., Johnson, E. G., Cohen, N. J., Bunge, S. A., & Ghetti, S. (2020). Changes in anterior and posterior hippocampus differentially predict item-space, item-time, and item-item memory improvement. *Developmental Cognitive Neuroscience*, *41*, 100741. <https://doi.org/10.1016/j.dcn.2019.100741>
- Lee, J. K., Wendelken, C., Bunge, S. A., & Ghetti, S. (2016). A time and place for everything: Developmental differences in the building blocks of episodic memory. *Child Development*, *87*(1), 194-210. <https://doi.org/10.1111/cdev.12447>
- Lenroot, R. K., & Giedd, J. N. (2006). Brain development in children and adolescents: insights from anatomical magnetic resonance imaging. *Neuroscience & Biobehavioral Reviews*, *30*(6), 718-729. <https://doi.org/10.1016/j.neubiorev.2006.06.001>
- Liang, J. C., & Preston, A. R. (2015). Medial temporal lobe subregional function in episodic memory: Insights from high-resolution of fMRI. In D. R. Addis, M. Barense, & A. Duarte (Eds.), *The wiley handbook on the cognitive neuroscience of memory* (pp. 108–130). New York: Wiley-Blackwell. <https://doi.org/10.1002/9781118332634.ch6>

- Little, T. D. (2013). *Longitudinal structural equation modeling*. New York, NY: Guilford Press.
- Lorsbach, T. C., & Reimer, J. F. (2005). Feature binding in children and young adults. *The Journal of Genetic Psychology: Research and Theory on Human Development, 166*(3), 313–327. <http://doi.org/10.3200/GNTP.166.3.313-328>
- MacAulay, R. K., Calamia, M. R., Cohen, A. S., Daigle, K., Foil, H., Brouillette, R., ... & Keller, J. N. (2017). Understanding heterogeneity in older adults: Latent growth curve modeling of cognitive functioning. *Journal of Clinical and Experimental Neuropsychology, 40*(3), 292–302. <https://doi.org/10.1080/13803395.2017.1342772>
- MacCallum, R. C., Browne, M. W., & Sugawara, H. M. (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods, 1*(2), 130. <https://doi.org/10.1037/1082-989X.1.2.130>
- Mathews, M. E., & Fozard, J. L. (1970). Age differences in judgments of recency for short sequences of pictures. *Developmental Psychology, 3*(2), 208–217. <https://doi.org/10.1037/h0029582>
- McArdle, J. J. (2009). Latent variable modeling of differences and changes with longitudinal data. *Annual Review of Psychology, 60*(1), 577–605. <https://doi.org/10.1146/annurev.psych.60.110707.163612>
- McNeish, D., An, J., & Hancock, G. R. (2018). The thorny relation between measurement quality and fit index cutoffs in latent variable models. *Journal of Personality Assessment, 100*(1), 43-52. <https://doi.org/10.1080/00223891.2017.1281286>
- Meade, A. W., Johnson, E. C., & Braddy, P. W. (2008). Power and sensitivity of alternative fit indices in tests of measurement invariance. *Journal of Applied Psychology, 93*(3), 568. <https://doi.org/10.1037/0021-9010.93.3.568>

- Meredith, W., & Tisak, J. (1990). Latent curve analysis. *Psychometrika*, 55(1), 107–122.
<https://doi.org/10.1007/BF02294746><https://doi.org/10.1007/BF02294746>
- Millsap, R. E. (2007). Structural equation modeling made difficult. *Personality and Individual Differences*, 42(5), 875-881. <https://doi.org/10.1016/j.paid.2006.09.021>
- Millsap, R. E. (2011). *Statistical approaches to measurement invariance*. New York, NY: Routledge. <https://doi.org/10.4324/9780203821961>
- Moscovitch, M., Cabeza, R., Winocur, G., & Nadel, L. (2016). Episodic memory and beyond: The hippocampus and neocortex in transformation. *Annual Review of Psychology*, 67(1), 105–134. <https://doi.org/10.1146/annurev-psych-113011-143733>
- Muthén, L. K., & Muthén, B. O. (1998–2017). Mplus user’s guide (8th ed.). Los Angeles, CA: Muthén & Muthén.
- Nadel, L., & Moscovitch, M. (1997). Memory reconsolidation, retrograde amnesia and the hippocampal complex. *Cognitive Neuroscience*, 217–227. [https://doi.org/10.1016/S0959-4388\(97\)80010-4](https://doi.org/10.1016/S0959-4388(97)80010-4)
- Nee, D. E., & Jonides, J. (2008). Dissociable interference-control processes in perception and memory. *Psychological Science*, 19(5), 490–500. <https://doi.org/10.1111/j.1467-9280.2008.02114.x>
- Ngo, C. T., Lin, Y., Newcombe, N. S., & Olson, I. R. (2019). Building up and wearing down episodic memory: Mnemonic discrimination and relational binding. *Journal of Experimental Psychology: General*. <https://doi.org/10.31234/osf.io/dhnjy>
- Ngo, C. T., Newcombe, N. S., & Olson, I. R. (2017). The ontogeny of relational memory and pattern separation. *Developmental Science*, 21(2), 1–11. <https://doi.org/10.1111/desc.12556>

- Ofen, N., Kao, Y.-C., Sokol-Hessner, P., Kim, H., Whitfield-Gabrieli, S., & Gabrieli, J. D. E. (2007). Development of the declarative memory system in the human brain. *Nature Neuroscience*, *10*(9), 1198–1205. <https://doi.org/10.1038/nn1950>
- O'Mara, S. (2005). The subiculum: what it does, what it might do, and what neuroanatomy has yet to tell us. *Journal of Anatomy*, *207*(3), 271-282. <https://doi.org/10.1016/j.cub.2017.09.042>
- Park, D. C., Smith, A. D., Lautenschlager, G., Earles, J. L., Frieske, D., Zwahr, M., & Gaines, C. L. (1996). Mediators of long-term memory performance across the life span. *Psychology and Aging*, *11*(4), 621–637. <https://doi.org/10.1037/0882-7974.11.4.621>
- Perlaki, G., Orsi, G., Plozer, E., Altbacker, A., Darnai, G., Nagy, S. A., ... & Bogner, P. (2014). Are there any gender differences in the hippocampus volume after head-size correction? A volumetric and voxel-based morphometric study. *Neuroscience Letters*, *570*, 119-123. <https://doi.org/10.1016/j.neulet.2014.04.013>
- Pintzka, C. W. S., Hansen, T. I., Evensmoen, H. R., & Håberg, A. K. (2015). Marked effects of intracranial volume correction methods on sex differences in neuroanatomical structures: a HUNT MRI study. *Frontiers in Neuroscience*, *9*, 238. <https://doi.org/10.3389/fnins.2015.00238>
- Poppenk, J., Evensmoen, H. R., Moscovitch, M., & Nadel, L. (2013). Long-axis specialization of the human hippocampus. *Trends in Cognitive Sciences*, *17*(5), 230–240. <https://doi.org/10.1016/j.tics.2013.03.005>
- Preacher, K. J. (2010). Latent growth curve models. In G. R. Hancock, L. M. Stapleton, & R. O. Mueller (Eds.), *The reviewer's guide to quantitative methods in the social sciences* (pp. 185-198). London: Routledge.

- Putnick, D. L., & Bornstein, M. H. (2016). Measurement invariance conventions and reporting: The state of the art and future directions for psychological research. *Developmental Review, 41*, 71–90. <https://doi.org/10.1016/j.dr.2016.06.004>
- Ranganath, C., Yonelinas, A. P., Cohen, M. X., Dy, C. J., Tom, S. M., & D’Esposito, M. (2004). Dissociable correlates of recollection and familiarity within the medial temporal lobes. *Neuropsychologia, 42*, 2–13. <https://doi.org/10.1016/j.neuropsychologia.2003.07.006>
- Raz, N., Lindenberger, U., Rodrigue, K. M., Kennedy, K. M., Head, D., Williamson, A., ... & Acker, J. D. (2005). Regional brain changes in aging healthy adults: general trends, individual differences and modifiers. *Cerebral Cortex, 15*(11), 1676-1689. <https://doi.org/10.1093/cercor/bhi044>
- Rice, K., Viscomi, B., Riggins, T., & Redcay, E. (2014). Amygdala volume linked to individual differences in mental state inference in early childhood and adulthood. *Developmental Cognitive Neuroscience, 8*, 153-163. <https://doi.org/10.1016/j.dcn.2013.09.003>
- Riggins, T. (2014). Longitudinal investigation of source memory reveals different developmental trajectories for item memory and binding. *Developmental Psychology, 50*(2), 449–459. <https://doi.org/10.1037/a0033622>
- Riggins, T., Blankenship, S. L., Mulligan, E., Rice, K., & Redcay, E. (2015). Developmental differences in relations between episodic memory and hippocampal subregion volume during early childhood. *Child Development, 86*(6), 1710–1718. <https://doi.org/10.1111/cdev.12445>
- Riggins, T., Geng, F., Blankenship, S. L., & Redcay, E. (2016). Hippocampal functional connectivity and episodic memory in early childhood. *Developmental Cognitive Neuroscience, 19*, 58-69. <https://doi.org/10.1016/j.dcn.2016.02.002>

- Riggins, T., Geng, F., Botdorf, M., Canada, K., Cox, L., & Hancock, G. R. (2018). Protracted hippocampal development is associated with age-related improvements in memory during early childhood. *NeuroImage*, *174*, 127–137.
<https://doi.org/10.1016/j.neuroimage.2018.03.009>
- Riggins, T., Miller, N. C., Bauer, P. J., Georgieff, M. K., & Nelson, C. A. (2009). Electrophysiological indices of memory for temporal order in early childhood: Implications for the development of recollection. *Developmental Science*, *12*(2), 209–219.
<https://doi.org/10.1111/j.1467-7687.2008.00757.x>
- Rolls, E. T. (2013). The mechanisms for pattern completion and pattern separation in the hippocampus. *Frontiers in Systems Neuroscience*, *7*, 1–21.
<https://doi.org/10.3389/fnsys.2013.00074>
- Rolls, E. T. (2016). Pattern separation, completion, and categorisation in the hippocampus and neocortex. *Neurobiology of Learning and Memory*, *129*, 4–28.
<https://doi.org/10.1016/j.nlm.2015.07.008>
- Sankar, T., Park, M. T. M., Jawa, T., Patel, R., Bhagwat, N., Voineskos, A. N., ... Chakravarty, M. M. (2017). Your algorithm might think the hippocampus grows in Alzheimer’s disease: Caveats of longitudinal automated hippocampal volumetry. *Human Brain Mapping*, *38*(6), 2875–2896. <https://doi.org/10.1002/hbm.23559>
- Sastre III, M., Wendelken, C., Lee, J. K., Bunge, S. A., & Ghetti, S. (2016). Age- and performance-related differences in hippocampal contributions to episodic retrieval. *Developmental Cognitive Neuroscience*, *19*, 42–50.
<https://doi.org/10.1016/j.dcn.2016.01.003>

- Satorra, A. (2000). Scaled and adjusted restricted tests in multi-sample analysis of moment structures. In *Innovations in multivariate statistical analysis* (pp. 233-247). Springer, Boston, MA. https://doi.org/10.1007/978-1-4615-4603-0_17
- Satorra, A., & Bentler, P. M. (2001). A scaled difference chi-square test statistic for moment structure analysis. *Psychometrika*, *66*(4), 507-514. <https://doi.org/10.1007/BF02296192>
- Schacter, D. L., & Tulving, E. (1994). What are the memory systems of 1994? In *Memory systems 1994* (pp. 1–38). Cambridge, MA: The MIT Press.
- Schlichting, M. L., Guarino, K. F., Schapiro, A. C., Turk-Browne, N. B., & Preston, A. R. (2017). Hippocampal structure predicts statistical learning and associative inference abilities during development. *Journal of Cognitive Neuroscience*, *29*(1), 37-51. https://doi.org/10.1162/jocn_a_01028
- Schlichting, M. L., Mack, M. L., Guarino, K. F., & Preston, A. R. (2019). Performance of semi-automated hippocampal subfield segmentation methods across ages in a pediatric sample. *NeuroImage*, *191*, 49–67. <https://doi.org/10.1016/j.neuroimage.2019.01.051>
- Schneider, W., & Ornstein, P. A. (2019). Determinants of memory development in childhood and adolescence. *International Journal of Psychology*, *54*(3), 307-315. <https://doi.org/10.1002/ijop.12503>
- Scoville, W. B., & Milner, B. (1957). Loss of recent memory after bilateral hippocampal lesions. *Journal of Neurology, Neurosurgery & Psychiatry*, *20*, 11–21. <https://doi.org/10.1136/jnnp.20.1.11>
- Seress, L. (2001). Morphological changes of the human hippocampal formation from midgestation to early childhood. In M. L. Collins & C. A. Nelson (Eds.), *Handbook of developmental cognitive neuroscience* (pp. 45–58). Cambridge, MA: The MIT Press.

- Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: uses in assessing rater reliability. *Psychological Bulletin*, *86*(2), 420–428. <https://doi.org/10.1037/0033-2909.86.2.420>
- Snodgrass, J. G., & Corwin, J. (1988). Pragmatics of measuring recognition memory: Applications to dementia and amnesia. *Journal of Experimental Psychology: General*, *117*(1), 34–50. <http://doi.org/10.1037/0096-3445.117.1.34>
- Spaan, P. E. J. (2015). Episodic and semantic memory functioning in very old age: Explanations from executive functioning and processing speed theories. *Cogent Psychology*, *2*(1), 1–24. <https://doi.org/10.1080/23311908.2015.1109782>
- Spearman, C. (1904). The proof and measurement of association between two things. *American Journal of Psychology*, *15*(1), 72–101. <https://doi.org/10.2307/1412159>
- Squire, L. R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review*, *99*(2), 195–231. <https://doi.org/10.1037/0033-295X.99.3.582>
- Squire, L. R., & Zola-Morgan, S. (1991). The medial temporal lobe memory system. *Science*, *253*(5026), 1380–1386. <https://doi.org/10.1126/science.1896849>
- Strange, B. A., Witter, M. P., Lein, E. S., & Moser, E. I. (2014). Functional organization of the hippocampal longitudinal axis. *Nature Reviews Neuroscience*, *15*(10), 655–669. <https://doi.org/10.1038/nrn3785>
- Suthana, N., Ekstrom, A., Moshirvaziri, S., Knowlton, B., & Bookheimer, S. (2011). Dissociations within human hippocampal subregions during encoding and retrieval of spatial information. *Hippocampus*, *21*(7), 694–701. <https://doi.org/10.1002/hipo.20833>

- Talmi, D., Grady, C. L., Goshen-Gottstein, Y., & Moscovitch, M. (2005). *Neuroimaging the serial position curve: a test of single-store versus dual-store models*. *Psychological Science*, *16*(9), 716–723. <https://doi.org/10.1111/j.1467-9280.2005.01601.x>
- Tamnes, C., Bos, M., van de Kamp, F., Peters, S., & Crone, E. (2018). Longitudinal development of hippocampal subregions from childhood to adulthood. *Developmental Cognitive Neuroscience*, *30*, 212–222. <https://doi.org/10.1016/j.dcn.2018.03.009>
- Tamnes, C. K., Walhovd, K. B., Engvig, A., Grydeland, H., Krogsrud, S. K., Østby, Y., ... & Fjell, A. M. (2014). Regional hippocampal volumes and development predict learning and memory. *Developmental Neuroscience*, *36*(3-4), 161-174. <https://doi.org/10.1159/00036244>
- Thompson, M. S., & Green, S. B. (2013). Evaluating between-group differences in latent variable means. In G. R. Hancock & R. O. Mueller (Eds.), *Structural equation modeling: A second course* (2nd ed.) (pp. 309- 341). Charlotte, NC: Information Age Publishing, Inc.
- Tisak, J., & Tisak, M. S. (1996). Longitudinal models of reliability and validity: A latent curve approach. *Applied Psychological Measurement*, *20*(3), 275–288. <https://doi.org/10.1177/014662169602000307>
- Tulving, E. (1972). Episodic and semantic memory. In E. Tulving & W. Donaldson (Eds.), *Organization of memory* (pp. 381–403). New York, NY: Academic Press.
- Tulving, E. (1993). What is episodic memory? *Current Directions in Psychological Science*, *2*(3), 67–70. <https://doi.org/10.1111/1467-8721.ep10770899>
- Tulving, E. (2002). Episodic memory: from mind to brain. *Annual Review of Psychology*, *53*(1), 1–25. <https://doi.org/10.1146/annurev.psych.53.100901.135114>

- Utsunomiya, H., Takano, K., Okazaki, M., & Mitsudome, A. (1999). Development of the temporal lobe in infants and children: analysis by MR-based volumetry. *American Journal of Neuroradiology*, 20(4), 717-723.
- Vargha-Khadem, F., Gadian, D. G., Watkins, K. E., Connelly, A., Van Paesschen, W., & Mishkin, M. (1997). Differential effects of early hippocampal pathology on episodic and semantic memory. *Science*, 277(5324), 376–380. <https://doi.org/10.1126/science.277.5324.376>
- Willet, J. B., & Sayer, A. G. (1996). Cross-domain analysis of change overtime: Combining growth modeling and covariance structure analysis. In G. A. Marcoulides & R. E. Schumacker (Eds.). *Advanced structural equation modeling. Issues and techniques* (pp. 125–157). Mahwah, NJ: Lawrence Erlbaum Associates, Publishers.
- Winterburn, J. L., Pruessner, J. C., Chavez, S., Schira, M. M., Lobaugh, N. J., Voineskos, A. N., & Chakravarty, M. M. (2013). A novel in vivo atlas of human hippocampal subfields using high-resolution 3 T magnetic resonance imaging. *Neuroimage*. 74, 254–265. <https://doi.org/10.1016/j.neuroimage.2013.02.003>
- Wisse, L. E., Daugherty, A. M., Olsen, R. K., Berron, D., Carr, V. A., Stark, C. E., ... & Bernstein, J. D. (2017). A harmonized segmentation protocol for hippocampal and parahippocampal subregions: Why do we need one and what are the key goals?. *Hippocampus*, 27(1), 3-11. <https://doi.org/10.1002/hipo.22671>
- Wothke, W. (1993). Nonpositive definite matrices in structural modeling. In K. A. Bollen and J. S. Long (Eds.), *Testing structural equation models* (pp. 256–293). Newbury Park, CA: Sage Publications.
- Yassa, M. A., & Stark, C. E. (2011). Pattern separation in the hippocampus. *Trends in Neurosciences*, 34(10), 515–525. <https://doi.org/10.1016/j.tins.2011.06.006>

- Yushkevich, P. A., Amaral, R. S. C., Augustinack, J. C., Bender, A. R., Bernstein, J. D., Boccardi, M., ... Zeineh, M. M. (2015b). Quantitative comparison of 21 protocols for labeling hippocampal subfields and parahippocampal subregions in in vivo MRI: Towards a harmonized segmentation protocol. *NeuroImage*, *111*, 526–541.
<https://doi.org/10.1016/j.neuroimage.2015.01.004>
- Yushkevich, P. A., Pluta, J. B., Wang, H., Xie, L., Ding, S. L., Gertje, E. C., ... Wolk, D. A. (2015a). Automated volumetry and regional thickness analysis of hippocampal subfields and medial temporal cortical structures in mild cognitive impairment. *Human Brain Mapping*, *36*(1), 258–287. <https://doi.org/10.1002/hbm.22627>
- Zijdenbos, A. P., Dawant, B. M., Margolin, R. A., & Palmer, A. C. (1994). Morphometric analysis of white matter lesions in MR images: method and validation. *IEEE Transactions on Medical Imaging*, *13*(4), 716–724. <https://doi.org/10.1109/42.363096>
- Zuber, S., Kliegel, M., & Ihle, A. (2016). An individual difference perspective on focal versus nonfocal prospective memory. *Memory and Cognition*, *44*(8), 1192–1203.
<https://doi.org/10.3758/s13421-016-0628-5>