

ABSTRACT

Title of Dissertation: RELATIONS BETWEEN LATENT EPISODIC MEMORY, NAP HABITUALITY, AND THE CORTEX DURING CHILDHOOD.

Tamara Lynn Allard, Doctor of Philosophy,
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Thesis Directed By: Professor Tracy Riggins,
Department of Psychology

During childhood, episodic memory demonstrates marked improvements that are supported by the protracted development of the hippocampus and a larger network of cortical regions. To date, most research has focused on associations with the hippocampus in this age group. Few studies have explored the contribution of cortical regions and no studies have explored this longitudinally. Thus, the first aim of this dissertation was to examine the longitudinal co-development of cortical thickness and surface area in memory-related cortical regions with a latent episodic memory variable in 4- to 8-year-old children ($N = 177$). Findings, uncorrected for multiple comparisons, demonstrated that a thinner cortex in multiple episodic memory network regions (i.e., inferior frontal gyrus, inferior parietal sulcus, lingual gyrus, middle temporal gyrus, precuneus, lateral occipital cortex, superior frontal gyrus, superior parietal lobule, superior temporal gyrus, and temporal pole) at age 4 predicted more rapid improvements in memory performance from age 4 to 6 years. Similarly, greater surface area in the precuneus and less surface area in the medial orbitofrontal gyrus at age 4 also predicted more rapid improvements in memory performance from

age 4 to 6 years. Additionally, results revealed that several regions demonstrate parallel co-development with latent episodic memory performance from age 4 to 8 years. Specifically, greater changes in cortical thickness and surface area of the entorhinal cortex were associated with greater changes in memory from age 4 to 6 years. Furthermore, cortical thickness of entorhinal cortex and surface area of anterior cingulate cortex, entorhinal cortex, inferior parietal sulcus, lingual gyrus, and superior temporal gyrus showed co-development with latent episodic memory from age 6 to 8 years. Together, these findings suggest that cortical thickness and surface area of the episodic memory network support improvements in memory performance during childhood. However, these findings did not survive correction for multiple comparisons. Although age-related differences were one focus of this investigation, individual differences were another. Specifically, during childhood children transition away from afternoon napping. This transition has previously been associated with differences in memory consolidation abilities and hippocampal maturation. These associations suggest that habitual nappers require more regular sleep to consolidate memories due to an immature episodic memory network. However, limited work has examined these associations outside the hippocampus. Therefore, the second aim of this dissertation was to examine whether regions that support longitudinal memory development differ as a function of nap habituality ($N = 44$). Findings revealed significant differences in cortical thickness of right inferior frontal gyrus and surface area of lateral occipital cortex, such that non-nappers demonstrated a thinner cortex and greater surface area in these regions compared to nappers, though these findings did not survive correction for multiple comparisons. Thus, although there is some evidence that memory-related cortical regions may differ based on nap habituality, additional work is needed to support this claim. Together this dissertation provides new data on the co-

development of memory with brain structure in the episodic memory network and identifies individual differences that may be associated with these brain structures.

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CORTEX DURING CHILDHOOD.

by

Tamara Lynn Allard

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Advisory Committee:

Professor Tracy Riggins, Ph.D., Chair
Associate Professor Elizabeth Redcay, Ph.D.
Assistant Professor Rachel Romeo, Ph.D.
Professor Rebecca Spencer, Ph.D.
Associate Professor Donald Bolger, Ph.D., Dean's Representative

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Dedication

I dedicate this to my family whose steadfast support of my educational aspirations has made this dissertation a reality. To my husband, Ethan, thank you for your unwavering encouragement, patience, and belief in my abilities. Your support has sustained me through this degree. You are my rock and I love you. To my parents Bernard Allard and Ava Grajeda-Allard, because of you I am a critical thinker and a hard worker, qualities required for a Ph.D. Without you, none of this would have been possible. I know that you walked so I could run. Thank you.

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List of Abbreviations

ACC = Anterior Cingulate Cortex

AG = Angular Gyrus

CA1 = Cornu Ammonis 1

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

dIPFC = Dorsolateral Prefrontal Cortex

DPC = Dorsal Parietal Cortex

DMN = Default Mode Network

ERC = Entorhinal Cortex

ICV = Intracranial Volume

IFG = Inferior Frontal Gyrus

IPS = Inferior Parietal Sulcus

LOC = Lateral Occipital Cortex

MTL = Medial Temporal Lob

PCC = Posterior Cingulate Cortex

PFC = Prefrontal Cortex

PHG = Parahippocampal Gyrus

pIPS = Posterior Intraparietal Sulcus

PRC = Perirhinal Cortex

SPL = Superior Parietal Lobule

ROI = Region of Interest

vIPFC = Ventrolateral Prefrontal Cortex

VPC = Ventral Parietal Cortex

Chapter 1: Introduction

Fifty years ago, Tulving (1972) suggested that the ability to remember past events is a distinct form of declarative memory. Specifically, he noted that episodic memory is different from semantic memory due to its auto-noetic quality that requires a person to perform mental time travel (Tulving, 1984, 2002). Today, most groups agree that episodic memories are specific and detailed memories for past events, that include information about event content (i.e., what), location (i.e., where), and timing (i.e., when).

Across childhood and into early adulthood, episodic memory demonstrates marked improvements (Canada et al., 2021, 2022; Lee et al., 2016; Picard et al., 2012; Riggins, 2014; Yim et al., 2013). These improvements are supported by the protracted development of the hippocampus (Botdorf et al., 2022; Canada et al., 2021; Lee et al., 2020; Riggins et al., 2018), an area strongly implicated in memory (Scoville & Milner, 1957). However, work in adults has demonstrated that episodic memory is also supported by a broad network of cortical regions including the MTL, PFC, and parietal cortex (e.g., Brewer et al., 1998; Buckner et al., 1999; Davachi et al., 2003; Lavenex & Amaral, 2000; Nyberg et al., 2000; Stern et al., 1996; Stevens et al., 2008; Wagner et al., 1998). Like the hippocampus, these regions undergo protracted structural development during childhood (Frangou et al., 2022; Gogtay et al., 2004) suggesting they may also play a role in the development of episodic memory abilities.

Although limited, some work in developmental populations has supported this hypothesis by demonstrating cross-sectional relations between memory performance and the cortex during childhood (see Ghetti & Bunge, 2012; Østby et al., 2012; Schommartz et al., 2023). These studies show that, for most regions, cortical thickness is negatively associated with episodic memory

performance across early to late childhood (e.g., ages 4 to 12 years). However, no study has examined these associations longitudinally. This is problematic because cross-sectional studies cannot provide information about change over time. In other words, studies that only assess one time point cannot make claims about whether changes in the brain support changes in memory. Furthermore, past work has demonstrated that cross-sectional and longitudinal studies sometimes result in different outcomes, with cross-sectional studies often underestimating relations between the brain and memory during development (Keresztes et al., 2022). Thus, the first aim of this study is to examine longitudinal co-development of memory performance and structural measures of cortical ROIs that support memory.

During early childhood children transition from biphasic sleep to monophasic sleep (i.e., cessation of the afternoon nap; Staton et al., 2020). These developmental shifts in memory, nap habits, and brain development are likely related. For example, some have theorized that habitual nappers may require more regular sleep due to an inefficiency in sleep-dependent memory consolidation caused by an immature episodic memory network (Lokhandwala & Spencer, 2022; Mason et al., 2021; Spencer & Riggins, 2022). Evidence in early childhood supports this hypothesis. Namely, habitual nappers do worse on an episodic memory task following a wake period compared to their non-napping counterparts and they demonstrate less hippocampal maturity (Kurdziel et al., 2013; Riggins & Spencer, 2020). Yet, no work has examined whether these findings generalize to other memory-related brain regions. Thus, the second aim of this dissertation is to examine associations between nap habituality and cortical regions that support episodic memory.

Exploring longitudinal brain development and how it relates to changes in memory and sleep requires knowledge of multiple literatures. Below I review literature examining the

developmental trajectory of episodic memory performance, structural brain maturation, and sleep habits across childhood (e.g., age 3 to 12 years). Investigations of infancy, toddlerhood, and adolescence are beyond the scope of this dissertation (see instead Galván, 2020; Johnson et al., 2020; Kopasz et al., 2010; Mason et al., 2021). Next, I will examine associations between memory and structural measures of brain maturation, including cortical thickness and surface area. These measures are thought to reflect synaptogenesis, gyrification, and myelination in cortical grey matter (Cafiero et al., 2019; Huttenlocher, 1979; Rakic, 2009). Therefore, they are proxies for neural development that have been regionally associated with memory performance during early to middle childhood (e.g., Schommartz et al., 2023). Notably, this review will not directly focus on measures of brain function because brain structure and function develop on distinct time scales for most brain regions (Gilmore et al., 2015). Finally, I will review relations between nap habitually, memory, and the brain. There is a burgeoning body of work examining the association between memory development, the brain, and sleep physiology (e.g., sleep architecture and microstructure). However, these measures are also beyond the scope of this dissertation and will not be covered (see instead Lokhandwala & Spencer, 2022). I will conclude by describing the study design and aims of this dissertation.

Memory Development

Episodic memory, unlike other forms of declarative memory, is thought to be a recent evolutionary development. As a result, it is comparatively late developing, early deteriorating, and more sensitive to neural deficits (Tulving, 2002). Longitudinal research from the past decade supports this theory, showing protracted development from early to middle childhood (Allard et al., 2023; Canada, Pathman, et al., 2020; Canada et al., 2022; Lee et al., 2016; Riggins, 2014). For example, previous studies have demonstrated improvement in memory abilities for feature binding

(e.g., source memory; Allard et al., 2023; Cheke & Clayton, 2015; Drummey & Newcombe, 2002; Lee et al., 2016; Lorsbach & Reimer, 2005; Riggins, 2014; Sluzenski et al., 2006), fine-grained details (i.e., pattern separation; (Canada et al., 2019; Ngo et al., 2017), and temporal order of events across early to late childhood (Canada, Pathman, et al., 2020).

However, not all episodic memory abilities develop at the same rate. Some demonstrate linear trajectories (Canada, Pathman, et al., 2020), while others show non-linear trajectories ((Allard et al., 2023; Riggins, 2014). Further, different developmental trajectories can be observed within the same task depending on the variable assessed (Allard et al., 2023; Lee et al., 2016). For example, one study found that hit rates (i.e., the ability to accurately identify a previous connection as old) on a feature binding task are relatively early developing, while false alarm rates (i.e., the inability to recognize a novel connection as new) are relatively late developing (Allard et al., 2023). This dynamic could suggest that these memory tasks reflect development of additional cognitive abilities. For example, late development of false alarms in Allard (2023) could suggest changes in process that support encoding, like attention. This reveals a key limitation of episodic memory research, that no single task is process pure. However, latent measures of episodic memory may be a potential solution. Specifically, using structural equation modeling (SEM), researchers can create a construct-level measure of episodic memory (i.e., a latent variable) by capturing the shared variance from a variety of tasks assessing varying memory abilities. Theoretically, this shared variance between tasks represents an episodic memory signal, while the associated error represents noise from uncommon sources that may be attributed to other cognitive abilities. The resulting latent variable is likely a better measure of actual episodic memory performance.

In children, a handful of studies have successfully utilized latent techniques to assess associations between latent episodic memory and age (Canada et al., 2022; Cheke & Clayton,

2015). These studies have demonstrated that a battery of episodic memory tasks featuring surface differences can be used to create a latent measure of episodic memory in children as young as age 4 years (Canada et al., 2022; Cheke & Clayton, 2015) and that the episodic memory construct remains consistent across childhood (Canada et al., 2022). In other words, previous work has demonstrated that a latent episodic memory variable can be created that assesses the same memory construct at ages 4, 5, 6, 7, and 8 years. Further, results from a longitudinal study show that these latent measures of episodic memory do undergo linear age-related increases from early to middle childhood (Canada et al., 2022). However, associations between latent measures of memory and brain development during childhood remain unclear.

Brain Development

Research in adults demonstrates that several cortical regions support memory performance across the lifespan (e.g., Brewer et al., 1998; Buckner et al., 1999; Davachi et al., 2003; Lavenex & Amaral, 2000; Nyberg et al., 2000; Stern et al., 1996; Stevens et al., 2008; Wagner et al., 1998). These included several subregions in the medial temporal cortex (i.e., parahippocampal gyrus, entorhinal cortex, and perirhinal cortex), prefrontal (i.e., IFG, medial orbitofrontal, superior frontal, rostral middle frontal gyrus, and ACC), parietal (i.e., precuneus, posterior parietal, and inferior parietal sulcus), and occipital cortices (lateral occipital gyrus and lingual gyrus). Moreover, all of these regions undergo some form of change across childhood, with varying trajectories (Ducharme et al., 2015; Frangou et al., 2022; Gogtay et al., 2004; Lenroot et al., 2007; Mills et al., 2014; Raznahan et al., 2011; Tamnes et al., 2017; Wang et al., 2019; Wierenga et al., 2014).

Global gray matter demonstrates an overall increase from age 4 to the onset of puberty, then gradually thins into adulthood (Gogtay et al., 2004; Lenroot et al., 2007). For example, Lenroot and colleagues examined total gray matter volume in participants aged 3 to 27 years and found that volumes peaked at age 8.5 years (Lenroot et al., 2007). Gray matter volume can be accessed via two different measures, cortical surface area, and cortical thickness. These two measures are thought to reflect different aspects of brain development. Specifically, early increases in cortical thickness are thought to reflect synaptogenesis, whereas later cortical thinning is thought to reflect synaptic pruning (Huttenlocher, 1979). In contrast, increases in cortical surface area reflect folding and gyrification due to the division of neural stem cells and intra-cortical myelination, whereas later decreases in cortical surface area reflect decreases in folding and gyrification due to apoptosis (Cafiero et al., 2019; Rakic, 2009).

Given cortical thickness and surface area measure separate processes, they do not co-vary (Cafiero et al., 2019; Im et al., 2008) and they demonstrate unique developmental trajectories (Hutton et al., 2009; Li et al., 2013; Lyall et al., 2015; Wierenga et al., 2014). For example, in Cafiero and colleagues (2019), the authors demonstrated that age-related effects on cortical thickness and surface area were distinct in all but 2 cortical regions (i.e., lingual gyrus and calcarine sulcus) when examining a sample of 5- to 7-year-old children.

While trajectories for cortical thickness and cortical surface are distinct, both demonstrate non-linear change from birth to puberty. For example, cortical surface area increases across early to middle childhood, peaks between 8 and 10 years, and then decreases into adulthood (Ducharme et al., 2015; Mills et al., 2014; Raznahan et al., 2011; Wierenga et al., 2014). Similarly, early increases in cortical thickness eventually give way to later decreases in late childhood and adolescence (Lenroot et al., 2007; Raznahan et al., 2011). However, it is debated when cortical

thickness reaches its peaks (Tamnes et al., 2017; Walhovd et al., 2017). Specifically, some suggest that cortical thickness peaks in late childhood (e.g., 8 to 10 years; Lenroot et al., 2007; Raznahan et al., 2011; Shaw et al., 2007) while others suggest that thinning has already begun in early childhood (i.e., prior to age 5 years; Ducharme et al., 2016; Gogtay et al., 2004; Mills et al., 2014; Wierenga et al., 2014). In spite of this conflicting evidence, several studies have demonstrated that cortical thickness peaks before to surface area (Raznahan et al., 2011; Wierenga et al., 2014).

Importantly, development changes in cortical thickness and surface are not uniform, rather sub-regions demonstrate maturation at distinct rates (Gogtay et al., 2004; Raznahan et al., 2011; Tamnes et al., 2017; Wierenga et al., 2014). For example, cortical thinning begins in the dorsal parietal cortices and then spreads rostrally, caudally, and laterally over the frontal, occipital, and temporal cortices (Gogtay et al., 2004). Importantly, a meta-analysis including over 17,000 subjects aged 3 to 90 years demonstrated that all cortical regions appear to exhibit the greatest thickness during childhood (e.g., 3 to 10 years) except for the entorhinal cortex, the temporopolar cortex, and the anterior cingulate cortices which peak in middle adulthood (Frangou et al., 2022). In contrast, most regions show peak cortical surface area values between ages 8 and 13 years (Wierenga et al., 2014).

Sleep Development

During childhood, sleep undergoes significant changes. Specifically, the average amount of sleep children receive over a 24-hour period changes with age (Bathory & Tomopoulos, 2017; Galland et al., 2012; Iglowstein et al., 2003; Matricciani et al., 2012). According to a meta-analysis conducted by Galland et al., (2012), infants sleep an average of 12.7 hours per day. This decreases to 11.5 hours per day for preschool-aged children (aged 4 to 5 years; Bathory & Tomopoulos,

2017). Past age 5 years, overall 24 hour sleep time continues to decrease by approximately 5.9 minutes each year until age 12 years (Galland et al., 2012).

From infancy through early childhood, children also take progressively fewer naps and the duration of naps decreases (Iglowstein et al., 2003; Kurth et al., 2016; Ohayon et al., 2004; Weissbluth, 1995). For example, infants take upwards of 4 naps per day lasting an average of 3 hours per nap. In contrast, children over 5 years take only one nap per day lasting an average of 1 hour per nap (Galland et al., 2012; Iglowstein et al., 2003; Staton et al., 2020; Weissbluth, 1995). Eventually, these trends lead to nap cessation sometime in early childhood. Specifically, whereas 97% of children under 2 years nap at least one day per week, by age 3 years, 67% of children nap and by age 6 years, 6% of children nap (Staton et al., 2020).

Associations Between Memory and the Brain

Extensive research in adults has demonstrated that the hippocampus plays a critical role in supporting the formation and consolidation of episodic memories (Davachi et al., 2003; Eichenbaum, 2004; Lavenex & Banta Lavenex, 2013; Scoville & Milner, 1957). In children, cross-sectional studies have demonstrated that structural measures of the hippocampus are related to episodic memory during development (see Botdorf et al., 2022 for review). Moreover, a handful of longitudinal studies have demonstrated that volumetric changes in the hippocampus across childhood are associated with improvements in episodic memory performance (Canada et al., 2021; Lee et al., 2020). Together these findings suggest that hippocampal development plays a role in memory improvement across childhood. Research in adults also demonstrates that episodic memory abilities are supported by a broad network of neocortical regions outside the hippocampus. These include several subregions in medial temporal (i.e., parahippocampal gyrus,

entorhinal cortex, and perirhinal cortex), prefrontal (i.e., anterior cingulate cortex, orbitofrontal cortex, inferior frontal gyrus, superior frontal gyrus, and rostral middle frontal gyrus), parietal (i.e., precuneus, posterior parietal, and inferior parietal sulcus), and occipital cortices (lateral occipital and lingual gyrus). Importantly, there is both theoretical and empirical evidence to support the role of these regions in episodic memory formation and retrieval.

For example, in the MTL, the hippocampus receives and binds sensory information from two distinct neural pathways (i.e., what and where) that are thought to provide distinct event-related details (Eichenbaum et al., 2012; Wixted & Squire, 2011). The “what” pathway carries item-related information from visual regions through the perirhinal cortex (PRC) and the lateral entorhinal cortex (ERC) to the hippocampus (Eichenbaum et al., 2012). In contrast, the “where” pathway carries contextual information from the parietal cortex and retrosplenial cortex through the parahippocampal gyrus (PHG) and medial ERC to the hippocampus (Eichenbaum et al., 2012). Therefore, it follows that PHG, ERC, and PRC would be associated with memory performance.

In the PFC and PPC there are two hypothesized roles for subregions in memory performance based on functional connections to larger networks. Specifically, subregions that align with the Default Mode Network, including bilateral medial orbital frontal, medial frontal pole, and anterior cingulate cortex (ACC), angular gyrus (AG), posterior cingulate cortex (PCC), and precuneus support memory recall (Amlien et al., 2018; Miotto et al., 2020; Yu, Daugherty, et al., 2018). Whereas other subregions in the PFC and PPC that align with control and attention networks, including dorsolateral PFC (dlPFC) in the rostral middle frontal gyrus, ventrolateral PFC (vlPFC) in the inferior frontal gyrus (IFG), inferior parietal sulcus (IPS), and superior parietal lobule (SPL) support improved attention during encoding (Miotto et al., 2020; Tang et al., 2018; Wendelken et al., 2011).

Finally, in the occipital cortex, associations with memory are thought to reflect the reactivation of encoded information during recall due to the consolidation of memories from the hippocampus to the neocortex (e.g., Alvarez & Squire, 1994; Norman & O'Reilly, 2003). This theory is often called the Neurobiological Model of Memory. There is unique functional evidence in the occipital cortex that supports this theory in two subregions, the lingual gyrus and the lateral occipital cortex (LOC; Karanian & Slotnick, 2015; Rosen et al., 2018; Wing et al., 2015). The nature of Neurobiological Model of Memory theory means that findings are likely task specific and associated with the given role of each occipital region.

In children, associations between memory and the larger episodic memory network are still an emerging area, with most structural studies surfacing in the last decade (see Table 1). Of these, most agree that cortical thickness of aforementioned subregions in the MTL, PFC, parietal, and occipital cortices is negatively associated episodic memory performance during early to late childhood. In other words, a thinner cortex in these ROIs is related to better memory performance. Additionally, one study found that thinning of subregions in the PFC mediates associations between memory performance and age from late childhood into adulthood suggesting that cortical thinning accounts for age-related differences in memory performance (Klijn et al., 2016). In contrast, little to no research has examined these dynamics in cortical surface area. One notable exception found positive associations between memory performance and cortical surface area in anterior right middle frontal gyrus and inferior frontal gyrus; in other words, greater surface area was related to better memory performance (Lyle et al., 2017). However, the study focused primarily on adolescents. In combination, these findings suggest that integrity of these cortical regions is related to individual differences in episodic memory performance.

Table 1*Peer-reviewed Articles that Examine Associations Between Memory and Cortical ROIs.*

Study	N	Mean Age (years)	Age Range (years)	ROI(s)	Memory Assessment(s)
Amlien et al (2018)	270	19.4	6 to 80	Isthmus Cingulate Lingual Gyrus Posterior Cingulate Cortex Precuneus	Source Memory
Bauer et al. (2019)	66	7.34	5 to 8	ACC mPFC Hippocampus	Self-Derivation through Integration (Stem Facts–Open Ended) Self-Derivation through Integration (Stem Facts–Total) Self-Derivation through Integration (Integration Facts–Open Ended) Self-Derivation through Integration (Integration Facts–Total)
Chad-Friedman et al (2021)	63	4.23 & 7.19	4 to 7	SPL	CMS Source Memory
Fjell et al (2019)	650	25.8	4 – 88	Hippocampus IPFC	CVLT-C Rey-Osterrieth Complex Figure test
Klijin et al., 2016	90	11.0 (child sample)	10 to 12, 18, & 25 to 32	IFG	Verbal Memory Task
Guillery-Girard et al (2013)	30	11.31	6 to 23	Anterior Middle Temporal Gyrus dlPFC Hippocampus Superior Temporal Cortex vlPFC	What-Where-When Paradigm
Kereteszes et al., (2017)	70	9.8	6 to 14	ERC Hippocampus	Source Memory MST
Ostby et al (2012)	107	13.9	8 to 19	Hippocampus mOFC	Rey-Osterrieth Complex Figure test
Schommartz et al., (2023)	63	6.37	5 to 7	Hippocampus ERC IFG Inferior Parietal Sulcus LOC Lateral Orbitofrontal Cortex Medial Orbitofrontal Cortex Precuneus Rostral Middle Frontal Cortex SPL	Object-Location Association Task
Sowell et al (2001)	35	N/A	7 to 16	Frontal Cortex MTL	CVLT-C Rey-Osterrieth Complex Figure test
Squeglia et al (2013)	185	N/A	12 to 14	IPS SPL	CVLT-C
Yu et al (2018)	120	13.6	5 to 25	dlPFC (Rostral Middle Frontal Cortex)	CVLT-C

However, these previous studies have focused on one memory tasks, which, as described above also includes non-memory related attention and cognitive processes. No study to date has explored relations between brain structure and a latent variable of episodic memory. Thus, it remains unclear whether these findings are attributable to the development of other cognitive functions. Furthermore, there is a lack of research examining longitudinal co-development of episodic memory and these regions. Therefore, the first aim of this dissertation was to examine the longitudinal co-development of extrahippocampal regions that support memory and a latent variable of memory during early to middle childhood. Specifically, I hypothesize that longitudinal changes in cortical thickness/surface area will be associated with longitudinal changes in memory performance.

Associations Between Memory and Sleep

In early childhood, evidence demonstrates that an afternoon nap has a positive impact on memory abilities including emotion memory (Kurdziel et al., 2018), temporal order memory (Lokhandwala & Spencer, 2021); verbal memory (Esterline & Gómez, 2021; Giganti et al., 2014; Spanò et al., 2018; H. Wang et al., 2022; Williams & Horst, 2014) and memory generalization (Sandoval et al., 2017; H. Wang et al., 2022). Furthermore, several studies have found that the memory benefit generated by an afternoon nap persists after overnight sleep (Kurdziel et al., 2013; Kurdziel et al., 2018; Lokhandwala & Spencer, 2021; Sandoval et al., 2017; Spanò et al., 2018; Williams & Horst, 2014) with some effects persisting 1 week later (Williams & Horst, 2014).

Some work has even suggested that napping and overnight sleep interact to produce a positive memory effect (Kurdziel et al., 2018). For example, Kurdziel and colleagues (2018),

tested whether an afternoon nap or an overnight sleep bout had an impact on memory performance across a 24-hour period. In the study, there were two conditions: the first was a nap condition and the second was a wake condition lasting the same duration. They found that there were memory deficits due to a missed afternoon nap, but only after an overnight sleep bout. Thus, the effects of an afternoon nap and overnight sleep have an interactive effect on memory performance.

In addition, there is evidence that the positive effect of an afternoon nap are not specific to early childhood. Specifically, a meta-analysis of 54 studies examining relations between cognition and afternoon naps in samples ranging from early childhood to mid-adulthood found that an afternoon nap has a positive effect on declarative memory and that these effects are not moderated by age (Leong et al., 2022). In short, an afternoon nap is good for everyone, regardless of age. However, work examining nap habituality demonstrates that the effects of missing an afternoon nap are not similar across age.

Past work looking at nap habituality in early childhood demonstrates that habitual nappers who miss their afternoon nap display more extreme memory decay on episodic memory tasks than non-nappers (Esterline & Gómez, 2021; Kurdziel et al., 2013; Kurdziel et al., 2018). Additionally, these memory differences are still present 24 hours later (Kurdziel et al., 2013; Kurdziel et al., 2018). For example, Kurdziel and colleagues (2013) examined memory performance across nap and wake session with both habitual napper and non-nappers. They found that habitual nappers, but not non-nappers, took a significant hit to their memory following the wake session compared to the nap session. Further, memories that were lost during the afternoon wake session were not recovered during overnight sleep. These findings suggest that an afternoon nap is critical for memory consolidation in habitual nappers.

Associations Between the Brain and Sleep

During sleep, memories are thought to be consolidated from the hippocampus to the cortex making them less vulnerable to decay (Rasch & Born, 2013). Importantly, children who are habitual nappers may be required to consolidate memories more often due to immaturity of memory regions. For example, habitual nappers may have a less mature hippocampus requiring more regular memory consolidation due to insufficient storage capacity (Spencer & Riggins, 2022). Evidence for this hypothesis comes from studies that show there are significant differences in both hippocampal subregions (Allard et al., Under Review) and hippocampal subfields (Riggins & Spencer, 2020) based on nap habituality.

However, research examining associations between brain structure and nap habituality has not extended beyond the hippocampus. Thus, it is unclear if these associations are specific to the hippocampus or if they generalize to cortical regions associated with memory. In other words, no research to date has examined whether the maturation of cortical memory regions is also related to the nap transition. It is possible that, in addition to changes in the hippocampus, the cortex also matures in a way that “allows for” the nap transition. However, changes in the cortex could also be a downstream result of the nap transition. Regardless, it would be interesting to determine if regions other than the hippocampus show differences around the nap transition, then a mechanism can be further explored.

Studies have shown that sleep is related to the maturation of the cortex (Cheng et al., 2021; Hansen et al., 2022; Taki et al., 2012). For example, the synaptic homeostasis hypothesis suggests that “sleep is the price we pay for neural plasticity” (Tononi & Cirelli, 2014). During sleep, the brain is disconnected from external stimuli allowing for enhanced synaptic up and downscaling. This process leads to stronger relevant connections and fewer irrelevant

connections. In addition to supporting general brain development, it is also hypothesized that this process supports the long-term storage of memories (Tononi & Cirelli, 2014).

Evidence in humans shows that shorter 24-hour sleep duration has been linked with reduced total gray matter volume, cortical surface area, and cortical thickness across childhood and adolescence (Cheng et al., 2021; Hansen et al., 2022; Taki et al., 2012). For example, past work has shown that shorter 24-hour sleep duration is associated with decreased cortical volumes and surface area in the orbital frontal region, superior and middle frontal gyri, inferior and middle temporal gyri, precuneus, posterior cingulate cortex, ventromedial prefrontal cortex, and dlPFC of the rostral medial prefrontal cortex across the middle and into late childhood (Cheng et al., 2021). Additionally, Hansen and colleagues (2022) showed that shorter sleep duration during weekdays in adolescents was significantly associated with reduced cortical thickness in the left middle temporal gyrus, right postcentral, and right superior frontal cortices. All these regions have previously been implicated in memory performance. Thus, these findings suggest that sleep habits may be associated with cortical development of memory regions outside the hippocampus.

Furthermore, the animal literature suggests that the process of synaptic regulation (i.e., downscaling and upscaling) can be observed across a single nap-like session (Maret et al., 2011; Yang & Gan, 2012). Specifically, research in 3-week-old mice (developmental equivalent to approximately 2.5 years in humans) demonstrates that both synaptogenesis and synaptic pruning occur during a single 2-hour sleep bout. Further, effects on synaptic pruning were above and beyond the effects of a wake period lasting the same duration (Yang & Gan, 2012).

It is possible that habitual napping during childhood could be linked to the need for consistent synaptic pruning in cortical memory regions. Specifically, in addition to more regular memory consolidation, immature memory systems may also require more regular synaptic

regulations via pruning and synaptogenesis to enhance or re-organize memories. Thus, changes in nap habits may be preceded by the maturation of cortical regions that results in a less consistent need for synaptic regulation. As a result, the second aim of this dissertation was to explore whether there are differences in cortical thickness and surface area of memory-related brain regions based on nap habituality.

Summary

In summary, previous work demonstrates that episodic memory, brain regions that support memory and sleep habits all undergo developmental changes across childhood. Further, these changes are likely related. Specifically, with sleep, memories are consolidated from the hippocampus to the cortex. Thus, changes in sleep habits during early childhood are unsurprisingly related to both episodic memory performance and volumetric measures of the hippocampus. However, the literature demonstrates that the hippocampus is not the only brain region that supports episodic memory in early childhood. Limited work has demonstrated cross-sectional associations between memory and a larger episodic memory network in childhood; however, longitudinal investigations are still lacking. Given previous associations between sleep habits and cortical regions included in the episodic memory network, it is possible that these regions also differ as a function of nap habituality. However, previous work has not examined differences in these other brain regions based on nap habituality.

Study Overview

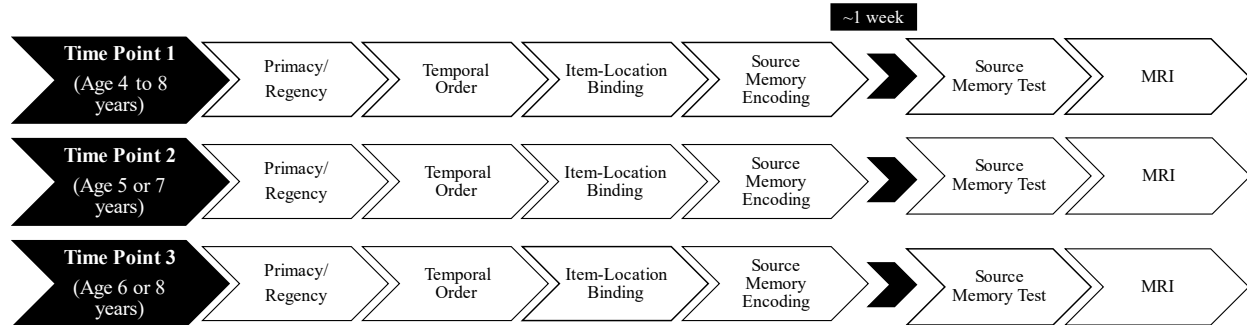
The proposed dissertation aimed to accomplish two goals. The first goal was to investigate associations between episodic memory and extra hippocampal brain regions that support memory in a longitudinal sample of 4 to 8-year-old children. This was assessed using

both a cross-sectional whole-brain approach and a longitudinal co-development approach. These results allow for the direct comparisons of longitudinal and cross-sectional methods in the same sample. The second goal was to examine whether individual differences in these brain regions are explained by differences in nap habituality using a cross-sectional sample of 3 to 5-year-old children. Importantly, brain regions for the second goal were partially motivated by the literature and partially by the results of the previous assessments.

To accomplish these goals, two separate data sets were used. The first data set (Study 1) utilized an accelerated longitudinal design with two cohorts assessed at three-time points. Specifically, the younger cohort was assessed at 4, 5, and 6 years of age. Whereas the older cohort was assessed at 6, 7, and 8 years of age (see Figure 1). Additionally, a cross-sectional sample of children were recruited at ages 5, 7, and 8 years. At each visit, subjects participated in an MRI scan and a battery of memory tasks that included two temporal order tasks, a source memory task, and an item-location binding task. This study was used to address the first aim of this dissertation, which was to examine longitudinal co-development of memory and cortical development in early to middle childhood.

Figure 1

Schematic depiction for the timing of data collection in Study 1.

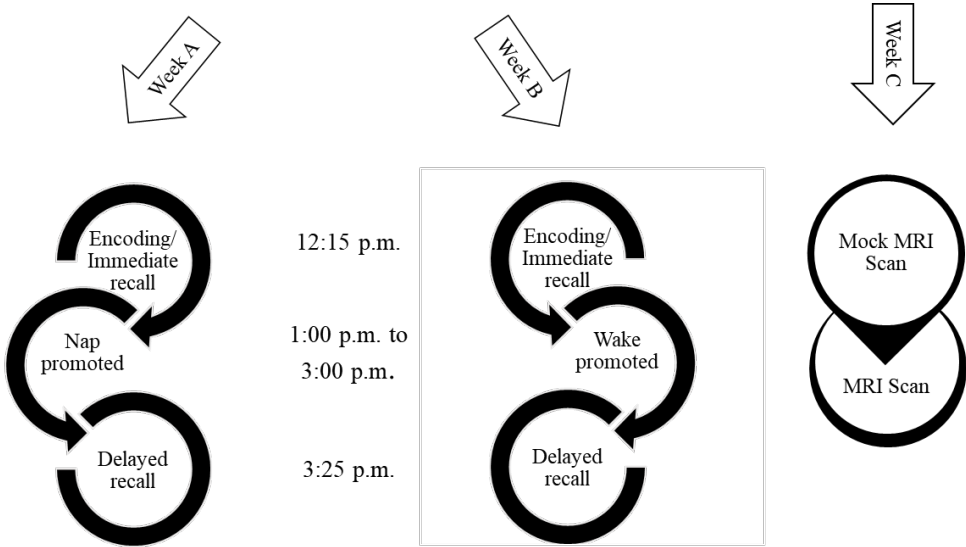


Note. Dark chevron represents an approximately 1-week delay.

Unfortunately, Study 1 was not designed to assess sleep habits and measures of nap habituality are rudimentary. Therefore, I used a second data set (Study 2) to examine associations between memory and the during early childhood. Study 2 includes a cross-sectional sample of 3 to 5-year-old children who were assessed at 3 separate visits, each one week apart. The first two visits were conducted in the child’s home, where they were tested on an item location binding task before and after both a nap and a wake session lasting the same duration. Nap and wake sessions were counterbalanced for order. During the third visit, children participated in an MRI scan where a T1 weighted image was collected (see Figure 2). Across the two-week testing period children also wore an actigraphy watch and parents completed a sleep diary to assess nap status. This study was used to assess the second aim of this dissertation, which was to examine differences in cortical thickness and surface area of regions that support memory development based on nap status during early childhood.

Figure 2

Schematic depiction for the timing of data collection in Study 2



Note. Week A and Week B were counterbalanced for order.

Chapter 2: Aims and Hypotheses

Aim 1: Examine relations between cortical thickness/surface area and memory performance during early to mid-childhood.

Part I: *Identify Memory-related ROIs using a Cross-sectional Sample of 4- to 8-year-olds.*

Hypothesis 1A: There will be significant negative associations between cortical thickness/surface area of ROIs (i.e., IFG, medial orbitofrontal cortex, superior frontal gyrus, rostral middle frontal gyrus, ACC, PCC, SPL, IPS, precuneus, ERC, PHG, temporal pole, superior temporal gyrus, middle temporal gyrus, lingual gyrus, LOC) and a composite memory variable such that greater cortical thickness/surface area will be associated with poorer memory performance. Hypothesis 1A will be assessed using both an *a priori* ROI analysis and an exploratory vertex by vertex whole brain analysis.

Part 2: *Examine Changes in ROIs and Latent Episodic Memory in 4-year-old and 6-year-old Longitudinal Cohorts.*

Hypothesis 1B: There will be age-related changes in cortical thickness/surface area of ROIs from ages 4 to 8 years. Hypothesis 1B will be assessed using latent growth curve modeling.

Research Question 1A: Are there age-related changes in cortical thickness/surface area of ROIs linear or non-linear from age 4 to 8 years. Further, are age-related changes in cortical thickness/surface area of ROIs linear or non-linear within the 4-year-old and 6-year-old cohorts. Research Question 1A will be assessed within cohorts using model comparison of latent growth curve models. Research Question 1A will be assessed between cohorts using t-tests of latent slopes and variances.

Hypothesis 1C: There will be linear age-related changes in Latent Episodic Memory.

Hypothesis 1C will be assessed using second order latent growth curve modeling.

Part 3: *Examine Co-Development of ROIs and Latent Episodic Memory.*

Hypothesis 1D: Longitudinal changes in cortical thickness/surface area will be associated longitudinal changes in memory performance. Hypothesis 1D will be assessed using bi-variate growth curve models.

Hypothesis 1E: cortical thickness/surface area at timepoint 1 will predict longitudinal changes in memory performance. Hypothesis 1D will be assessed using bi-variate growth curve models.

Aim 2: Examine differences in cortical thickness/surface area as a function of nap status.

Hypothesis 2A: There will be differences in cortical thickness/surface area of ROIs that longitudinal co-development with memory performance based on nap status during early childhood after controlling potential confounds. Specifically, cortical thickness/surface area will be greater habitual nappers compared to non-nappers. Hypothesis 2A will be assessed using both an *a priori* ROI analysis and an exploratory vertex by vertex whole brain analysis.

Table 2

Summary of Aims and Hypothesis.

Aim	Hypothesis
1) Examine relations between cortical thickness/surface area and memory performance during early to mid-childhood.	
P1) Identify ROIs Cross-sectionally	H1 _A) There will be significant negative associations between cortical thickness/surface area of ROIs and a composite memory variable such that greater cortical thickness/surface area will be associated with poorer memory performance.
P2) Examine Changes in ROIs and Latent Episodic Memory	H1 _B) There will be age-related changes in cortical thickness/surface area of ROIs from ages 4 to 8 years. RQ1 _A) Are age-related changes in cortical thickness/surface area of ROIs from ages 4 to 8 years linear or non-linear? H1 _C) There will be linear age-related changes in Latent Episodic Memory.
P3) Examine Co-Development.	H1 _D) Longitudinal changes in cortical thickness/surface area will be associated longitudinal changes in memory performance. H1 _E) Cortical thickness/surface area at timepoint 1 will predict longitudinal changes in memory performance.
2) Examine differences in cortical thickness/surface area as a function of nap status.	H2 _A) There will be differences in cortical thickness/surface area of ROIs that co-develop with memory performance based on nap status during early childhood after controlling potential confounds. Specifically, cortical thickness/surface area will be greater habitual nappers compared to non-nappers.

Chapter 3: Methods

To accomplish the objectives of this examination, I used two separate studies with pre-existing data. Study 1 includes a longitudinal sample of 4 to 8-year-old children that utilized a cohort sequential design. This study assessed brain structure via a T1-weighted MRI scan and memory via four memory tasks (i.e., Item-location Binding, Primacy, Temporal Order, and Source Memory) at three time points approximately one year apart. This study was used primarily to address Aim 1.

Study 2 includes a cross-sectional sample of 3 to 5-year-old children that explored associations between memory, sleep, and the brain during early childhood. This study assessed brain structure via a T1-weighted MRI scan and actigraphy to assess nap status. This study was used to address Aim two. In this chapter, I will describe the participants and methods used in each study.

Study 1 - Hippocampal-Memory Network Development and Episodic Memory in Early Childhood.

Participants.

The sample collected in Study 1 was drawn from a longitudinal dataset investigating memory and brain development in 4 to 8-year-old children. Previous papers have examined memory and hippocampal development from early to mid-childhood in this sample (e.g., Allard et al., 2023; Canada et al., 2019; Canada et al., 2020; Canada et al., 2021; Canada et al., 2022; Riggins et al., 2018). However, to date, relations between memory and cortical thickness have not been examined.

Recruitment. Subjects for Study 1 were recruited from the greater Baltimore-Washington area via the Infant and Child Studies Consortium, community advertisements, and word of mouth. Exclusion criteria included a diagnosis for a neurological condition, premature birth, developmental delays, or disabilities.

Demographics and descriptive statistics. Participants for Study 1 are 200 4- to 8-year-olds (100 were male). An accelerated longitudinal or cohort sequential design with three-time points was used. Specifically, two longitudinal cohorts were followed. The first cohort (61 participants) was enrolled at age 4 years (4-year-old cohort) and subsequently tested at ages 5 and 6 years. Participants enrolled in this younger cohort were over-sampled to ensure enough usable data was collected as there was also an MRI portion of the study that is not discussed here. The second cohort (41 participants) was enrolled at age 6 years (6-year-old cohort) and subsequently tested at ages 7 and 8 years. In addition, some children ($N=98$) participated at age 5, 7, or 8 years only, providing cross-sectional data for one time-point. To reduce bias, children with partial data were included. Children with only one time-point were included in the cohort and time-point that corresponded most closely to their age. For example, data for a child who was 7 years old was considered part of time-point 2 for the 6-year-old cohort.

Importantly, this experimental design provides an overlapping time point at age 6, allowing for an approximation of the longitudinal trajectory of memory and cortical development from age 4 to 8 years (Duncan et al., 1996). Of the 200 children recruited, 184 (89 females) provided usable brain and memory data during at least one-time point with a total of 329 usable scans across all three-time points. Longitudinally, 102 provided memory data at one-time point, 19 provided memory data at only two time points, and 63 provided data at all three-time points. Only 1 participant did not provide usable data at any of the three-time points and was excluded

from all analyses. Further, seven participants did not provide usable data at timepoint 1 but did provide usable data at timepoint 2 and/or timepoint 3. In the younger cohort, approximately 82 participants provided usable data during at least one-time point and in the older cohort, approximately 102 participants provided usable data during at least one-time point.

Table 3

Available MRI Sample and Average Age of Participants in Each Age Block for Study 1

M_{AGE} (N MRI Data)

<i>Age group</i>	<i>4 years</i>	<i>5 years</i>	<i>6 years</i>	<i>7 years</i>	<i>8 years</i>	<i>Total</i>
<i>Cross-Sectional Sample</i>	4.43 (46)	5.58 (29)	6.46 (41)	7.54 (30)	8.56 (31)	6.34 (177)
<i>4-year-old cohort</i>	4.43 (46)	5.47 (44)	6.43 (42)			
<i>6-year-old cohort</i>			6.46 (41)	7.55 (34)	8.52 (42)	

Note. Gold cells denote individuals providing MRI data for the 4-year-old cohort. Red cell denotes individuals providing MRI data for the 6-year-old cohort. M_{AGE}= Mean age in years.

Participants in this sample were 56% Caucasian, 13% Black, 5% Asian, and 19% Multiracial. Further, 15.5% of the subjects’ ethnicity was reported as Hispanic or Latino. Approximately 7% of parents did not reveal their child’s race and 4.5% did not reveal their child’s ethnicity. The sample consisted mostly of middle-to-high-income families (median = >\$105,000, range = < \$15,000 - >\$105,000) with 4% of the sample not revealing their income. Moreover, 81% of recruited children had a parent or parents who achieved at least a four-year college degree.

Prior to data collection, all methods were approved by the University of Maryland Institutional Review Board. Additionally, all parents provided informed consent and children provided verbal (i.e., children younger than 7 years) or written (i.e., children older than 7 years) assent depending on their age. Participants received age-appropriate prizes and parents/guardians received monetary compensation.

Behavioral Tasks.

Findings from behavioral tasks collected in Study 2 have previously been reported elsewhere. Therefore, prior to each task description, citations for relevant lab publications are provided.

Item-location binding task (Allard et al., 2023; Canada et al., 2021). To assess participants' feature binding abilities, they completed an item-location binding task (see Allard et al., 2023; Lorscheid & Reimer, 2005). Importantly, the present task and therefore a similar description of this task was previously used in another publication by the present author (Allard et al., 2023). Specifically, a forced-choice (yes/no) item-location recognition task was adapted from Lorscheid and Reimer (2005). First, participants viewed a practice booklet that familiarized subjects with eight black-and-white line drawings of common images (e.g., a balloon, heart, fish, lion, kite, snowman, pumpkin, and a frog), and a square 3×3 grid that would be used in the task. Next, the practice booklet was used to demonstrate an example of a potential test sequence. Specifically, for each trial, three different drawings were displayed sequentially in different grid squares and the participant was prompted to remember location of each picture on the grid. Finally, before starting a test trial, participants completed two practice trials to ensure their understanding of the task.

The task contained 16 target trials and 16 lure trials. Importantly, target and lure trials were presented in random order for a total of 32 trials. In target trials, the line drawing was presented in the same grid location during both the encoding and the testing phase. In lure trials, the line drawing was presented in a different grid location during the encoding and the testing phase. At the conclusions of each test trial, participants were asked to verbally respond "yes" if they believed the test trial was a target trial and to respond "no" if they believed it was a lure trial. The experimenter was responsible for recording all answers.

At the onset of each trial, "Ready?" was presented on the test screen. This was followed by a one-second blank screen. Next, the 3×3 grid was presented, initiating the encoding phase. During the encoding phase, three different line drawings were presented sequentially in three distinct locations on the 3×3 grid. The grid remained on the screen for a total of 3 seconds, with one second for each image to be displayed in a new location. Following encoding, a 4-second delay was initiated before the test phase. At the end of the delay, children were presented with the test item (i.e., a line drawing in a location on the grid). Then children were asked to determine if they had seen that line drawing in that location on the grid. The test item remained on the screen until the child responded. Each trial was followed by a two-second interval.

To assess performance on the item-location binding task, d' was calculated and included as indicator in the latent episodic memory variable (Snodgrass & Corwin, 1988). Specifically, d' is computed by Z transforming hit rates and false alarm rates. Then corrected false alarm rates are subtracted from corrected hit rates (i.e., $d' = Z(\text{Hit Rate}) - Z(\text{False Alarm Rate})$).

Source memory task (Canada et al., 2021; Riggins et al., 2018). To further assess participants' feature binding abilities, they completed a source memory task that assessed whether they could bind novel information to the context where it was first encountered (see

Canada et al., 2021; Drummey & Newcombe, 2002; Riggins, 2014). Importantly, unlike the other memory tasks, the source memory task was administered across 2 test sessions separated by approximately 1 week.

During the first session, participants were taught some new information (e.g., “Glass is made from sand.”) from two separate sources, a male puppet named “Henry” and an adult female named “Abby.” To maintain consistency across participants, all facts were presented via video recording. Each child was presented with 12 facts in total, 6 from each source. Importantly, facts were presented in source-based blocks. In other words, participants learned all 6 facts from Henry followed by all 6 facts from Abby, or vice versa. The order of the blocks was then counterbalanced across all subjects. For this task, there were 3 lists of facts. Each list contained different, but similar facts (e.g., “Honey is the only food that never goes bad.” or “Grapes are the most popular fruit in the world”). Lists were randomly assigned across all subjects and longitudinal subjects were provided with a different list at each time point. At the onset of the task, participants were prompted to remember the facts and informed that they would be tested the following week. However, participants were not informed that they needed to remember the source of each fact. Before children were told a fact, an experimenter would ask if the child already knew the fact (e.g., “What is glass made from?”). When a child demonstrated they knew a fact, the fact was omitted and a different fact from was presented from the same source. Each source was assigned 8 possible facts from each list. Therefore, if a participant knew at least 3 facts from one of the sources, that child received fewer facts in total ($N = 4$).

During the second session, participants’ memory for the first session was assessed. This was accomplished using a 22-item trivia questionnaire provided verbally by the experimenter. Importantly, before starting the questionnaire participants were informed that they had learned

some of the answers the week before from either Henry or Abby. They were also told that they may have learned some of the answers elsewhere (e.g., from school) and that they may not know the answer to some questions. Of the 22 facts on the trivia questions, children had learned 6 from Abby, 6 from Henry, 5 were considered to be commonly known facts by children (e.g., “What do you use to brush your teeth?”), and 5 were considered to be uncommonly known facts by children (e.g., “What animal’s tongue is two times as long as its body?”). For each list, there were two possible random orders of the facts. These were counterbalanced across participants.

For each question (e.g., “What is glass made from?”) participants were first given the opportunity to demonstrate free recall of the fact. When a participant expressed that they did not know the answer, a recognition option was given instead. For this option children were provided four multiple-choice answers (e.g., Chameleon, Dog, Ant Eater, Snake). Once an answer was provided, the experimenter asked who taught the child that fact. Again, subjects were first provided with a free recall opportunity. If the participant indicated they did not know who had taught them the fact, they were provided with another recognition option. For this option, children were provided with five multiple-choice answers (e.g., parent, teacher, Abby, Henry, or they just knew). Proportion correct answers for both fact and source questions were calculated and included as an indicator for the latent episodic memory variable.

Order memory-recognition (primacy) (Canada et al., 2020; Canada et al., 2021). To assess recognition memory for temporal order, a primacy task was included. Specifically, this investigation utilized a modified version of a primacy task used in several other studies that examined primacy abilities in children (Alden, 1994; Mathews & Fozard, 1970). Prior to the primacy test phase, participants were provided with 2, 4 item practice lists to ensure they understood the task. During this practice phase, children were provided feedback on their

performance to help solidify the task instructions. During the test phase, participants were provided with four lists of images (2 lists included 8 items and 2 included 12 items). All lists were comprised of black and white line sketches of ordinary objects (e.g., a bicycle, a bear, an airplane, etc.). In this paradigm, participants were prompted to remember the order of the images. Each image was presented to the child sequentially with a verbal cue (e.g., bicycle, bear, airplane) at a rate of 1 image every 2 seconds.

After the experimenter finished presenting a list, the participant was either immediately provided with two forced-choice questions concerning the order of two images in the list (e.g., “which image came first”) or they were given a distractor task (e.g., instructions to draw a picture) prior to the forced-choice questions. The purpose of the forced choice questions was to assess the child’s primacy judgment by determining which of two pictures presented in a list the participant remembered seeing first. The order of the lists and which lists were followed by a distractor task was counterbalanced across subjects. Each pair of images that were used for primacy judgments were presented one image apart in the list sequence. Additionally, one pair was taken from the first part of the list (i.e., the first 4 or first 6 items) and the second pair from the second part of the list (i.e., the last 4 or 6 items). Specifically, the pairing of item 2 with item 4 and item 5 with item 7 was always used for the 8-item list. Similarly, for one of the 12 item lists, item 3 was paired with item 5 and item 7 was always paired with item 9. However, on the other 12 item list, item 4 was paired with item 6 and item 8 was paired with item 10. This list design was used to avoid overlaps of already paired items with other items and to avoid using the first and last items in each list. After completion of the task, proportion of correct responses was included as the indicator variable in the latent episodic memory variable.

Temporal memory recall (Canada et al., 2020; Canada et al., 2021). An ordered sequence recall task was used to assess children’s memory for the temporal order of events, (see Bauer et al., 2013; Canada et al., 2020; Pathman & Ghetti, 2014). To ensure children understood the task, they were shown a 4-item practice sequence (Yard). Next, subjects were randomly assigned to two 9-item test sequences (either Fair, Park, or Pet Shop) and the sequences were counterbalanced for order. Each sequence was introduced using a verbal cue related to the sequence (e.g., for the Yard sequence they would say “I’m going to show you how I work in the yard.”). Then, the child was shown laminated cards with each item for that the sequence which were introduced with a distinct verbale cue (e.g., “mow the lawn”). Importantly, no temporal or causal language cues were given (e.g., “first”, “finally”, “next”). Once all 9 items were introduced, the original verbal event cue was given a second time, (e.g., “That’s how I work in the yard”). Finally, the item cards were rearranged, and participants were asked to rebuild the sequence in its original form. Importantly, for one of the 9-item picture sequences, participants were given a randomly assigned distractor task (e.g., tic-tac- toe) between the presentation of the sequence and sequence reconstruction.

After the participant reassembled the sequence, performance was recorded. Specifically, performance was scored on the number of contiguous pairs in the child’s reconstruction (two adjacent items placed in the same order as the original sequence). Thus, each sequence included 8 potential contiguous pairs. Next, accuracy across the immediate and delayed reconstructions were compared. Results demonstrated that the reconstructions were similar. Therefore, the proportion of contiguous pairs recalled across both 9 item sequences was included in the construction of the latent episodic memory variable.

Intelligence. Measures of visual spatial and verbal IQ were collected using age-appropriate subtests of the Wechsler Preschool and Primary Scale of Intelligence (WPPSI) (i.e., for 4 to 5 year olds) and the Wechsler Intelligence Scale for Children (WISC) (i.e., for 6 to 8 year olds). Scaled scores for the block design (i.e., visual-spatial IQ) and word recognition (verbal IQ) subtests were used to determine whether associations between episodic memory and the brain were specific or attributed to general cognitive ability.

MRI.

Acquisition. Prior to MR data acquisition, all participants were screened for MRI contraindications (i.e., metal, claustrophobia) and completed training in a mock scanner that allowed the child to become comfortable with the scanner environment. The scanner used for both Study 1 and Study 2 was a Siemens 3.0-T scanner (MAGNETOM Trio Tim System, Siemens Medical Solutions, Erlangen, Germany) with a 32-channel coil. A high-resolution T1-weighted magnetization-prepared rapid gradient-echo (MPRAGE) image sequence that consisted of 176 sagittal slices (.9 mm isotropic; 1900 ms TR; 2.32ms TE; 900ms inversion time; 9° flip angle; pixel matrix= 256 x 256) was used to acquire structural data.

Processing. Structural T1-weighted image preprocessing consisted of smoothing, skull stripping, image registration, motion correction, and subcortical segmentation. Standard procedures such as cortical surface reconstruction, cortical, and subcortical segmentation to determine white-matter boundaries were conducted using FreeSurfer Version 5.1 for Study 1 and 6.0 for Study 2 (surfer.nmr.mgh.harvard.edu; Fischl, 2012; Fischl et al., 2002). Importantly, in our lab, we previously examined the effects of using these two different versions of FreeSurfer in the sample data set by processing cases in both version 6.0 in version 5.1 and then conducting

correlation analysis. Results from these analyses were similar and suggest that differences in Freesurfer versions do not drive the observed effects (Ewell et al., In Press; Fitter et al., 2022).

After initial processing, accuracy of boundary lines was evaluated by trained editors. In the case of errors extending for more than 7 slices, such as inclusion of the skull from either the gray/white or pial line, editors made corrections. Corrections were made by first changing the watershed value within FreeSurfer to either enhance or decrease the skull strip. If the error still remained manual edits were made (Ducharme et al., 2016).

After all corrections were made, a third independent editor rated each brain on a scale from 1 to 5, with 1 being perfect quality and 5 being unusable. Scans that were rated as unusable contained extreme banding from motion and unreliable white pial boundaries that were unclear to the naked eye. To retain maximum data, scans rated 4 or higher were included in this analysis. In Study 1, 14 brains were rated as 4 or higher. In Study 2, 2 brains were rated as 4 or higher. Importantly, images for all subjects that provided usable data at more than one timepoint were additionally processed with longitudinal pipeline (Reuter et al., 2012) in FreeSurfer. This pipeline conforms subject data from all available time points to a common subject-template (called the BASE). Then, information from each cross-sectional time point and the BASE is used to create a new set of longitudinally processed runs for each subject. As a result, each run is processed in reference to other timepoints from the same subject leading to less variability. Cortical thickness was then computed by determining the distance between boundary lines (Fischl & Dale, 2000). The Desikan-Killiany Atlas was used for cortical parcellation (Desikan et al., 2006).

Study 2 - Hippocampal Development and Sleep-Dependent Memory Consolidation in Preschoolers.

Participants.

The sample collected in Study 2 were drawn from a larger dataset investigating memory, sleep, and brain development in 3 to 5-year-olds. Previous papers have examined associations between emotion regulation, parenting, and 24-hour behaviors with structural measures of the cortex and hippocampus (e.g., Allard et al., Under Review; Ewell et al., In Press; Fitter et al., 2022; St. Laurent et al., 2022). However, past work has not examined differences in cortical thickness or surface area based on nap status.

Recruitment. All subjects recruited for Study 2 were considered to be typically developing children at the time of data collection and were living in the greater Baltimore-Washington area. Further, participants for this study were recruited via the same channels described for Study 1. Exclusion criteria for this study included abnormal circadian function; any brain abnormality, brain trauma, psychiatric disorder, neurological disorder, learning disability, or developmental delay; a familial history of autism spectrum disorder; and premature birth prior to 35 weeks gestation.

Demographics and Descriptive Statistics. Participants for Study 2 are a cross-sectional sample of 56, 3 to 5-year-old children who partook in a larger study assessing the effect of nap habits on memory and brain development in early childhood. Of the 56 participants that provided usable data during at least 1 time point, 46 provided a T1 scan. Of these, 2 scans were deemed unusable.

In this sample, 64.3% were Caucasian, 16.1% were Black, 7.1% were Asian, and 8.9% were Multiracial. Additionally, 11% of parents did not reveal their child’s race. Furthermore, 8.6% of participants identified as Hispanic or Latino, 87.5% identified as not Hispanic or Latino, and 3.6% of parents did not reveal their child’s ethnicity. Importantly, this sample consisted mostly of middle-to high-income families (median = >\$195,000, range = < \$15,000 - >\$195,000). Moreover, most (94.5%) of the recruited children had a parent or parents who achieved at least a four-year college degree.

In Study 2, actigraphy and parent report was used to categorize children into three distinct nap status groups based on previous literature. The three groups are habitual nappers (i.e., children who nap 5 or more days per week), intermediate nappers (children who nap 2 to 4 days per week), and non-habitual nappers (children who nap 2 or less days per week). Cut offs for these categories are based on past literature (Allard et al., Under Review; Desrochers et al., 2016; Kurdziel et al., 2013; Kurdziel et al., 2018). Based on this criteria, Study 2 consisted of 14 habitual nappers, 19 intermediate-nappers, and 11 non-nappers (see table 4).

Table 4

Number of Children Contributing Behavioral and Neuroimaging Data for Study 2

<i>Nap Status</i>	<i>N (MRI)</i>	<i>Mean (MRI age)</i>	<i>Range (MRI age)</i>
<i>Nappers</i>	14	4.057925636	3.26 - 5.00
<i>Intermediate-Nappers</i>	19	4.420423412	3.40 - 5.77
<i>Non-Nappers</i>	11	4.361643836	3.21 - 5.82
<i>Total</i>	44	4.279997628	3.21 - 5.82

Prior to data collection, all methods were approved by the University of Maryland Institutional Review Board. Additionally, parents provided informed consent and children provided verbal assent. Participants received age-appropriate prizes and parents/guardians received monetary compensation.

Nap Habitually.

Actigraphy. Two weeks prior to the MR scan, children were given an actigraphy watch that they were told to wear continuously over the following two weeks. These watches record environmental light exposure and participant movement levels allowing an experienced coder to differentiate sleep from wake. Furthermore, to verify estimates, parents were instructed to record event markers (via a button on the watch) just prior to sleep onset and just after sleep offset. If event markers were not present, sleep was scored manually. Importantly scoring of watches was conducted using Philips Respironics and a previously standardized protocol (Acebo et al., 2005). Nap status was calculated using the following formula: (total days napped/total days recorded)⁷. Of the 44 participants included, 31 provided usable actigraphy data (i.e., 3 or more days).

Parent Report Measures. Two weeks prior to the MR scan, parents were provided with a sleep diary. It required parents to record all sleep bouts, including naps and overnight sleep, during the same two-week period that the child was instructed to wear the actigraphy watch. Average number of napping days was calculated using the same formula used for actigraphy. Of the 44 children that provided data for this examination, nap status for 10 was derived from sleep diaries. If a participant was missing actigraphy data and the sleep diary ($N=3$), a lab specific questionnaire was used (i.e., “How many days a week does your child nap?”).

24-hour Sleep Duration

Average 24-hour sleep duration was calculated using actigraphy data by computing the amount of time between bedtime and wake-up time for naps and overnight sleep separately, then durations were averaged across all available days. Next, the average nap and overnight durations were summed.

MRI.

Acquisition. During an earlier visit that occurred in the child's home, subjects were introduced to the MR environment via a fabric tunnel and an audio track that featured scanner sounds. Additionally, a book was read to each child that demonstrated the purpose of the "brain camera" and the order of events that would occur during their visit to the Maryland Neuroimaging Center. Importantly, the acquisition process at the neuroimaging center was identical to Study 1.

Processing. T1 processing was identical to Study 1, with the sole exception that Freesurfer version 6.0 was used instead of version 5.1. Furthermore, cortical thickness and surface area were also acquired similar to Study 1. (surfer.nmr.mgh.harvard.edu; Fischl, 2012; Fischl et al., 2002). For the purpose of Aim 2, hippocampal volumes and ICV were additionally calculated using Freesurfer's automated subcortical segmentation process. Then, automated hippocampal volumes were adjusted using the Automatic Segmentation Adapter Tool (ASAT; nitrc.org/projects/segadapter; Wang et al., 2012).

Chapter 4: Data Analysis

Aim 1: Examine relations between cortical thickness/surface area and memory performance during early to mid-childhood.

Selection of ROIs.

Existing literature demonstrates that several cortical regions support episodic memory abilities during childhood (see Appendix B). Based on this literature, 16 ROIs were selected for this analysis using the Desikan-Killiany Atlas for cortical parcellation (Desikan et al., 2006). This atlas was chosen because it is the commonly used in the developmental memory literature and utilizing it allows for finding comparison across a variety of previous studies (e.g., Amlien et al., 2018; Bauer et al., 2019; Chad-Friedman et al., 2021; Fjell et al., 2019; Schommartz et al., 2023; Squeglia et al., 2013; Yu et al., 2018). These regions include IFG, medial orbitofrontal cortex (mOFC), superior frontal gyrus, rostral middle frontal gyrus, ACC, PCC, SPL, IPS, precuneus, ERC, PHG, temporal pole, superior temporal gyrus, middle temporal gyrus, lingual gyrus, LOC. IFG and ACC are comprised of several subregions. However, past research has not found differential effects in these subregions, therefore values for cortical thickness and surface area were averaged across subregions to create a singular value per ROI.

A Latent Measure of Memory Development.

A previous study published from our lab produced a latent episodic memory variable using confirmatory factor analysis (Canada et al., 2022). Specifically, this variable was created using data from Study 1 described in the methods section, and included the item location binding task, source memory task, primacy task, and temporal order memory task. This latent episodic memory variable demonstrated strong factorial invariance and trajectory convergence between

the 4-year-old and 6-year-old cohort (see Canada et al., 2022). Together, this suggests that this structure of latent episodic memory assesses the same latent variable across cohorts and ages. As a result, this variable was a good candidate for this analysis and was used as a measure of episodic memory performance. Further, to assess cross-sectional associations of the memory variable, a composite memory variable was extracted from the confirmatory factor analysis. This is critical as latent structures cannot be assessed in qdec. Importantly, when the latent memory variable is extracted, it is considered a “composite” variable and is no longer “latent.” Thus, when the variable is extracted, it is referred to it as the “composite memory variable.” When the variable is not extracted, it is referred to as the “latent episodic memory variable.”

Power Analysis.

To assess whether our sample was large enough to address *Hypothesis 1A*, a power analysis was conducted using G*Power version 3.1.9.6 (Faul et al., 2007), based on findings from Schommartz et al., (2023) ($N = 63$). This study found a moderate effect size of $f^2 = .14$ when examining associations between cortical thickness and episodic memory performance in children aged 5 to 7 years (Cohen, 1988). Based on these findings, a significance criterion of $\alpha = .05$, and power = .80, the minimum sample size needed for this effect size is $N = 156$. Thus, the obtained sample size of $N = 177$ is adequate to test the study hypothesis.

Power analysis in latent growth curve modeling typically reveals that large samples, often in the thousands, are required to conduct models with small degrees of freedom. For example, I conducted a power analysis using quantpsy for the proposed latent growth curve model for *Hypothesis 1B* (Depicted in Figure 8; MacCallum et al., 2006; Preacher & Coffman, 2006). In the analysis, I assumed an $\alpha = .05$, power = .80, and $df = 1$. Findings revealed that this model

would technically require a sample of 856 participants to detect large effects. However, previous work has demonstrated that changes in both the amygdala and the hippocampus can be detected using latent growth curve models with similar sample sizes (Canada, Botdorf, et al., 2020; Canada et al., 2021). This is likely because omnibus approaches do not account for model parameters. One solution to this, is Monte Carlo simulation that assess power using predicted effects sizes and variances (Muthén & Muthén, 2002). Therefore, to assess power for *Hypothesis 1B*, I used a Monte Carlo simulation to assess whether a sample of 82 (i.e., N for the 4-year-old cohort) could detect changes in cortical thickness given a moderate effect size of 0.3 for the slope, assuming other common parameters suggested by Muthen and Muthen (2002). The result showed that the effect would be detected 88% of the time. However, note that I was unable to fully account for data missingness thus this is likely an over-estimation of power.

Importantly, for *Hypothesis 1C*, no a priori power analysis was conducted, because we have previously demonstrated that this sample is large enough to detect changes in latent episodic memory during early childhood using a model with fewer degrees of freedom (Canada et al., 2022). Additionally, no power analysis was conducted for *Hypothesis 1D* because no previous analysis has examined co-development of memory and these cortical ROIs.

Outliers.

Past research has demonstrated that general linear models are highly susceptible to the effects of extreme outliers (Osborne & Overbay, 2004). Further, previous work shows that extreme outliers that are at least 3 standard deviations above or below the grand mean can significantly bias findings. For example, extreme outliers can mask true effects, even when values are naturally occurring in the sample (Barnett & Lewis, 1994). Thus, prior to analysis,

extreme outliers were identified using a box plot method and excluded if values were greater than 3 standard deviations above the mean. The same process was used in Study 1 and Study 2.

Cross-sectional ROI Analysis.

To assess cross-sectional associations between the composite memory variable and cortical ROIs, data from the first wave of Study 1 ($N = 177$) was used. Specifically, separate lateralized regressions were conducted for each cortical measure (i.e., thickness and surface area) to predict performance on the composite episodic memory variable. All analyses controlled for age and sex. Additionally, IQ was explored as potential covariate to ensure that associations between episodic memory and the cortex were not attributed to general cognitive abilities. Further, to ensure that findings were specific to regions associated with memory performance, associations between the composite episodic memory variable and total gray matter volume were also assessed.

Given the exploratory nature of this analysis, all significant findings are reported. However, this approach will yield 64 separate comparisons, thus, p values were adjusted based on the false discovery rate to control for the likelihood of false positives. However, both are reported. By reporting both criteria, results can be evaluated using both liberal (uncorrected) and conservative (corrected) approaches.

Cross-sectional Whole-brain Analysis.

Following the ROI analysis, a whole brain vertex-by-vertex analysis was employed to confirm previously identified regions and to investigate previously unidentified regions. In Freesurfer's QDEC application, linear regressions examining associations between cortical thickness, surface area, and the composite memory variable were conducted controlling for age,

sex. Additionally, Monte Carlo simulations were utilized to correct for multiple comparisons across both hemispheres and estimate appropriate cluster sizes (Hagler et al., 2006). All analyses utilized a minimum threshold of $p < .05$.

Latent Growth Curve Modelling.

To best characterize and assess longitudinal growth in episodic memory performance and the brain, latent growth curve models were used. These are a specific subclass of Structural Equation Models that were selected because of their ability to effectively assess change over time (McArdle, 2009). Additionally, these models are robust to potential ceiling and floor effects and allow for the comparison of slopes between cohorts. To reduce bias in estimates and address missing data, full information maximum likelihood (FIML) estimation was utilized. All latent analyses were conducted in Mplus (v8; Muthén & Muthén, 2017).

Longitudinal Development of Cortical Thickness and Surface Area. To assess changes in cortical thickness, surface area, and total gray matter volume across early to middle childhood, separate lateralized cohort-specific first-order latent growth models were used for each cortical measure. To estimate latent intercepts for all variables at ages 4- and 6-years, the loadings for all waves were constrained to 1. Further, to characterize an appropriate slope within each cohort, the loading for timepoint 1 (i.e., age 4 in the younger cohort or age 6 in the older cohort) was constrained to 0, the loading for timepoint 2 (i.e., age 5 in the younger cohort or age 7 in the older cohort) was freely estimated, and the loading for timepoint 3 (i.e., age 6 in the younger cohort or age 8 in the older cohort) was constrained to 2. These models allowed characterization of both linear and non-linear growth trajectories. However, they were also just identified with no remaining degrees of freedom. This limits the ability to determine model fit and could have resulted in partial under-identification during the assessment of co-development.

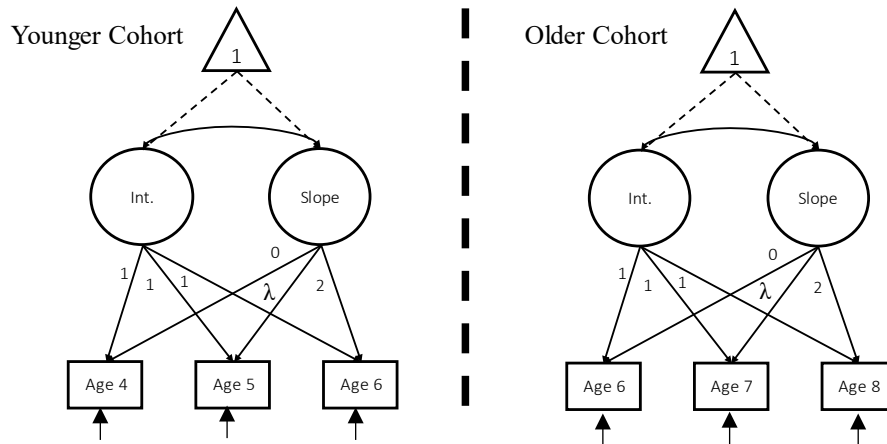
Therefore, once a loading for timepoint 2 was estimated each model was re-run by fixing timepoint 2 to the estimated loading value. This allows for a data-driven characterization of growth trajectories that does not assume ROIs exhibit linear or set ratios of non-linear (e.g., for example twice as much change from 4 to 5 years compared to 5 to 6 years). Importantly, this strategy sometimes resulted in negative latent slope variances, creating an impossible solution. When this occurred, residuals in the latent growth models for ROIs were constrained to be equal. If this still resulted in a negative variance and that variance was non-significant, the variance of that latent variable was constrained to zero. This resolved all impossible solutions. Notably, latent growth curve models were not corrected for multiple comparisons due to the theoretically driven nature of these models. Once an acceptable model was determined, t-test were conducted to assess differences in estimated latent slopes between cohorts. This allowed for the characterization of linearity from 4 to 8 years.

To determine if growth trajectories are lateralized, a t-test was conducted comparing estimated latent slopes and variances for the left and right hemispheres. If a region demonstrated similar growth across hemispheres, future analyses with that region assessed bilateral effects by averaging across hemispheres to create a single measure of cortical thickness for each ROI or by adding values across hemispheres to create a single measure of surface area for each ROI. However, to best characterize associations, both lateral and bilateral models are reported.

Finally, to determine if there were associations between the developmental trajectories of cortical thickness and surface area, two candidate regions were selected that demonstrated the most common combination of developmental trajectories. For these regions, latent slopes were extracted from latent growth models and regressions were conducted in the younger and older cohort separately.

Figure 3

Conceptual Depiction of the Latent Growth Models used to Analyze the Development of ROIs



Note. Growth models for the younger cohort are depicted on the left and for the older cohort on the right. The square boxes represent measures of cortical thickness or surface area at each age (e.g., Age 4 would reflect cortical thickness at Age 4). The λ symbol represents a freely estimated loading.

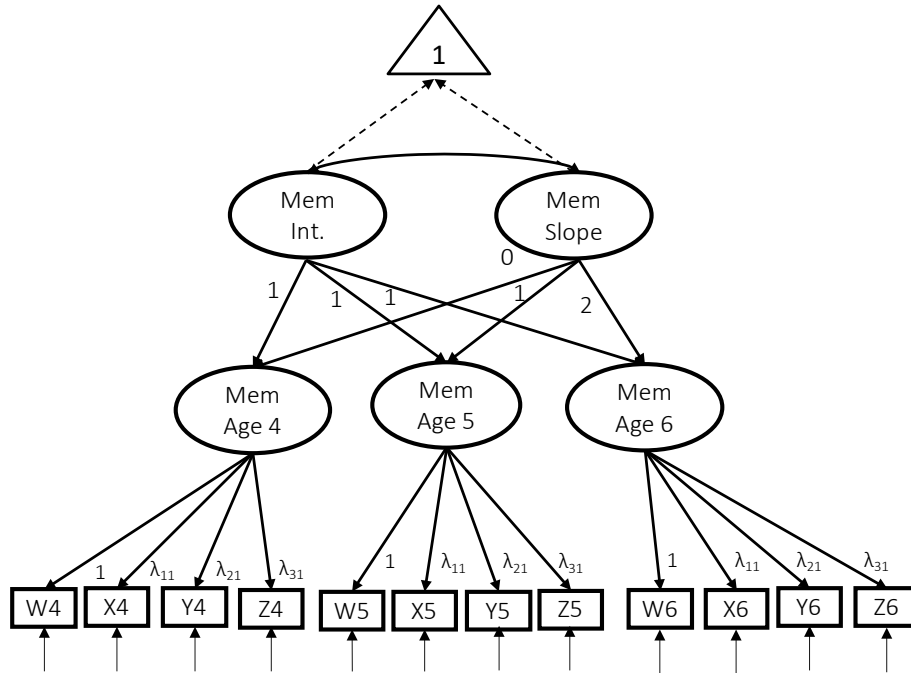
Longitudinal development of Latent Memory. Previous work in our lab assessed longitudinal development of the latent episodic memory variable using piecewise second-order growth curve models and found linear increases from age 4 to 8 years (Canada et al., 2022). However, for the purpose of the co-development models used in this investigation, piecewise models were not an ideal fit. Specifically, piecewise models would produce different time scales for change in memory and change in ROIs. For example, the latent memory slope would measure change from 4 to 5 years and 5 to 6 years, while the latent ROI slope would only measure change from 4 to 6 years. As a result, simple multi-cohort (i.e., 4-year-old and 6-year-

old) second order latent growth curve models were used to assess change in the latent episodic memory variable (Figure 8).

To estimate latent intercepts for the latent episodic memory variable at ages 4- and 6- years, the loadings for all waves were constrained to 1. Further, to characterize an appropriate slope within each cohort, the loading for timepoint 1 (i.e., age 4 in the younger cohort or age 6 in the older cohort) was constrained to 0, the loading for timepoint 2 (i.e., age 5 in the younger cohort or age 7 in the older cohort) was constrained to 1, and the loading for timepoint 3 (i.e., age 6 in the younger cohort or age 8 in the older cohort) was constrained to 2. Importantly, this model assumes linear change from 4 to 6 years and from 6 to 8 years. Additionally, error covariance parameters for each memory indicator (i.e., the behavioural tasks) were estimated across waves. For example, performance on the item-location binding task at wave 1 was covaried with performance on the item-location binding task at the following 2 waves. This allowed models to account for any additional components of the task that are not attributed to the latent measure of episodic memory. Next, to characterize linearity from 4 to 8 years, t-test were conducted to assess differences in estimated latent slopes between cohorts.

Figure 4

Conceptual Diagram of Linear Second-order Latent Growth Curve Models for Episodic Memory in the 4-year-old Cohort



Note. W= Temporal Order, X= Source Memory, Y= Feature-Binding, Z=Primacy. Error covariances omitted for clarity.

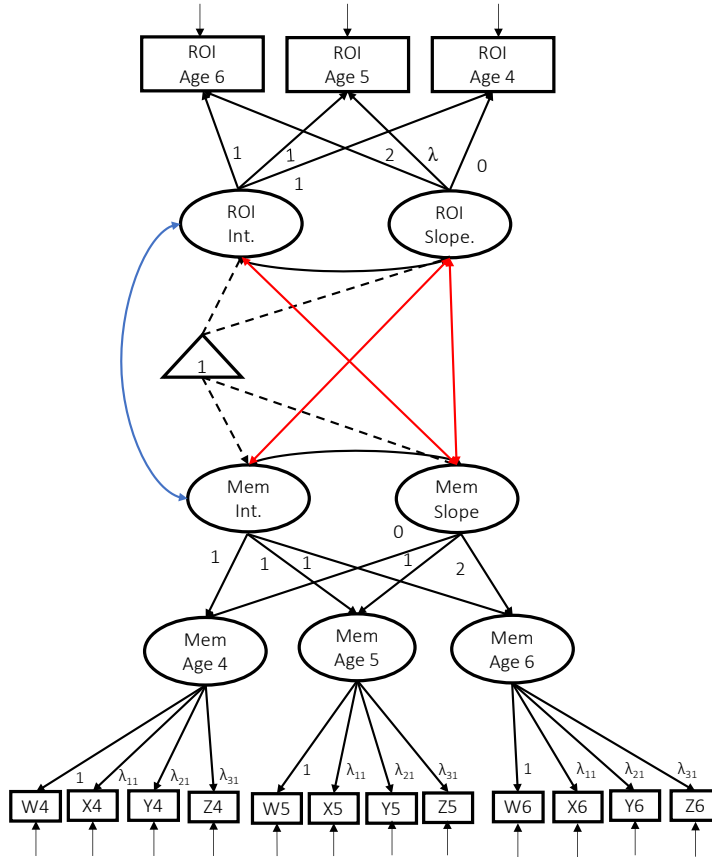
Co-development ROI's and latent memory using bivariate growth curve models. To assess the parallel development of latent episodic memory and cortical measures of each ROI (i.e., cortical thickness surface area, total gray matter volume), bivariate growth curve models were used. Bivariate growth curve models are a type of latent growth analysis that examine associations between latent intercepts (i.e., the starting value at age 4 in the younger cohort or age 6 in the older cohort) and latent slopes (i.e., change in variables from 4 to 6 or 6 to 8 years) of multi-domain growth curve models (Curran & Hancock, 2021, 2021; McArdle, 2009). As a

result, these models can provide information about the co-development of brain-behavior relations. Specifically, for this investigation, multi-domain (i.e., episodic memory and cortical measures of each ROI) multi-cohort (e.g., 4 and 6-year-old cohorts) developmental trajectories were modeled longitudinally and associations between latent intercepts and latent slopes for each domain were determined (Figure 5). Next, associations between latent variables (i.e., slopes and intercepts) were corrected for multiple comparisons by adjusting p values based on the false discovery rate. Like cross-sectional analysis, both corrected and uncorrected findings are reported.

Assessing Model Fit for Longitudinal Models. To assess overall model fit, root mean square error of approximation ($RMSEA \leq 0.08$ is considered good fit) was calculated and examined; CFI and SRMR will not be assessed (Hancock, personal communication, May 30, 2023).

Figure 5

Conceptual Diagram of Bivariate Growth Curve Model Assessing Co-development of Episodic Memory and Selected ROIs.



Note. Red and blue lines represent correlations between latent intercepts and latent slopes.

Dotted lines represent the estimated means structure. *Note.* Error covariances omitted for clarity.

W= Temporal Order, X= Source Memory, Y= Feature-Binding, Z=Primacy.

Aim 2: Examine differences in cortical thickness/surface area as a function of nap status.

Preliminary Analysis.

Power analyses were not conducted prior to addressing aim 2 part 1 because no previous analysis has examined differences in cortical thickness based on nap status. Therefore, there is no estimated effect size available. To identify potentially confounding variables, a one-way ANOVA was conducted to examine differences in age, average 24-hour sleep duration, and total gray matter volume based on nap status. Additionally, a chi-squared test was used to examine differences in sex.

ROI Analysis

To assess differences in the brain based on nap status, I used separate one-way ANCOVAs for each cortical ROI and cortical measure (i.e., thickness, surface area, total gray matter volume). When these analyses identified significant differences based on nap status, a Tukey's HSD was used to assess post hoc comparisons in that region. Further, p values were adjusted using the false discovery rate to control for multiple comparisons. However, given the potentially large number of included cortical ROIs and the a priori nature of these assessments, findings from both before and after the adjustment are presented. Finally, to explore whether associations between ROIs and nap status may be attributed to downstream effects from the hippocampus, relations between significant ROIs and hippocampal volumes were assessed using regression, controlling for ICV.

Vertex by Vertex Analysis.

Following the ROI analysis, a second whole brain vertex-by-vertex analysis was employed to confirm previously identified regions that differ based on nap status and to investigate previously unidentified regions. However, in contrast to the proposal, I was not able to conduct ANOVA's in QDEC because this application can only handle variables with two factors. Therefore, regressions examining associations between cortical thickness, surface area, and average naps per week controlling for age were conducted instead. Additionally, Monte Carlo simulations accounting for both right and left hemisphere were utilized to estimate proper cluster sizes and correct for multiple comparisons (Hagler et al., 2006). All analyses utilized a minimum threshold of $p < .05$.

Chapter 5: Results

Aim 1: Preliminary Analysis

Outlier analysis uncovered 6 outliers that were at least 3 standard deviations above the mean in Study 1. This included 1 from ERC at timepoint 1, 3 from PHG at timepoint 1, 1 from rostral middle frontal gyrus at timepoint 1, and 1 from rostral middle frontal gyrus at timepoint 3. These values were not included in analyses.

Preliminary analysis examined associations between potential covariates (i.e., age, sex, IQ) and the composite memory variable. Results demonstrated that age significantly predicted performance on composite memory variable $F(1,174) = 2.040, p < .016$. Specifically, there was a positive association between the composite memory variable and age such that older ages had higher scores on the composite variable memory, ($\beta = 0.047, p < .001$). In contrast, there was no significant difference in the composite memory variable based on sex, $F(1,174) = 0.139, p < .71$. However, sex will still be included as a covariate in all cross-sectional analyses due to the a president in the brain literature (Lenroot et al., 2007). Additionally, a regression analysis showed that there was no significant association of the composite memory variable with visual-spatial IQ, $F(1,174) = 3.338, p = .07$ or verbal IQ $F(1,174) = 3.338, p = .08$. Therefore, IQ will not be included as a covariate.

Next, to ensure that all associations between the composite memory variable and measures of ROIs were not attributed to the cortex generally, associations between the composite memory variable and total gray matter volume were examined. Findings showed that there was a significant positive association between total gray matter volume and the composite memory variable, $F(1,174) = 8.398 p = .004$. However, this association became non-significant ($p = .13$)

when controlling for age and sex, $F(3,172) = 64.46$ $p < .001$. As a result, it can be concluded that all cross-sectional associations are likely ROI specific.

Aim 1 (Part 1): Cross-Sectional Associations between Memory and ROIs

ROI Analysis.

In analyses examining cortical thickness, results demonstrated a negative association between the right ACC and the composite memory variable when controlling for age and sex ($p = .02$; see Table 5). In other words, a thinner ACC was associated with better performance on the composite memory variable in 4 to 8-year-olds (see Figure 6). There were no other significant associations between cortical thickness and the composite memory variable.

In analysis examining cortical surface area, results demonstrated a positive association between the composite memory variable and surface area of the left medial orbital frontal gyrus, bilateral rostral middle frontal gyrus, left ACC, left SPL, and bilateral precuneus ($ps < .05$; see Table 5). In other words, greater surface area in these regions was associated with better performance on the composite memory variable in 4 to 8-year-olds (see Figure 7). There were no other significant associations between cortical surface area and the composite memory variable.

Next, significance values were adjusted using a false discovery rate to control for multiple comparisons. Unfortunately, no significant association for thickness or surface area survived the correction for multiple comparisons (see Table 5). Thus, although relations were detected, given our sample size and the number of comparisons it is possible that these associations were spurious.

Table 5

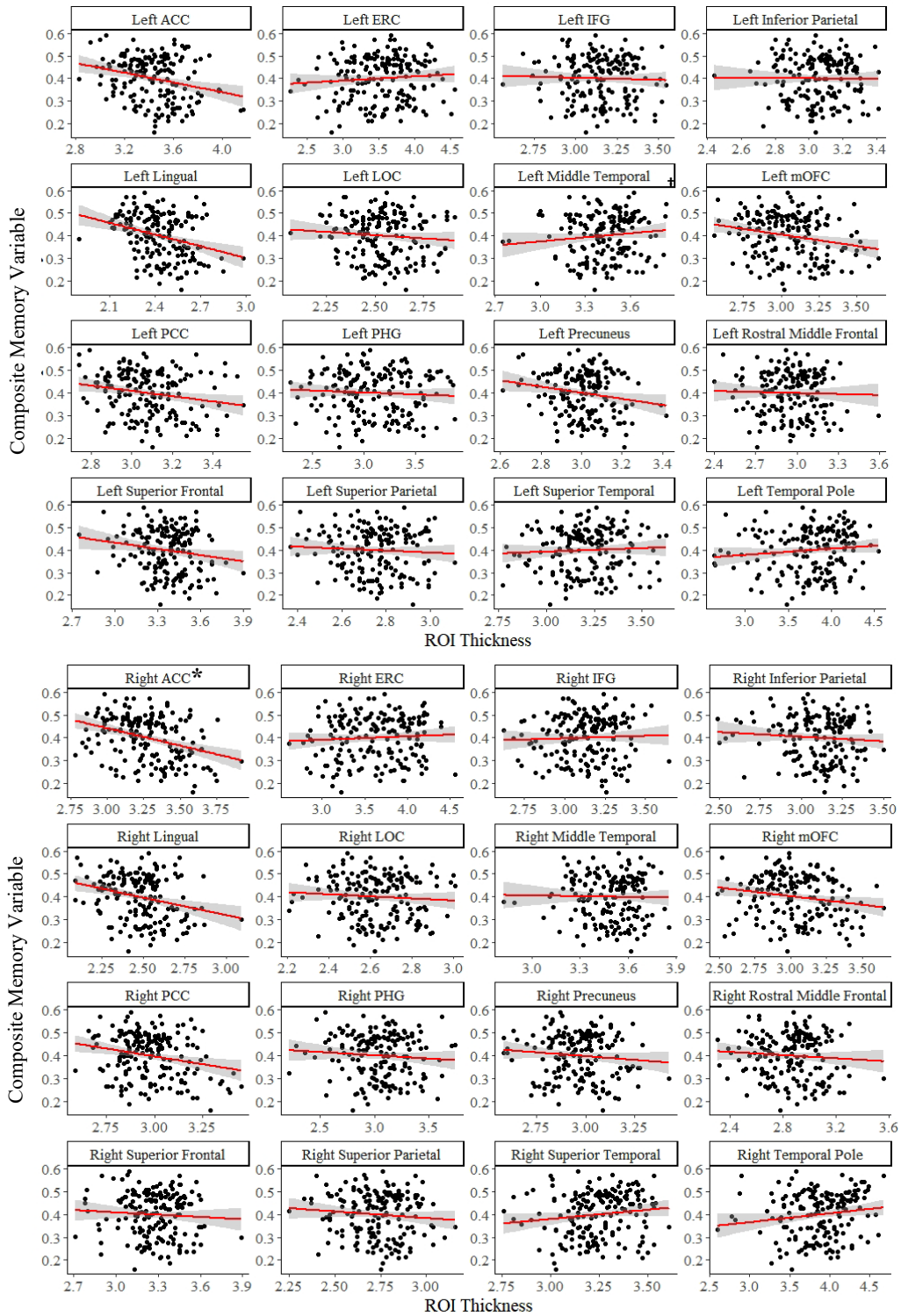
Cross-Sectional Associations Between Performance on the Composite Memory Variable and Selected ROIs

<i>ROI</i>	<i>Cortical Thickness</i>			<i>Surface Area</i>		
	<i>Beta</i>	<i>p</i>	<i>Adj. p</i>	<i>Beta</i>	<i>p</i>	<i>Adj. p</i>
<i>Right IFG</i>	0.044	0.128	0.671	5.71E-06	0.6157	0.863
<i>Left IFG</i>	0.015	0.6	0.863	1.29E-05	0.262	0.762
<i>Right mOFC</i>	0.024	0.315	0.801	3.58E-05	0.139	0.671
<i>Left mOFC</i>	-0.003	0.916	0.977	4.44E-05*	0.043	0.388
<i>Right Superior Frontal</i>	-2E-04	0.993	0.993	5.36E-06	0.412	0.824
<i>Left Superior Frontal</i>	0.012	0.683	0.878	5.63E-06	0.388	0.801
<i>Right Rostral Middle Frontal</i>	-8E-04	0.975	0.993	1.66E-05	0.0195	0.388
<i>Left Rostral Middle Frontal</i>	0.015	0.593	0.863	1.37E-05*	0.0495	0.396
<i>Right ACC</i>	-0.058*	0.02	0.388	2.46E-05	0.293	0.801
<i>Left ACC</i>	0.004	0.883	0.977	4.64E-05*	0.0364	0.388
<i>Right PCC</i>	-0.042	0.2052	0.742	2.83E-05	0.11	0.671
<i>Left PCC</i>	-0.021	0.513	0.863	2.18E-05	0.243	0.744
<i>Right Superior Parietal</i>	0.021	0.526	0.863	9.18E-06	0.22	0.742
<i>Left Superior Parietal</i>	0.004	0.903	0.977	1.63E-05*	0.0336	0.388
<i>Right Inferior Parietal</i>	0.009	0.747	0.917	6.73E-06	0.363	0.801
<i>Left Inferior Parietal</i>	0.022	0.48	0.863	8.61E-06	0.306	0.801
<i>Right Precuneus</i>	0.014	0.7	0.878	2.71E-05**	0.00347	0.222
<i>Left Precuneus</i>	0.013	0.759	0.917	2.31E-05*	0.0267	0.388
<i>Right ERC</i>	-0.001	0.9141	0.977	9.56E-05	0.1235	0.671
<i>Left ERC</i>	0.007	0.58	0.863	9.67E-05	0.2202	0.742
<i>Right PHG</i>	-0.008	0.687	0.878	2.63E-05	0.6209	0.863
<i>Left PHG</i>	0.022	0.1467	0.671	2.74E-05	0.489	0.863
<i>Right Temporal Pole</i>	0.008	0.595	0.863	4.55E-05	0.5855	0.863
<i>Left Temporal Pole</i>	0.006	0.666	0.878	7.99E-05	0.343	0.801
<i>Right Superior Temporal</i>	0.04	0.209	0.742	1.68E-05	0.244	0.744
<i>Left Superior Temporal</i>	0.029	0.348	0.801	1.71E-07	0.9897	0.993
<i>Right Middle Temporal</i>	0.028	0.371	0.801	1.04E-05	0.48	0.863
<i>Left Middle Temporal</i>	0.052	0.0657	0.467	1.09E-05	0.427	0.828
<i>Right Lingual Gyrus</i>	-0.021	0.513	0.863	-1.18E-05	0.37883	0.801
<i>Left Lingual Gyrus</i>	-0.043	0.185	0.742	-6.03E-07	0.9638	0.993
<i>Right LOC</i>	0.007	0.827	0.951	3.69E-06	0.6701	0.878
<i>Left LOC</i>	0.009	0.796	0.943	-1.92E-06	0.8321	0.951

Note. Significant parameters are bolded based on uncorrected associations. * $p < .05$. ** $p < .01$.

Figure 6

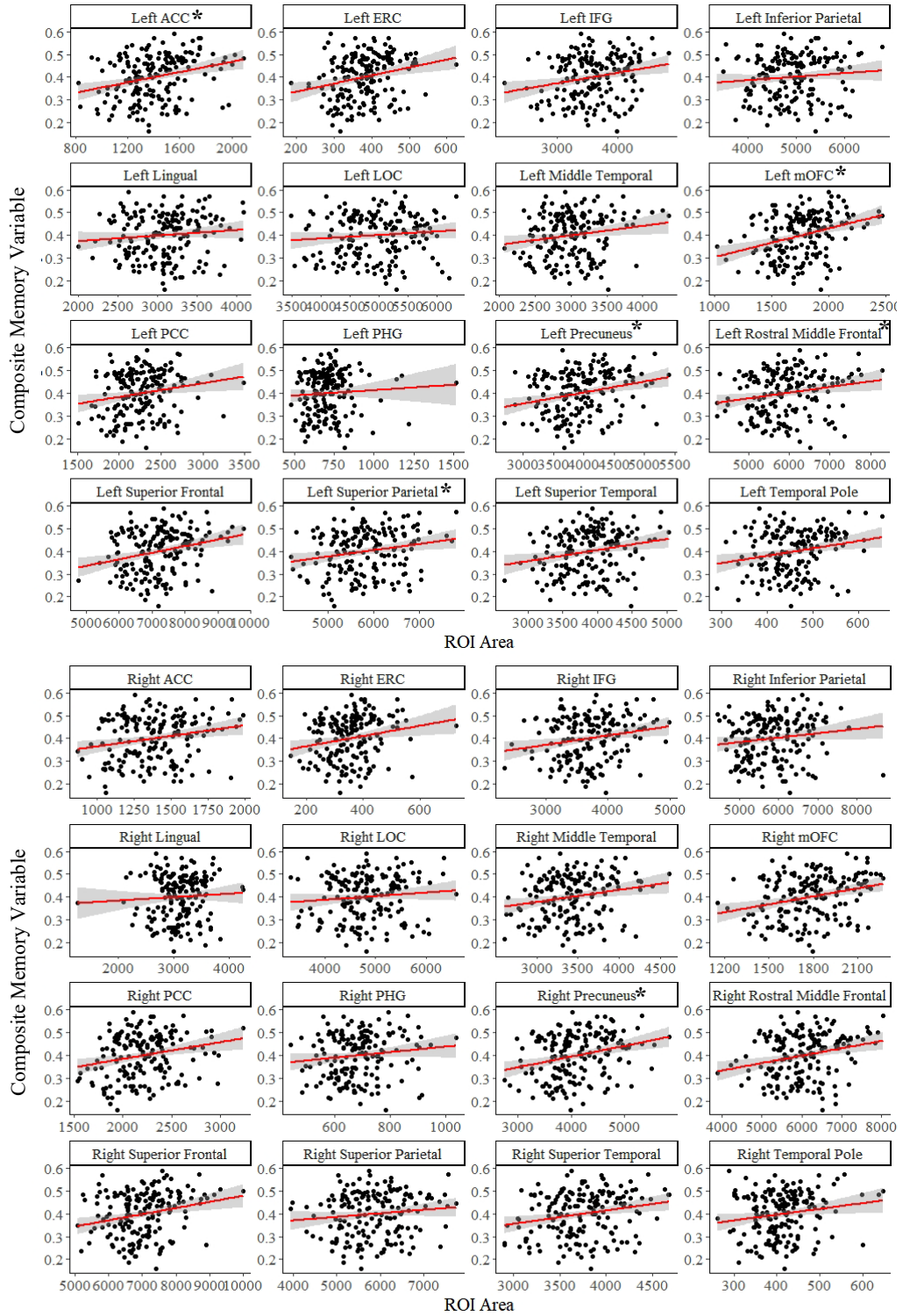
Associations Between Cortical Thickness and the Composite Memory Variable.



Note. * $p < .05$ uncorrected.

Figure 7

Associations Between Surface Area and the Composite Memory Variable.



Note. * $p < .05$ uncorrected.

Whole Brain Analysis.

To further estimate associations between episodic memory and measures of cortical maturation, a vertex-by-vertex analyses was conducted on both hemispheres controlling for age and sex. Following correction for multiple comparisons, there were no significant associations between cortical thickness and the composite memory variable. There were also no associations between the composite episodic memory variable and cortical surface area.

Aim 1 (Part 2): Memory Development

Fit indicators for second-order linear growth models in the younger (0.000 (0.000-0.059)) and the older (0.000 (0.000-0.039)) cohort were acceptable. Furthermore, mean slopes demonstrated significant positive change in performance from age 4 to 6 years and from age 6 to 8 years (Table 5). Importantly, there was no significant difference between the slope of the 4-year-old cohort and the 6-year-old cohort, $t(187) = 0.265, p = 0.40$. Together this suggests that latent episodic memory shows significant linear increases from age 4 to 8 years (Figure 8). These findings are consistent with Canada et al., (2022), which demonstrated the same pattern of results in this data set using piecewise models. As a result, these second-order linear growth curve models were used to assess the co-development of latent episodic memory and selected ROIs.

Table 6

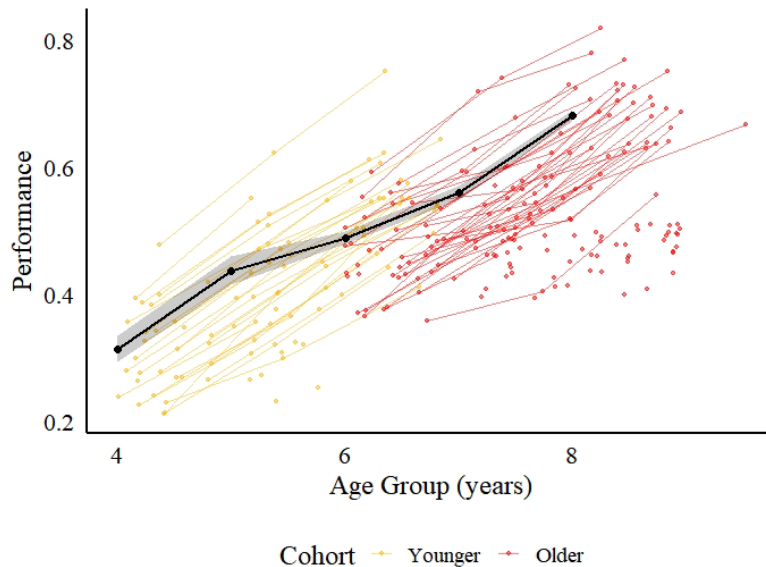
Growth Parameters for the Latent Episodic Memory Variable by Cohort.

Cohort	Intercept	Slope 4-6 years	Slope 6-8 years
4-year-old cohort	0.309 (0.019)***	0.113 (0.015)***	-
6-year-old cohort	0.462 (0.027)***	-	0.107 (0.017)***

Note. Significant parameters are bolded. *** $p < .001$.

Figure 8

Developmental Trajectory of Latent Episodic Memory Performance from 4 to 8 Years



Note. Longitudinal subjects are represented by connected dots with each dot representing a single timepoint. Dots that are not connected to other dots represent subjects assessed cross-sectionally. Participants depicted in gold were part of the younger cohort and participants depicted in red were part of the older cohort. The black line represents the mean developmental trajectory, and the dark shadow represents the 95% confidence interval.

Aim 1 (Part 2): Cortical Development

ROI Development Fit Indices.

Fit indices for most latent growth models examining the development of cortical thickness were “acceptable” (RMSEA < 0.08) or “satisfactory” (RMSEA between .08 and 0.10; see Table 7). However, a few regions demonstrated “poor: fit (i.e., right temporal pole in the younger cohort, along with left ACC and left lingual gyrus in the older cohort; RMSEA = .102 -

.134). Similarly, fit indicators for most latent growth models examining the development of cortical surface area were “acceptable” (RMSEA<0.08) or demonstrated “satisfactory” fit (RMSEA between .08 and 0.10; see Table 8). However, there were two exceptions that demonstrated “poor” fit (RMSEA >.10). These were left and right ACC in the younger cohort. Importantly, models with poor fit may suggest a mismatch between the observed data and the growth model (e.g., complex non-linearity within the cohort). Thus, findings from these models are reported for completeness but should be interpreted with caution.

ROI Intercept and Slope Variances.

Intercept variability was significant for all ROIs in both the younger and older cohorts suggesting that measures of each ROI demonstrate significant individual variability at both 4 and 6 years. In contrast, slopes for most models did not demonstrate significant variability. This suggests that children change at a similar rate from 4 to 6 years and from 6 to 8 years. However, there were some exceptions in the older cohort. In models of cortical thickness, right ACC, right superior temporal gyrus, and left ERC demonstrated significant slope variability ($p < .05$). In models of surface area, bilateral middle temporal gyrus, right IPS, and right precuneus demonstrated significant slope variability in the older cohort ($ps < .05$). These results suggest that there is significant individual variability in growth trajectories for these ROIs.

Cortical Thickness Development.

In the younger cohort, measures of cortical thickness for most regions demonstrated significant thinning from age 4 to 6 years (Figure 9). Only a handful of regions did not demonstrate significant change during this developmental period. These include bilateral ERC, bilateral temporal pole, left lateral occipital cortex, and left PHG (see Tables 9 – 10). Further,

only left mOFC demonstrated significant thickening. In the older cohort, most regions also demonstrated significant cortical thinning from age 6 to 8 years (Figure 9). The regions that did not demonstrate significant change during this developmental period were bilateral ERC, bilateral temporal pole, left mOFC, and left superior frontal gyrus. Additionally, no region in the older cohort demonstrated significant thickening. In sum, these findings suggest that early to middle childhood is marked by significant cortical thinning in the episodic memory network.

To assess whether change in cortical thickness was linear from 4 to 8 years, t-tests were conducted to examine if slopes were significantly different based on cohort. Results showed that most regions did not demonstrate significant differences, suggesting that thinning was linear from age 4 to 8 years (Tables 9-10). However, there were three exceptions. Specifically, right inferior parietal sulcus, right lingual gyrus, and right rostral middle frontal gyrus demonstrated significant differences in slopes based on cohort, indicating non-linear cortical thinning from 4 to 8 years. In both right lingual gyrus and right rostral middle frontal, there was significantly more thinning from 6 to 8 years than from 4 to 6 years implying an accelerating growth trajectory. In contrast, right inferior parietal sulcus demonstrated significantly more thinning from 4 to 6 years than from 6 to 8 years implying a decelerating growth trajectory (Table 10).

Table 7*Fit Indices for Cortical Thickness Growth Models*

Fit Indices for Thickness Development	Younger Cohort		Older Cohort	
	Left Hemisphere	Right Hemisphere	Left Hemisphere	Right Hemisphere
Inferior Frontal Gyrus	0.000 (0.000 - 0.155)	0.000 (0.000 - 0.000)	0.075 (0.000 - 0.199)	0.000 (0.000 - 0.161)
Posterior Cingulate Cortex	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.146)
Anterior Cingulate Cortex	0.000 (0.000 - 0.117)	0.098 (0.000 - 0.230)	<i>0.119 (0.000 - 0.232)</i>	0.059 (0.000 - 0.188)
Entorhinal Cortex	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.067 (0.000 - 0.193)	0.000 (0.000 - 0.164)
Inferior Parietal Sulcus	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)
Lateral Occipital Cortex	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.136)	0.000 (0.000 - 0.090)	0.000 (0.000 - 0.000)
Lingual Gyrus	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.160)	<i>0.139 (0.000 - 0.250)</i>	0.000 (0.000 - 0.164)
Medial Orbitofrontal	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.069)	0.000 (0.000 - 0.114)	0.000 (0.000 - 0.000)
Middle Temporal Gyrus	0.000 (0.000 - 0.197)	0.083 (0.000 - 0.220)	0.000 (0.000 - 0.124)	0.000 (0.000 - 0.112)
Parahippocampal Gyrus	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.055)	0.000 (0.000 - 0.157)	0.000 (0.000 - 0.059)
Precuneus	0.000 (0.000 - 0.093)	0.000 (0.000 - 0.114)	0.000 (0.000 - 0.096)	0.000 (0.000 - 0.135)
Rostral Middle Frontal	0.000 (0.000 - 0.119)	0.071 (0.000 - 0.211)	0.000 (0.000 - 0.141)	0.000 (0.000 - 0.152)
Superior Frontal Gyrus	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.108)	0.000 (0.000 - 0.150)
Superior Parietal Lobule	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.148)	0.000 (0.000 - 0.154)
Superior Temporal Gyrus	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.149)	0.000 (0.000 - 0.125)	0.000 (0.000 - 0.117)
Temporal Pole	0.000 (0.000 - 0.166)	<i>0.150 (0.051 - 0.256)</i>	0.000 (0.000 - 0.061)	0.000 (0.000 - 0.104)

Note. Models that demonstrate poor fit are italicized.

Table 8*Fit Indices for Surface Area Growth Models*

Fit Indices for Surface Area Development	Younger Cohort		Older Cohort	
	Left Hemisphere	Right Hemisphere	Left Hemisphere	Right Hemisphere
Inferior Frontal Gyrus	0.000 (0.000 - 0.184)	0.000 (0.000 - 0.108)	0.000 (0.000 - 0.136)	0.000 (0.000 - 0.148)
Posterior Cingulate Cortex	0.000 (0.000 - 0.137)	0.000 (0.000 - 0.142)	0.000 (0.000 - 0.144)	0.000 (0.087 - 0.207)
Anterior Cingulate Cortex	<i>0.146 (0.000 - 0.252)</i>	<i>0.106 (0.000 - 0.236)</i>	0.000 (0.000 - 0.122)	0.000 (0.000 - 0.000)
Entorhinal Cortex	0.000 (0.000 - 0.279)	0.000 (0.000 - 0.118)	0.000 (0.000 - 0.107)	0.000 (0.000 - 0.079)
Inferior Parietal Sulcus	0.056 (0.000 - 0.202)	0.000 (0.000 - 0.126)	0.000 (0.000 - 0.120)	0.085 (0.000 - 0.267)
Lateral Occipital Cortex	0.000 (0.000 - 0.150)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.124)	0.000 (0.000 - 0.000)
Lingual Gyrus	0.000 (0.000 - 0.101)	0.000 (0.000 - 0.166)	0.000 (0.000 - 0.129)	0.063 (0.000 - 0.174)
Medial Orbitofrontal	0.000 (0.000 - 0.132)	0.000 (0.000 - 0.050)	0.000 (0.000 - 0.123)	0.000 (0.000 - 0.000)
Middle Temporal Gyrus	0.000 (0.000 - 0.154)	0.064 (0.000 - 0.207)	0.000 (0.000 - 0.160)	0.000 (0.000 - 0.119)
Parahippocampal Gyrus	0.000 (0.000 - 0.176)	0.000 (0.000 - 0.106)	0.065 (0.000 - 0.192)	0.052 (0.000 - 0.184)
Precuneus	0.029 (0.000 - 0.223)	0.000 (0.000 - 0.166)	0.000 (0.000 - 0.165)	0.062 (0.000 - 0.190)
Rostral Middle Frontal	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.165)	0.085 (0.000 - 0.206)	0.000 (0.000 - 0.070)
Superior Frontal Gyrus	0.000 (0.000 - 0.186)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.136)	0.000 (0.000 - 0.141)
Superior Parietal Lobule	0.068 (0.000 - 0.191)	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.148)	0.000 (0.000 - 0.073)
Superior Temporal Gyrus	0.000 (0.000 - 0.171)	0.000 (0.000 - 0.117)	0.000 (0.000 - 0.225)	0.074 (0.000 - 0.198)
Temporal Pole	0.000 (0.000 - 0.000)	0.000 (0.000 - 0.159)	0.000 (0.000 - 0.130)	0.000 (0.000 - 0.114)

Note. Models that only demonstrated poor fit are italicized.

Table 9*Growth Parameters for Cortical Thickness in The Left Hemisphere*

Growth Parameters for Left Hemisphere Thickness	Younger Cohort			Older Cohort			Difference of slopes between cohorts
	Freely Estimated Loading	Latent Estimates		Freely Estimated Loading	Latent Estimates		
		Slope	Intercept		Slope	Intercept	
Inferior Frontal Gyrus	0.613	-0.023**	3.133***	0.701	-0.020*	3.149***	-0.282
Posterior Cingulate Cortex	0.047	-0.013***	2.959***	0.768	-0.013*	2.967***	0.000
Anterior Cingulate Cortex	0.321	-0.029**	3.421***	0.328	-0.023***	3.342***	-0.832
Entorhinal Cortex	-3.310	0.009	3.537***	1.832	-0.004	3.559***	0.933
Inferior Parietal Sulcus	-0.434	-0.012*	2.985***	0.412	-0.014*	3.024***	0.256
Lateral Occipital Cortex	0.664	-0.016	2.537***	1.318	-0.019***	2.524***	1.100
Lingual Gyrus	1.155	-0.016**	2.484***	1.426	-0.017*	2.395***	0.128
Medial Orbitofrontal	6.225	0.009**	2.944***	2.841	0.009	2.928***	0.000
Middle Temporal Gyrus	0.731	-0.032***	3.377***	1.042	-0.028***	3.427***	-0.465
Parahippocampal Gyrus	3.073	-0.001	3.095***	2.284	-0.014*	3.103***	1.511
Precuneus	0.410	-0.019***	2.985***	1.205	-0.013*	2.969***	-0.849
Rostral Middle Frontal	1.100	-0.041***	2.968***	0.862	-0.036***	2.973***	-0.505
Superior Frontal Gyrus	0.523	-0.020**	3.346***	1.161	-0.006	3.322***	-1.414
Superior Parietal Lobule	0.674	-0.020**	2.739***	0.059	-0.016*	2.736***	-0.400
Superior Temporal Gyrus	1.006	-0.013*	3.209***	1.022	-0.019**	3.231***	0.768
Temporal Pole	16.101	-0.003	3.768***	2.585	-0.008	3.886***	0.411

Note. Significant parameters are bolded. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 10*Growth Parameters for Cortical Thickness in The Right Hemisphere*

Growth Parameters for Right Hemisphere Thickness	Younger Cohort			Older Cohort			Difference of slopes between cohorts
	Freely Estimated Loading	Latent Estimates		Freely Estimated Loading	Latent Estimates		
		Slope	Intercept		Slope	Intercept	
Inferior Frontal Gyrus	1.189	-0.024**	3.119***	1.461	-0.017**	3.124***	-0.707
Posterior Cingulate Cortex	0.284	-0.024***	2.881***	0.893	-0.012***	2.884***	-1.536
Anterior Cingulate Cortex	0.700	-0.030***	3.303***	1.464	-0.021**	3.194***	-0.976
Entorhinal Cortex	-0.370	0.018	3.631***	1.065	0.005	3.625***	0.707
Inferior Parietal Sulcus	-1.490	-0.007*	3.631***	1.187	-0.029***	3.060***	3.051**
Lateral Occipital Cortex	0.394	-0.014*	2.613***	0.913	-0.022***	2.591***	0.753
Lingual Gyrus	1.211	-0.019***	2.522***	3.657	-0.009**	2.439***	-2.357**
Medial Orbitofrontal	1.329	-0.023**	3.051***	1.354	-0.019*	2.951***	-0.351
Middle Temporal Gyrus	0.919	-0.031***	3.437***	1.175	-0.036***	3.447***	0.581
Parahippocampal Gyrus	1.899	-0.017**	3.053***	1.875	-0.024**	3.013***	0.700
Precuneus	0.782	-0.019**	2.962***	0.784	-0.019***	2.928***	0.000
Rostral Middle Frontal	0.741	-0.052***	2.911***	0.520	-0.025***	2.862***	-2.108*
Superior Frontal Gyrus	0.675	-0.024***	3.272***	1.410	-0.018**	3.264***	-0.651
Superior Parietal Lobule	1.145	-0.020**	2.739***	0.924	-0.024***	2.706***	0.434
Superior Temporal Gyrus	1.949	-0.010*	3.229***	1.145	-0.019***	3.261***	1.046
Temporal Pole	1.927	0.002	3.860***	5.281	0.001	3.941***	0.102

Note. Significant parameters are bolded. * $p < .05$. ** $p < .01$. *** $p < .001$.

Surface Area Development.

In the younger cohort, measures of surface area for most regions demonstrated significant increases from age 4 to 6 years (Figure 13). However, left LOC and bilateral mOFC did not demonstrate any significant change during this developmental period (Tables 11-12). Additionally, right LOC demonstrated significant decreases in cortical surface area (Table 12). In the older cohort, findings were more variable (Tables 11-12). Whereas many regions demonstrated significant increases from age 6 to 8 years (i.e., bilateral IFG, bilateral ERC, bilateral middle temporal gyrus, bilateral superior frontal gyrus, bilateral temporal pole, left ACC, left superior temporal pole, and right rostral middle frontal gyrus), several others demonstrated significant decreases (i.e., bilateral IPS, bilateral lingual gyrus, bilateral precuneus, bilateral SPL, left PCC, right LOC, right mOFC) from age 6 to 8 years. Additionally, some demonstrated no significant change in cortical surface area from age 6 to 8 years (i.e., bilateral PHG, left LOC, left, mOFC, left rostral middle frontal gyrus, right ACC, right PCC, and right superior temporal gyrus). Together, these findings may suggest that some regions demonstrate increases in surface area from 4 to 8 years while other regions may peak between ages 6 and 8 years and begin to demonstrate decreases in surface area.

To assess the developmental trajectory of surface area from age 4 to 8 years, t-tests were conducted to examine if slopes were significantly different based on cohort. Results demonstrated that most regions show significant differences in slope between the younger and older cohort, suggesting that surface area exhibits non-linear change from age 4 to 8 years. Specifically, most regions showed greater increases in surface area from 4 to 6 years than from 6 to 8 years (i.e., bilateral IFG, bilateral middle temporal gyrus, bilateral superior frontal gyrus, bilateral superior temporal gyrus, left ACC, right PHG, right rostral middle frontal gyrus, and

right temporal pole) suggesting a decelerating growth trajectory. In contrast, only right ERC demonstrated an accelerating trajectory from 4 to 8 years. Another subset of regions demonstrated an inverted U-shape trajectory (i.e., bilateral IPS, bilateral lingual gyrus, bilateral precuneus, bilateral SPL, left PCC, left rostral middle frontal gyrus). In other words, these regions increased from 4 to 6 years and then decreased from 6 to 8 years. These findings could suggest that surface area of these regions peaks between ages 4 and 8 years. Finally, there were two additional regions that showed non-linear change that did not fit any of these trajectories. For example, right ACC exhibited significant increases in surface area from 4 to 6 years, but no change from 6 to 8 years. In contrast, right mOFC demonstrated no significant change from 4 to 6 years, then significant decreases from 4 to 8 years.

Importantly, there were a handful of regions that did not demonstrate non-linear change in surface area from 4 to 8 years. For example, left ERC and left temporal pole demonstrated linear increases in surface area from 4 to 8 years, whereas right LOC demonstrated linear decreases from 4 to 8 years. Additionally, left LOC and left mOFC demonstrated no significant change from 4 to 8 years. In sum, these findings demonstrate that growth trajectories of cortical surface area in the episodic memory network vary from 4 to 8 years, though most are non-linear.

Table 11*Growth Parameters for Surface Area in The Left Hemisphere.*

Growth Parameters for Left Hemisphere Surface Area	Younger Cohort			Older Cohort			Difference of slopes between cohorts
	Freely Estimated Loading	Latent Estimates		Freely Estimated Loading	Latent Estimates		
		Slope	Intercept		Slope	Intercept	
Inferior Frontal Gyrus	1.042	68.119***	3581.605***	1.42	38.908***	3696.515***	4.131***
Posterior Cingulate Cortex	1.597	15.979***	2229.527***	-4.6	-2.449**	2325.082***	5.380***
Anterior Cingulate Cortex	0.99	18.680***	1345.165***	2.454	9.092***	1446.390***	2.127*
Entorhinal Cortex	1.941	6.365*	403.262***	1.481	10.384***	399.272***	-1.017
Inferior Parietal Sulcus	1.05	36.777***	4942.362***	-1.532	-11.624*	4962.892***	4.369***
Lateral Occipital Cortex	113.641	0.019	5198.686***	-2.367	-8.324	5162.537***	1.372
Lingual Gyrus	2.009	17.268*	2978.648***	-2.658	-7.397**	3123.611***	3.134**
Medial Orbitofrontal	7.787	-0.785	1775.404***	7.8	1.010	1853.949***	-0.535
Middle Temporal Gyrus	1.125	59.566***	3068.680***	1.275	44.936***	3114.760***	1.818*
Parahippocampal Gyrus	1.69	5.356***	649.460***	1.577	2.177	680.944***	1.353
Precuneus	1.31	29.978***	3908.901***	-2.818	-4.809**	4089.497***	8.302***
Rostral Middle Frontal	0.717	20.776	5908.884***	1.367	-4.265	6143.481***	1.886*
Superior Frontal Gyrus	1.087	134.914***	7068.399***	1.536	84.235***	7421.281***	3.527***
Superior Parietal Lobule	4.528	12.916***	5800.419***	-0.9	-13.772*	5938.262***	3.728***
Superior Temporal Gyrus	1.57	42.637***	3849.018***	1.196	22.426***	4029.104***	2.708**
Temporal Pole	0.405	11.086***	437.958***	1.19	6.321**	462.863***	1.227

Note. Significant parameters are bolded. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 12*Growth Parameters for Surface Area In The Right Hemisphere*

Growth Parameters for Right Hemisphere Surface Area	Younger Cohort			Older Cohort			Difference of slopes between cohorts
	Freely Estimated Loading	Latent Estimates		Freely Estimated Loading	Latent Estimates		
		Slope	Intercept		Slope	Intercept	
Inferior Frontal Gyrus	0.93	73.511***	3649.794***	1.638	27.883***	3835.147***	6.174***
Posterior Cingulate Cortex	1.042	15.770***	2150.285***	-2.748	-1.428	2280.881***	3.737***
Anterior Cingulate Cortex	1.034	11.047*	1279.461***	49.154	0.058	1402.994***	2.535**
Entorhinal Cortex	4.634	1.187	371.514***	1.833	6.558**	376.109***	-2.574**
Inferior Parietal Sulcus	1.275	49.768**	5755.899***	0.007	-385.61***	5835.976***	4.422***
Lateral Occipital Cortex	0.165	-19.360*	5124.716***	1.357	-32.061***	5100.848***	0.985
Lingual Gyrus	1.888	17.612**	3092.706***	-1.32	-9.035***	3165.480***	4.527***
Medial Orbitofrontal	37.975	-0.306	1843.572***	-0.215	-11.948*	1910.690***	1.990*
Middle Temporal Gyrus	1.12	60.667***	3418.581***	1.459	25.769***	3542.801***	6.328***
Parahippocampal Gyrus	0.937	4.645***	621.839***	17.682	0.927	676.320***	2.734**
Precuneus	1.342	30.388***	4066.537***	-78.266	-0.170*	4263.667***	8.721***
Rostral Middle Frontal	0.84	47.753***	5889.030***	2.569	26.264***	6235.729***	1.725*
Superior Frontal Gyrus	1.068	142.576***	6913.457***	1.82	79.581***	7236.441***	4.754***
Superior Parietal Lobule	6.31	10.502**	5782.840***	-0.42	-21.489**	5888.787***	4.124***
Superior Temporal Gyrus	1.55	21.041***	3717.089***	1.92	2.964	3859.218***	2.324*
Temporal Pole	0.793	13.111***	420.954***	1.52	7.335***	424.062***	1.895*

Note. Significant parameters are bolded. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 9

Development of Cortical Thickness and Surface Area by Hemisphere.

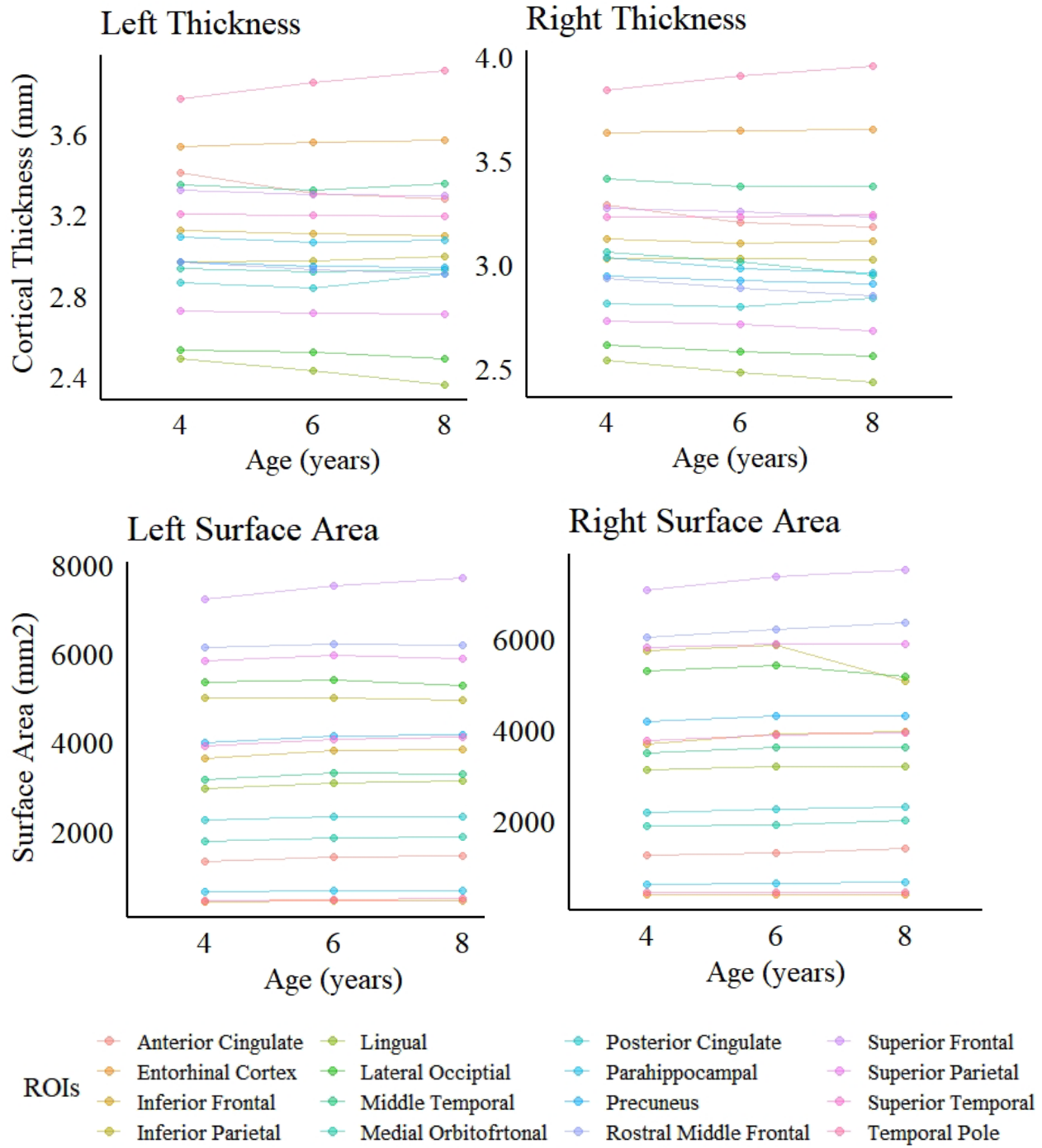
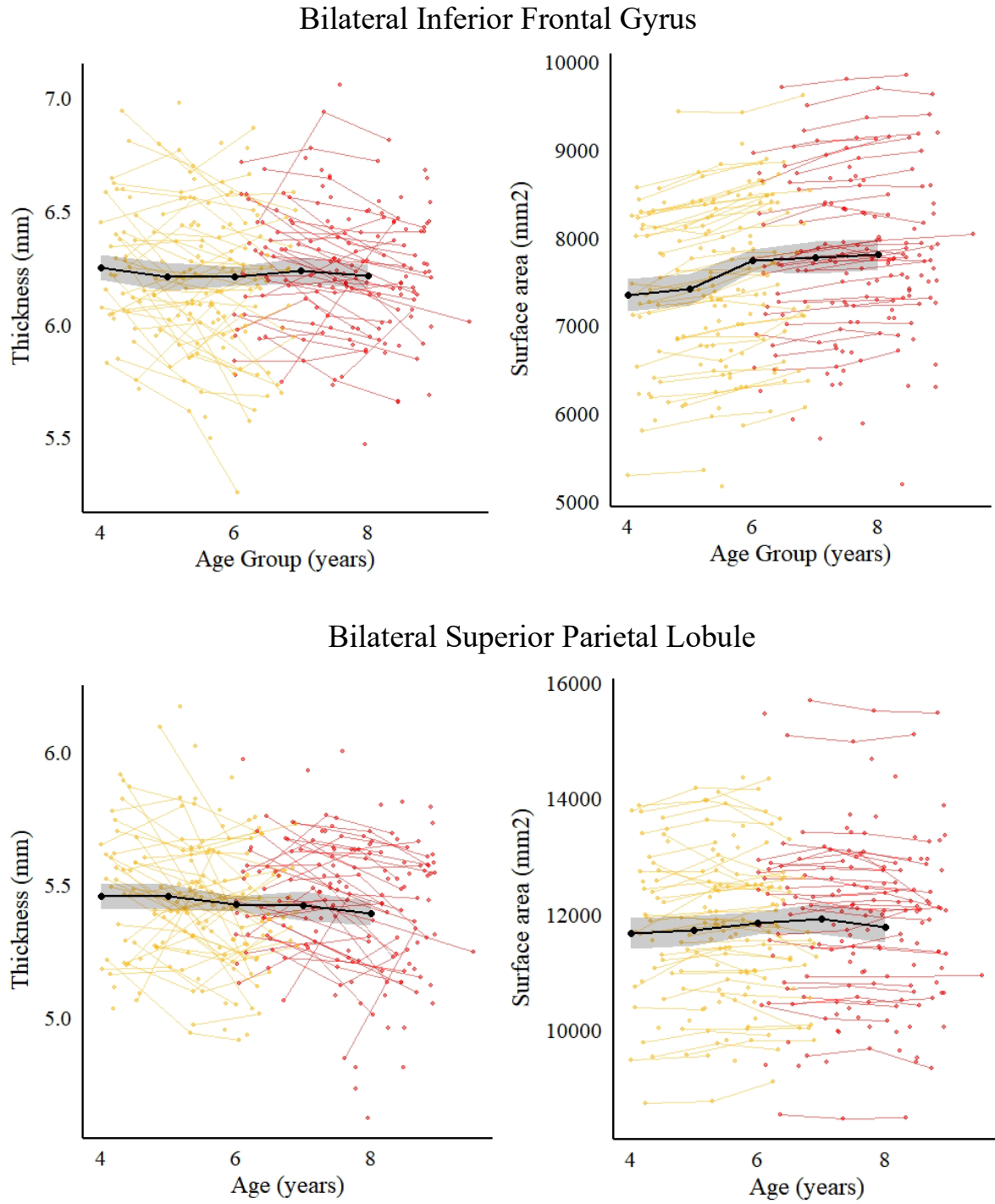


Figure 10

Examples of Developmental Trajectories by Subject in Two Selected Cortical Regions



Note. Longitudinal subjects are represented by connected dots with each dot representing a single timepoint. Dots that are not connected to other dots represent subjects assessed cross-sectionally. Participants depicted in gold were part of the younger cohort and participants depicted in red were part of the older cohort. The black line represents the mean developmental trajectory, and the dark shadow represents the 95% confidence interval.

Hemispheric Differences in Cortical Development.

To assess whether cortical development was lateralized, t-tests were conducted to determine if slopes were significantly different based on hemisphere. For cortical thickness, results showed that most regions did not demonstrate hemispheric differences in cortical thinning for either cohort. Regions that did demonstrate differences were mOFC and PHG in the younger cohort, along with mOFC and IPS in the older cohort (see Appendix A). Specifically, PHG in the younger cohort did not demonstrate a significant change in the left hemisphere. However, the right hemisphere showed significant thinning during this same developmental period. In contrast, mOFC in the younger cohort demonstrated a completely different pattern of results. Namely, the right hemisphere demonstrated significant thinning, while the left hemisphere demonstrated significant thickening. In the older cohort, right hemisphere IPS and mOFC demonstrated significantly greater change in cortical thickness than left hemisphere.

For surface area, results showed that most regions did not demonstrate hemispheric differences in development from 4 to 6 years. Four regions that did demonstrate differences were ACC, ERC, rostral middle frontal gyrus, and superior temporal gyrus. Specifically, ACC, ERC, and superior temporal gyrus exhibited faster growth in the left hemisphere, while rostral middle frontal exhibited faster growth in the right hemisphere.

In comparison, half of all regions demonstrated hemispheric differences in development from age 6 to 8 years. Specifically, IFG, PCC, ERC, lingual gyrus, PHG superior frontal gyrus, SPL, and temporal pole showed similar developmental slopes across hemispheres (see Appendix A), whereas ACC, IPS, LOC, mOFC, middle temporal gyrus, precuneus, rostral middle frontal, and superior temporal pole demonstrated lateralized development. However, the way in which hemispheres differed varied greatly from region to region. For example, ACC, middle temporal gyrus, and superior temporal gyrus demonstrated significantly faster growth in the left hemisphere. Additionally, precuneus demonstrated significantly faster decreases in the left hemisphere, whereas IPS and LOC demonstrated faster decreases in the right hemisphere. Finally, two regions demonstrated hemispheric change in opposing directions. Namely, mOFC, demonstrated increases in left hemisphere but decreases in right hemisphere and rostral middle frontal demonstrated decreases in right hemisphere but increases in left hemisphere.

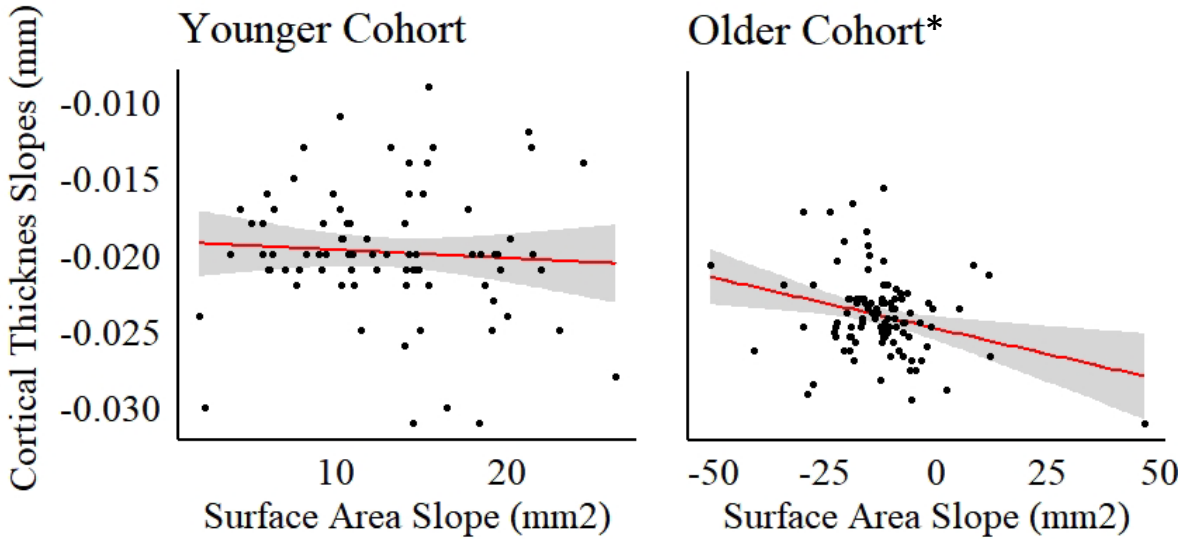
Associations between development of cortical thickness and surface area.

To assess whether developmental trajectories of cortical thickness and surface area were similar within the same ROI, two representative regions were utilized, one each from the most common developmental trajectories of cortical surface area (i.e., the decelerating growth trajectory and the inverted U trajectory). These regions were left superior temporal gyrus (decelerating) and left SPL (inverted U). Results demonstrated that there were no associations between cortical surface area and cortical thickness in the younger cohort for either region ($ps > 0.54$). However, in the older cohort, there was a negative association between the cortical thickness and surface area of both SPL $F(1,97) = 8.186, p < .005$, and superior temporal gyrus $F(1,96) = 10.48, p < .002$, such that decreases in thickness was associated with increases in surface area (Figure 10).

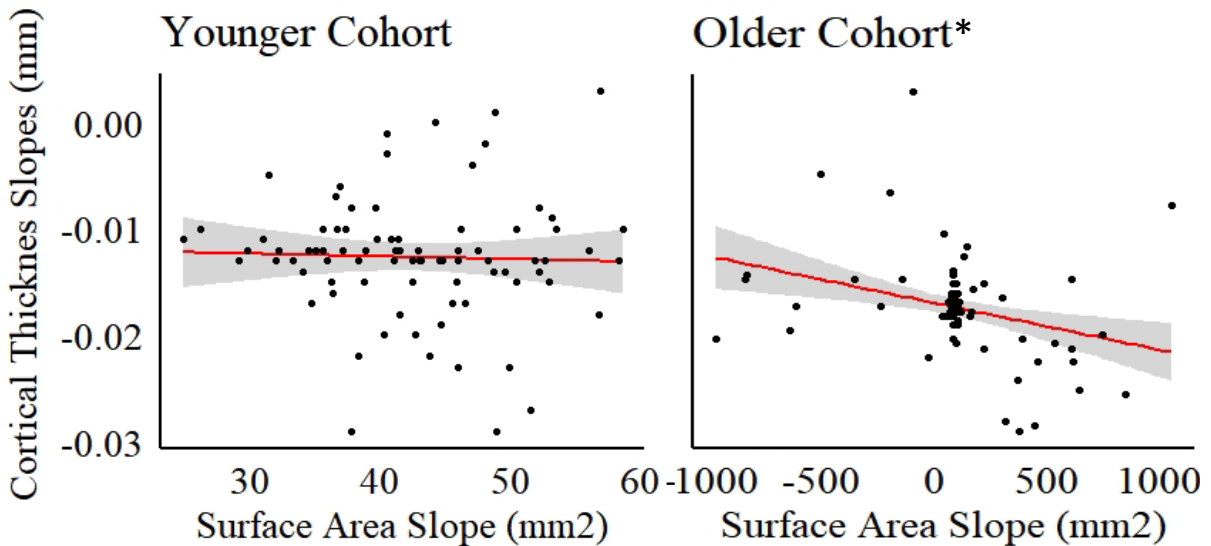
Figure 11

Associations Between the Slopes of Cortical Thickness and Surface Area

Superior Parietal Lobule (Inverted U Trajectory)



Superior Temporal Gyrus (Decelerating Trajectory)



Note. * $p < .05$.

Total Gray Matter Volume Development.

Fit indicators for latent growth models examining the development of total gray matter volume were acceptable in the younger (RMSEA = 0.000 (0.000 - 0.156)), but poor in the older (RMSEA = 0.126 (0.031 - 0.223)) cohort. Similar to other models with poor fit, findings from the older cohort are reported for completeness but should be interpreted with caution. Furthermore, intercept variability was significant for both the younger and older cohorts ($p < .009$) suggesting that total gray matter volume demonstrates significant individual variability at both 4 and 6 years. However, slope variability was not significant ($p > .346$) indicating that children demonstrate similar developmental trajectories.

Latent means demonstrated that there was significant growth in total gray matter volume for the younger ($M = 160.989$, $p < .001$) but not the older ($M = 34.833$, $p = .08$) cohort, meaning, on average, total gray matter volume increases from age 4 to 6 years then does not demonstrate significant change from 6 to 8 years. To confirm that change in total gray matter volume was linear from 4 to 8 years, t-tests were conducted to examine if slopes were significantly different based on cohort. Results showed that there were significant differences, $t(145) = 3.883$, $p < .001$. Specifically, there was more growth in total gray matter volume from 4 to 6 years than from 6 to 8 years indicating a decelerating trajectory.

Aim 1 (Part 3): Co-development of Memory and the Cortex

After characterizing ROI development, co-development with the latent episodic memory variable was assessed. Fit indices for all co-development models examining both cortical thickness and surface area were acceptable (see Tables 13, 18, 19). Importantly, each development model (i.e., the ROI model and the memory model) included two key growth

parameters (i.e., the intercept and the slope). Specifically, the intercept represents the starting point for each variable (e.g., thickness at age 4) and the slope represents change in the variable across the following two years (e.g., thinning from age 4 to age 6). Thus, each co-development model yielded 4 regressions between these growth parameters. These regressions include associations between both intercepts, associations between the ROI intercept and the latent episodic memory slope, associations between the latent episodic memory intercept and the ROI slope, and associations between both slopes. Importantly, none of the findings from Aim 1 Part 3 survived correction for multiple correction. Thus, reported findings should be interpreted with caution.

Associations between the ROI Intercept and the Memory Intercept.

Relations between intercepts in these bivariate growth curve models indicate an association between the ROI and the latent episodic memory at age 4 in the younger cohort and age 6 in the older cohort. For cortical thickness, results revealed a negative association of bilateral ACC intercept (Table 14) and left ACC intercept (Table 20) with the latent episodic memory intercept in the younger cohort. This suggests that a thinner ACC at age 4 is associated with better memory performance. In contrast, the intercept of left mOFC was negatively associated with the latent episodic memory intercept in the older cohort. This suggests that a thicker mOFC is associated with better memory performance at age 6.

For surface area, results revealed a positive association between the right medial orbitofrontal gyrus and the latent episodic memory intercept in the younger cohort (Table 26). This suggests that greater surface area in the middle temporal gyrus at age 4 is associated with better memory performance. There were no other associations between intercepts. Together these

findings demonstrate that the direction of cross-sectional associations is highly dependent on the region, cortical measure, and age group being examined.

Table 13

Fit Indices for Bivariate Growth Curve Models Examining Associations Between ROI Thickness and the Latent Episodic Memory Variable.

Fit Indices for Bilateral Co-development Models	Cortical Thickness		Cortical Surface Area	
	Younger Cohort RMSEA (90% CI)	Older Cohort RMSEA (90% CI)	Younger Cohort RMSEA (90% CI)	Older Cohort RMSEA (90% CI)
Inferior Frontal Gyrus	0.010 (0.000 - 0.056)	0.047 (0.000 - 0.074)	0.040 (0.000 - 0.071)	0.008 (0.000 - 0.054)
Posterior Cingulate Cortex	0.000 (0.000 - 0.050)	0.031 (0.000 - 0.063)	0.031 (0.000 - 0.065)	0.022 (0.000 - 0.058)
Anterior Cingulate Cortex	0.027 (0.000 - 0.063)	0.000 (0.000 - 0.047)	0.049 (0.000 - 0.078)	--
Entorhinal Cortex	0.011 (0.000 - 0.057)	0.043 (0.000 - 0.071)	--	0.000 (0.000 - 0.047)
Inferior Parietal Sulcus	0.007 (0.000 - 0.056)	0.000 (0.000 - 0.052)	0.017 (0.000 - 0.059)	--
Lateral Occipital Cortex	0.040 (0.000 - 0.071)	0.030 (0.000 - 0.063)	--	--
Lingual Gyrus	0.049 (0.000 - 0.077)	0.052 (0.000 - 0.078)	0.055 (0.015 - 0.082)	0.035 (0.000 - 0.066)
Medial Orbitofrontal	--	0.056 (0.000 - 0.082)	0.021 (0.000 - 0.060)	--
Middle Temporal Gyrus	0.000 (0.000 - 0.033)	0.058 (0.000 - 0.083)	0.036 (0.000 - 0.069)	--
Parahippocampal Gyrus	--	0.051 (0.000 - 0.077)	0.000 (0.000 - 0.046)	0.065 (0.038 - 0.089)
Precuneus	0.000 (0.000 - 0.050)	0.042 (0.000 - 0.070)	0.054 (0.012 - 0.081)	--
Rostral Middle Frontal	0.043 (0.000 - 0.073)	0.017 (0.000 - 0.056)	--	--
Superior Frontal Gyrus	0.017 (0.000 - 0.059)	0.039 (0.000 - 0.068)	0.043 (0.000 - 0.073)	0.023 (0.000 - 0.058)
Superior Parietal Lobule	0.015 (0.000 - 0.058)	0.041 (0.000 - 0.070)	0.000 (0.000 - 0.051)	0.000 (0.000 - 0.052)
Superior Temporal Gyrus	0.045 (0.000 - 0.074)	0.038 (0.000 - 0.068)	--	--
Temporal Pole	0.033 (0.000 - 0.066)	--	0.036 (0.000 - 0.069)	0.000 (0.000 - 0.043)

Note. Dashes represent regions that show developmental lateralization.

Associations between the ROI Intercept and the Slope of Memory Development.

In the younger cohort, relations between the ROI intercept and the latent episodic memory slope would indicate an association between the ROI at age 4 and change in memory performance from 4 to 6 years. In the older cohort, this association would indicate a relation between the ROI at age 6 and change in memory performance from 6 to 8 years.

For cortical thickness, there were negative associations of six bilateral ROIs (i.e., IFG, LOC, lingual gyrus, middle temporal gyrus, precuneus, SPL; Table 14), nine left lateralized ROIs (i.e., IFG, IPL, LOC, lingual gyrus, mOFC, precuneus, SPL, superior temporal gyrus, and

temporal pole; Table 20), and five right lateralized ROIs (IFG, middle temporal, precuneus, superior frontal gyrus, SPL; Table 22) at age 4 with the latent episodic memory slope from age 4 to 6 years. In other words, a thinner cortex in these regions at age 4 predicted more rapid improvements in latent episodic memory performance from age 4 to 6 years. In contrast, there was a positive association of bilateral middle temporal gyrus, right IPL, and right middle temporal gyrus at age 6 with the latent episodic memory slope from age 6 to 8 years. Suggesting that a thicker cortex in these regions at age 6 was associated with more rapid improvements in latent episodic memory performance from age 6 to 8 years.

For surface area, there were positive associations of right-lateralized precuneus with the latent episodic memory slope in the younger cohort. Additionally, there was a negative association between right PHG and the latent episodic memory slope in the younger cohort. This suggests that greater surface area in the precuneus and less surface area in the PHG at age 4 was associated with more rapid improvements in latent episodic memory performance from age 4 to 6 years. There were also no associations in the older cohort of surface area ROI intercepts with the latent episodic memory slope. Thus, associations between ROI intercepts and the latent episodic memory slope may be specific to the younger cohort.

Associations between the Memory Intercept and the Slope of ROI Development.

In the younger cohort, relations between the latent episodic memory intercept and the ROI slope would indicate an association between latent episodic memory performance at age 4 and development of the ROI from 4 to 6 years. In the older cohort, this association would indicate an association between latent episodic memory performance at age 6 and development of the ROI from 6 to 8 years.

For cortical thickness, latent episodic memory performance at age 4 was positively associated with the slope of two right lateralized ROIs (mOFC and rostral middle frontal gyrus; Table 22) from age 4 to 6 years. In contrast, latent episodic memory performance at age 6 was negatively associated with the slope of the bilateral ERC from age 6 to 8 years. There were no associations with surface area. Together this could suggest that better latent episodic memory performance at age 4 predicts less cortical thinning in rostral middle frontal gyrus and mOFC from age 4 to 6 years. Additionally, better performance on the latent episodic memory variable at age 6 may predict more cortical thinning in the ERC from age 6 to 8 years.

Associations between the Slope of ROI Development and the Slope of Memory Development.

Relations between slopes indicate an association between the rate of change in the ROI and the rate of change in the latent episodic memory variable from age 4 to 6 in the younger cohort and age 6 to 8 in the older cohort.

For cortical thickness, there was a negative association between the slopes of right ERC and the latent episodic memory variable in the younger cohort, such that more thinning predicted more rapid improvements in memory performance from 6 to 8 years (Table 22). However, in the older cohort, there was a positive association between the slopes of bilateral ERC and left ERC with the latent episodic memory variable, such that increases in ERC thickness predicted more rapid improvements in memory performance from 6 to 8 years (Table 15 and Table 21).

For surface area, findings demonstrated there was a positive association between the slopes of bilateral lingual gyrus, left ACC, left IPS, and right lingual gyrus with the latent episodic memory variable in the older cohort, such that greater increases in these predicted more rapid improvements in memory performance from 6 to 8 years (Table 25). However, there were

also several ROIs (i.e., bilateral ERC, left ERC, and left superior temporal gyrus; Tables 25 and 17) that demonstrated a negative association with the latent episodic memory variable, such that decreases in surface area are associated with more rapid improvements in memory performance from 6 to 8 years. Importantly, there were no associations between surface area development and memory development in the younger cohort.

Table 14*Associations Between Memory and Bilateral ROI Thickness Parameters in the Younger Cohort*

Bilateral Thickness in the Younger Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>p</i>
Inferior Frontal Gyrus	0.001	0.973	-0.005*	0.554	0.001	0.389	0	0.987
Posterior Cingulate Cortex	-0.001	0.973	-0.002	0.711	0	0.69	0	0.987
Anterior Cingulate Cortex	-0.005*	0.448	-0.002	0.395	0.001	0.138	0	0.987
Entorhinal Cortex	-0.006	0.973	-0.002	0.395	0.002	0.272	-0.001	0.987
Inferior Parietal Sulcus	0	0.973	-0.003	0.554	0	0.183	0	0.987
Lateral Occipital Cortex	-0.001	0.973	-0.003*	0.395	0.001	0.036	0	0.987
Lingual Gyrus	-0.001	0.973	-0.005*	0.71	0	0.472	0	0.987
Middle Temporal Gyrus	0	0.919	-0.003*	0.554	0.001	0.369	0	0.987
Precuneus	0	0.819	-0.004**	0.084	0.001*	0.514	0	0.987
Rostral Middle Frontal	-0.001	0.65	-0.003	0.235	0.001	0.395	0	0.987
Superior Frontal Gyrus	-0.001	0.757	-0.004	0.138	0.001	0.395	0	0.987
Superior Parietal Lobule	-0.001	0.631	-0.005*	0.0945	0.001	0.553	0	0.987
Superior Temporal Gyrus	0	0.878	-0.003	0.235	0.001	0.514	0	0.987
Temporal Pole	-0.001	0.823	-0.005	0.138	0.001	0.646	0	0.987

Note. Significant uncorrected parameters are bolded. * $p < .05$. ** $p < .01$.

Table 15*Associations Between Memory and Bilateral ROI Thickness Parameters in the Older Cohort*

Bilateral Thickness in the Older Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	0.003	0.663	0	0.903	-0.001	0.869	0	0.93
Posterior Cingulate Cortex	0.001	0.84	0	0.903	0	0.999	0	0.93
Anterior Cingulate Cortex	0.001	0.84	0.002	0.765	0	0.999	0	0.93
Entorhinal Cortex	0.006	0.663	-0.001	0.903	-0.003*	0.57	0.002*	0.735
Inferior Parietal Sulcus	-0.004	0.663	0.005	0.42	0	0.999	-0.001	0.93
Lateral Occipital Cortex	0.001	0.822	0.002	0.765	-0.001	0.84	0	0.93
Lingual Gyrus	0.002	0.663	0	0.903	0	0.999	0	0.93
Medial Orbitofrontal	0.006*	0.663	-0.001	0.842	-0.001	0.57	0	0.933
Middle Temporal Gyrua	-0.001	0.822	0.004*	0.27	-0.001	0.589	0	0.933
Parahippocampal Gyrus	-0.004	0.663	0.003	0.765	-0.001	0.57	0	0.957
Precuneus	0.002	0.663	0	0.903	0	0.869	0	0.957
Rostral Middle Frontal	0.006	0.663	-0.001	0.903	-0.001	0.671	0	0.957
Superior Frontal Gyrus	-0.004	0.663	0.003	0.6	-0.001	0.671	0	0.957
Superior Parietal Lobule	0.002	0.822	0.001	0.903	0	0.869	0	0.957
Superior Temporal	0.001	0.822	0.001	0.903	-0.001	0.671	0	0.957

Note. Significant uncorrected parameters are bolded. * $p < .05$.

Table 16

Associations Between Memory and Bilateral ROI Surface Area Growth Parameters in the Younger Cohort

Bilateral Area in the Younger Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	-8.186	0.836	16.128	0.45	-0.172	0.897	0.203	0.859
Posterior Cingulate Cortex	0.114	0.973	4.502	0.454	0.01	0.897	0.024	0.859
Anterior Cingulate Cortex	1.086	0.836	1.949	0.45	-0.044	0.897	0.258	0.859
Inferior Parietal Sulcus	4.21	0.836	-4.036	0.744	0.143	0.849	0.799	0.859
Lingual Gyrus	-5.742	0.836	1.163	0.799	-0.832	0.897	0.45	0.859
Medial Orbitofrontal Cortex	3.225	0.836	2.62	0.454	-0.03	0.897	0.011	0.859
Middle Temporal	0.64	0.967	1.202	0.784	-0.322	0.897	-0.353	0.859
Parahippocampal Gyrus	1.079	0.836	-1.931	0.45	0.075	0.897	-0.047	0.859
Precuneus	6.469	0.836	9.926	0.45	-0.403	0.897	0.468	0.859
Superior Frontal Gyrus	-2.314	0.948	3.525	0.784	-0.479	0.897	-0.138	0.859
Superior Parietal Lobule	4.356	0.836	8.685	0.454	-0.295	0.897	-0.072	0.859
Temporal Pole	-0.531	0.836	0.752	0.465	0.121	0.897	0.028	0.859

Note. Significant uncorrected parameters are bolded. * $p < .05$.

Table 17

Associations Between Memory and Bilateral ROI Surface Area Growth Parameters in the Older Cohort

Bilateral Area in the Older Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	3.18	0.791	-0.942	0.899	0.403	0.706	-0.149	0.825
Posterior Cingulate Cortex	4.365	0.791	-4.712	0.899	-0.976	0.706	0.42	0.288
Entorhinal Cortex	0.693	0.791	0.081	0.899	0.343	0.706	-0.32*	0.147
Lingual Gyrus	-5.242	0.791	0.97	0.899	-0.236	0.706	0.434*	0.147
Parahippocampal Gyrus	0.662	0.791	0.136	0.899	0.239	0.706	-0.198	0.147
Superior Frontal Gyrus	24.996	0.791	-8.911	0.899	-0.181	0.706	-0.181	0.825
Superior Parietal Lobule	-1.654	0.791	9.551	0.899	-0.058	0.706	-0.058	0.89
Temporal Pole	0.795	0.791	-0.134	0.899	-0.039	0.706	-0.039	0.825

Note. Significant parameters are bolded. * $p < .05$.

Table 18

Fit Indices for Lateralized Bivariate Growth Curve Models Examining Associations Between ROI Thickness and the Latent Episodic Memory Variable

Fit Indices for Lateralized Thickness Co-development	Younger Cohort		Older Cohort	
	Left Hemisphere	Right Hemisphere	Left Hemisphere	Right Hemisphere
Inferior Frontal Gyrus	0.000 (0.000 – 0.051)	0.016 (0.000 – 0.059)	0.039 (0.000 – 0.068)	0.045 (0.000 – 0.073)
Posterior Cingulate Cortex	0.005 (0.000 – 0.056)	0.000 (0.000 – 0.054)	0.025 (0.000 – 0.060)	0.043 (0.000 – 0.071)
Anterior Cingulate Cortex	0.000 (0.000 – 0.050)	0.030 (0.000 – 0.065)	0.000 (0.000 – 0.047)	0.000 (0.000 – 0.049)
Entorhinal Cortex	0.007 (0.000 – 0.056)	0.019 (0.000 – 0.059)	0.066 (0.000 – 0.089)	0.000 (0.000 – 0.056)
Inferior Parietal Sulcus	0.000 (0.000 – 0.055)	0.021 (0.000 – 0.060)	0.017 (0.000 – 0.056)	0.034 (0.000 – 0.065)
Lateral Occipital Cortex	0.043 (0.000 – 0.073)	0.042 (0.000 – 0.073)	0.039 (0.000 – 0.068)	0.024 (0.000 – 0.059)
Lingual Gyrus	0.056 (0.000 – 0.082)	0.031 (0.000 – 0.065)	0.040 (0.000 – 0.069)	0.056 (0.023 – 0.082)
Medial Orbitofrontal	0.020 (0.000 – 0.060)	0.033 (0.000 – 0.066)	0.059 (0.029 – 0.084)	0.058 (0.028 – 0.083)
Middle Temporal Gyrus	0.000 (0.000 – 0.038)	0.000 (0.000 – 0.044)	0.053 (0.015 – 0.079)	0.059 (0.029 – 0.084)
Parahippocampal Gyrus	0.025 (0.000 – 0.062)	0.041 (0.000 – 0.072)	0.065 (0.038 – 0.089)	0.000 (0.000 – 0.048)
Precuneus	0.000 (0.000 – 0.036)	0.028 (0.000 – 0.063)	0.053 (0.015 – 0.079)	0.021 (0.000 – 0.058)
Rostral Middle Frontal	0.046 (0.000 – 0.075)	0.040 (0.000 – 0.071)	0.023 (0.000 – 0.059)	0.036 (0.000 – 0.067)
Superior Frontal Gyrus	0.018 (0.000 – 0.058)	0.015 (0.000 – 0.058)	0.044 (0.000 – 0.072)	0.038 (0.000 – 0.068)
Superior Parietal Lobule	0.037 (0.000 – 0.069)	0.043 (0.000 – 0.073)	0.036 (0.000 – 0.066)	0.039 (0.000 – 0.068)
Superior Temporal Gyrus	0.019 (0.000 – 0.059)	0.011 (0.000 – 0.056)	0.058 (0.027 – 0.083)	0.015 (0.000 – 0.056)
Temporal Pole	0.026 (0.000 – 0.063)	0.032 (0.000 – 0.065)	0.051 (0.000 – 0.078)	0.000 (0.000 – 0.051)

Table 19

Fit Indices for Lateralized Bivariate Growth Curve Models Examining Associations Between ROI Surface Area and the Latent Episodic Memory Variable

Fit Indices for Lateralized Area Co-development	Younger Cohort		Older Cohort	
	Left Hemisphere	Right Hemisphere	Left Hemisphere	Right Hemisphere
Inferior Frontal Gyrus	0.056 (0.019 – 0.083)	0.000 (0.000 – 0.044)	0.000 (0.000 – 0.050)	0.000 (0.000 – 0.047)
Posterior Cingulate Cortex	0.024 (0.000 – 0.061)	0.025 (0.000 – 0.062)	0.019 (0.000 – 0.057)	0.019 (0.000 – 0.057)
Anterior Cingulate Cortex	0.049 (0.000 – 0.078)	0.031 (0.000 – 0.065)	0.029 (0.000 – 0.062)	0.029 (0.000 – 0.062)
Entorhinal Cortex	0.038 (0.000 – 0.069)	0.016 (0.000 – 0.082)	0.022 (0.000 – 0.059)	0.000 (0.000 – 0.042)
Inferior Parietal Sulcus	0.040 (0.000 – 0.071)	0.037 (0.000 – 0.069)	0.032 (0.000 – 0.064)	0.051 (0.009 – 0.077)
Lateral Occipital Cortex	0.032 (0.000 – 0.066)	0.028 (0.000 – 0.063)	0.000 (0.000 – 0.052)	0.028 (0.000 – 0.062)
Lingual Gyrus	0.060 (0.026 – 0.086)	0.043 (0.000 – 0.073)	0.035 (0.000 – 0.066)	0.035 (0.000 – 0.066)
Medial Orbitofrontal	0.023 (0.000 – 0.061)	0.003 (0.000 – 0.055)	0.000 (0.050 – 0.076)	0.044 (0.000 – 0.072)
Middle Temporal Gyrus	0.046 (0.000 – 0.075)	0.041 (0.000 – 0.071)	0.000 (0.000 – 0.047)	0.045 (0.000 – 0.073)
Parahippocampal Gyrus	0.000 (0.000 – 0.054)	0.041 (0.000 – 0.071)	0.061 (0.032 – 0.086)	0.043 (0.000 – 0.071)
Precuneus	0.050 (0.000 – 0.078)	0.038 (0.000 – 0.070)	0.031 (0.000 – 0.063)	0.034 (0.000 – 0.064)
Rostral Middle Frontal	0.056 (0.019 – 0.083)	0.054 (0.013 – 0.082)	0.036 (0.000 – 0.066)	0.000 (0.000 – 0.050)
Superior Frontal Gyrus	0.045 (0.019 – 0.075)	0.029 (0.000 – 0.064)	0.041 (0.000 – 0.069)	0.037 (0.000 – 0.067)
Superior Parietal Lobule	0.024 (0.000 – 0.062)	0.017 (0.000 – 0.059)	0.000 (0.000 – 0.053)	0.016 (0.000 – 0.056)
Superior Temporal Gyrus	0.013 (0.000 – 0.057)	0.030 (0.000 – 0.064)	0.030 (0.000 – 0.063)	0.000 (0.000 – 0.046)
Temporal Pole	0.000 (0.000 – 0.052)	0.056 (0.020 – 0.083)	0.025 (0.000 – 0.060)	0.000 (0.000 – 0.053)

Table 20*Associations of Memory with Left ROI Thickness Parameters in the Younger Cohort*

Left Thickness in the Younger Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	Adj. p	Estimate	Adj. p	Estimate	Adj. p	Estimate	Adj. p
Inferior Frontal Gyrus	0.001	0.853	-0.005*	0.072	0.001	0.928	0	0.982
Posterior Cingulate Cortex	0.001	0.886	-0.003	0.31	0	0.971	0	0.982
Anterior Cingulate Cortex	-0.006*	0.384	0	0.958	0.002	0.528	-0.001	0.982
Entorhinal Cortex	-0.004	0.853	-0.001	0.926	0	0.971	0	0.982
Inferior Parietal Sulcus	0.001	0.853	-0.003*	0.096	0.001	0.928	0	0.982
Lateral Occipital Cortex	-0.001	0.853	-0.003*	0.072	0.001	0.928	0	0.982
Lingual Gyrus	-0.001	0.853	-0.006*	0.072	0	0.971	0.001	0.982
Medial Orbitofrontal	-0.002	0.853	-0.002	0.32	0	0.974	0	0.982
Middle Temporal Gyrus	0.002	0.853	-0.003	0.149	0	0.974	0	0.982
Parahippocampal Gyrus	-0.002	0.853	0	0.958	0.001	0.528	0	0.982
Precuneus	-0.001	0.853	-0.004*	0.072	0	0.928	0	0.982
Rostral Middle Frontal	0	0.946	-0.003	0.32	0.001	0.928	0	0.982
Superior Frontal Gyrus	-0.002	0.853	-0.003	0.31	0.001	0.928	0	0.982
Superior Parietal Lobule	-0.001	0.853	-0.005**	0.072	0	0.971	0	0.982
Superior Temporal Gyrus	0.001	0.853	-0.005*	0.072	0	0.971	0	0.982
Temporal Pole	0.003	0.853	-0.011*	0.089	0	0.971	0	0.982

Note. Significant uncorrected parameters are bolded. * $p < .05$. ** $p < .01$.

Table 21*Associations of Memory with Left ROI Thickness Parameters in the Older Cohort*

Left Thickness in the Older Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	Adj. p	Estimate	Adj. p	Estimate	Adj. p	Estimate	Adj. p
Inferior Frontal Gyrus	0	0.917	0.001	0.833	0	0.975	0	0.983
Posterior Cingulate Cortex	0.003	0.743	-0.001	0.861	0	0.916	0	0.957
Anterior Cingulate Cortex	0.001	0.789	0.003	0.595	0	0.975	0	0.957
Entorhinal Cortex	0.008	0.743	-0.001	0.861	-0.003	0.691	0.002*	0.48
Inferior Parietal Sulcus	-0.003	0.743	0.003	0.595	0	0.916	0	0.983
Lateral Occipital Cortex	0.002	0.789	0.001	0.638	0	0.975	0	0.864
Lingual Gyrus	0.001	0.789	0.001	0.69	0	0.975	-0.001	0.48
Medial Orbitofrontal	0.007*	0.336	-0.002	0.595	-0.001	0.891	0	0.983
Middle Temporal Gyrus	-0.001	0.917	0.004	0.595	-0.001	0.691	0	0.983
Parahippocampal Gyrus	-0.002	0.789	0.002	0.861	-0.001	0.691	0	0.983
Precuneus	0.002	0.743	-0.001	0.833	-0.001	0.814	0	0.983
Rostral Middle Frontal	0.004	0.743	0	0.873	0	0.975	0	0.983
Superior Frontal Gyrus	-0.003	0.743	0.003	0.595	-0.001	0.691	0	0.983
Superior Parietal Lobule	0.003	0.743	0	0.873	-0.001	0.86	0	0.983
Superior Temporal Gyrus	0.001	0.789	0.002	0.69	-0.001	0.814	0	0.983
Temporal Pole	0.005	0.743	-0.005	0.595	-0.002	0.691	0.001	0.983

Note. Significant uncorrected parameters are bolded. * $p < .05$.

Table 22*Associations of Memory with Right ROI Thickness Parameters in the Younger Cohort*

Right Thickness in the Younger Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	Adj. <i>p</i>	Estimate	Adj. <i>p</i>	Estimate	Adj. <i>p</i>	Estimate	Adj. <i>p</i>
Inferior Frontal Gyrus	0	0.962	-0.005*	0.159	0.001	0.534	0	0.93
Posterior Cingulate Cortex	-0.002	0.962	-0.002	0.683	0	0.772	0	0.928
Anterior Cingulate Cortex	-0.003	0.962	-0.004	0.192	0	0.772	0.001	0.928
Entorhinal Cortex	-0.005	0.962	-0.003	0.683	0.001	0.293	-0.001*	0.704
Inferior Parietal Sulcus	-0.001	0.962	-0.003	0.33	0	0.534	0	0.928
Lateral Occipital Cortex	0	0.962	-0.003	0.192	0.001	0.293	0	0.928
Lingual Gyrus	-0.002	0.962	-0.004	0.165	0	0.495	0	0.928
Medial Orbitofrontal	-0.003	0.962	-0.001	0.791	0.002*	0.088	-0.001	0.928
Middle Temporal Gyrus	-0.002	0.962	-0.003*	0.159	0.001	0.293	0	0.928
Parahippocampal Gyrus	0.001	0.962	0	0.937	0.001	0.293	0	0.928
Precuneus	0	0.962	-0.003*	0.159	0.001	0.293	0	0.93
Rostral Middle Frontal	-0.003	0.962	-0.004	0.33	0.002**	0.088	0	0.93
Superior Frontal Gyrus	0	0.962	-0.005*	0.159	0.001	0.293	0	0.928
Superior Parietal Lobule	-0.001	0.962	-0.005*	0.159	0.001	0.293	0	0.928
Superior Temporal Gyrus	-0.001	0.962	0	0.937	0.001	0.293	0	0.93
Temporal Pole	-0.003	0.962	0	0.937	0.002	0.495	-0.001	0.928

Note. Significant uncorrected parameters are bolded. * $p < .05$.**Table 23***Associations of Memory with Right ROI Thickness Parameters in the Older Cohort*

Right Thickness in the Older Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	Adj. <i>p</i>	Estimate	Adj. <i>p</i>	Estimate	Adj. <i>p</i>	Estimate	Adj. <i>p</i>
Inferior Frontal Gyrus	0.006	0.64	0	0.921	-0.002	0.464	0	0.745
Posterior Cingulate Cortex	-0.002	0.8	0.002	0.712	0	1	0	0.745
Anterior Cingulate Cortex	-0.001	0.904	0	0.921	0	0.896	-0.001	0.745
Entorhinal Cortex	0.006	0.8	0	0.998	-0.003	0.464	0	0.992
Inferior Parietal Sulcus	-0.006	0.64	0.007*	0.296	0	0.896	-0.001	0.745
Lateral Occipital Cortex	0	0.904	0.002	0.59	-0.001	0.708	0	0.745
Lingual Gyrus	0.003	0.734	-0.002	0.59	0	1	0	0.992
Medial Orbitofrontal	0.005	0.64	0	0.921	-0.002	0.464	0	0.745
Middle Temporal Gyrus	-0.002	0.8	0.005*	0.288	-0.001	0.518	0	0.818
Parahippocampal Gyrus	-0.006	0.64	0.004	0.58	-0.001	0.787	0	0.992
Precuneus	0.002	0.8	0.001	0.712	0	0.896	0	0.992
Rostral Middle Frontal	0.01	0.64	-0.003	0.62	-0.002	0.464	0.001	0.832
Superior Frontal Gyrus	-0.004	0.64	0.003	0.58	-0.001	0.708	0	0.992
Superior Parietal Lobule	0.001	0.904	0	0.921	0	1	0	0.992
Superior Temporal Gyrus	0.002	0.8	0.001	0.921	-0.001	0.708	0	0.992
Temporal Pole	-0.003	0.827	0.004	0.59	0	0.708	0	0.745

Note. Significant uncorrected parameters are bolded. * $p < .05$.

Table 24*Associations of Memory with Left ROI Surface Area Parameters in the Younger Cohort*

Left Surface Area in the Younger Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	-6.479	0.64	10.462	0.33	0.112	0.972	0.152	0.906
Posterior Cingulate Cortex	-2.7	0.64	6.672	0.33	0.354	0.838	0.143	0.906
Anterior Cingulate Cortex	2.17	0.64	3.928	0.33	-0.086	0.838	0.515	0.592
Entorhinal Cortex	2.328	0.64	-1.394	0.33	0.372	0.712	-0.045	0.906
Inferior Parietal Sulcus	13.702	0.64	-10.893	0.33	0.157	0.972	0.754	0.906
Lateral Occipital Cortex	11.129	0.64	-11.771	0.33	-0.041	0.712	0.023	0.906
Lingual Gyrus	-8.098	0.64	2.899	0.651	-1.173	0.838	1.149	0.592
Medial Orbitofrontal	0.783	0.792	3.288	0.33	-0.138	0.972	0.149	0.906
Middle Temporal Gyrus	-4.679	0.64	1.684	0.721	-0.413	0.972	0.221	0.906
Parahippocampal Gyrus	1.2	0.64	-1.683	0.33	0.03	0.972	0.001	0.994
Precuneus	6.892	0.64	7.682	0.33	-0.386	0.838	0.466	0.832
Rostral Middle Frontal	-6.525	0.64	8.375	0.519	0.12	0.972	2.029	0.592
Superior Frontal Gyrus	-4.81	0.64	3.137	0.739	-0.012	0.993	0.214	0.906
Superior Parietal Lobule	4.579	0.64	8.07	0.335	-0.427	0.838	0.031	0.936
Superior Temporal Gyrus	-6.567	0.64	6.754	0.33	-0.561	0.838	0.128	0.906
Temporal Pole	-0.826	0.64	0.811	0.33	0.259	0.838	0.075	0.906

Note. Significant uncorrected parameters are bolded.**Table 25***Associations of Memory with Left ROI Surface Area Parameters in the Older Cohort*

Left Surface Area in the Older Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	-1.08	0.978	2.692	0.808	0.151	0.956	0.194	0.596
Posterior Cingulate Cortex	1.534	0.978	-2.073	0.808	-0.015	0.956	0.015	0.726
Anterior Cingulate Cortex	1.139	0.978	1.911	0.808	-0.8	0.634	0.719*	0.144
Entorhinal Cortex	0.28	0.978	0.6	0.808	0.526	0.634	-0.469**	0.08
Inferior Parietal Sulcus	15.955	0.923	0.377	0.955	0.306	0.921	0.341*	0.166
Lateral Occipital Cortex	16.008	0.923	-7.35	0.808	-0.296	0.921	0.165	0.552
Lingual Gyrus	-3.726	0.978	1.93	0.808	-0.179	0.921	0.254	0.204
Medial Orbitofrontal	6.984	0.923	-1.571	0.808	0.002	0.996	-0.09	0.552
Middle Temporal Gyrus	4.26	0.978	0.796	0.921	-0.337	0.956	-0.29	0.552
Parahippocampal Gyrus	1.139	0.978	-0.408	0.808	0.302	0.634	-0.209	0.204
Precuneus	2.869	0.978	2.884	0.808	-0.199	0.634	0.189	0.166
Rostral Middle Frontal	-4.474	0.978	7.75	0.808	0.125	0.956	-0.814	0.204
Superior Frontal Gyrus	17.007	0.978	-7.388	0.808	0.412	0.956	0.245	0.726
Superior Parietal Lobule	-0.263	0.983	7.216	0.808	0.505	0.921	0.149	0.726
Superior Temporal Gyrus	5.339	0.978	-2.61	0.808	1.107	0.634	-0.805*	0.166
Temporal Pole	-0.138	0.978	0.313	0.808	0.45	0.763	-0.255	0.263

Note. Significant uncorrected parameters are bolded. * $p < .05$. ** $p < .01$.

Table 26*Associations of Memory with Right ROI Surface Area Parameters in the Younger Cohort*

Right Surface Area in the Younger Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	-1.304	0.931	5.459	0.586	-0.312	0.957	0.082	0.804
Posterior Cingulate Cortex	2.071	0.868	3.221	0.586	0.232	0.957	-0.487	0.512
Anterior Cingulate Cortex	-2.085	0.868	2.173	0.586	-0.026	0.957	-0.165	0.785
Entorhinal Cortex	3.199	0.552	-1.182	0.586	0.127	0.848	-0.136*	0.512
Inferior Parietal Sulcus	13.597	0.868	-10.891	0.586	0.206	0.957	0.636	0.704
Lateral Occipital Cortex	4.818	0.868	-3.684	0.799	-0.157	0.957	0.276	0.804
Lingual Gyrus	-3.991	0.868	-0.572	0.9	-0.57	0.957	0.214	0.704
Medial Orbitofrontal	5.437*	0.352	1.797	0.586	0.027	0.957	-0.012	0.704
Middle Temporal Gyrus	-1.864	0.868	1.748	0.799	-0.155	0.957	-0.261	0.704
Parahippocampal Gyrus	1.133	0.868	-2.238*	0.376	0.098	0.957	-0.113	0.588
Precuneus	6.246	0.868	11.756*	0.376	-0.417	0.957	0.406	0.704
Rostral Middle Frontal	-1.334	0.931	7.231	0.586	-0.597	0.957	0.825	0.704
Superior Frontal Gyrus	-0.835	0.931	2.695	0.866	-1.539	0.848	-0.48	0.704
Superior Parietal Lobule	4.39	0.868	8.568	0.586	-0.132	0.957	-0.162	0.704
Superior Temporal Gyrus	-3.798	0.868	4.165	0.586	-0.863	0.955	0.889	0.588
Temporal Pole	0.16	0.931	0.191	0.866	0.023	0.957	0.054	0.804

Note. Significant uncorrected parameters are bolded. * $p < .05$.**Table 27***Associations of Memory with Right ROI Surface Area Parameters in the Older Cohort*

Right Surface Area in the Older Cohort	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>	Estimate	<i>Adj. p</i>
Inferior Frontal Gyrus	6.593	0.87	-4.286	0.995	0.663	0.935	-0.444	0.557
Posterior Cingulate Cortex	1.696	0.984	-1.974	0.995	-0.186	0.935	0.043	0.709
Anterior Cingulate Cortex	0.197	0.984	0.812	0.995	0.01	0.935	-0.006	0.557
Entorhinal Cortex	1.579	0.87	-0.798	0.995	0.25	0.935	-0.197	0.557
Inferior Parietal Sulcus	24.819	0.984	-11.327	0.995	-0.545	0.935	-0.161	0.773
Lateral Occipital Cortex	-0.688	0.984	1.154	0.995	0.68	0.935	-0.885	0.557
Lingual Gyrus	-6.715	0.87	0.048	0.995	-0.171	0.935	0.431*	0.528
Medial Orbitofrontal	4.492	0.87	-0.718	0.995	0.14	0.935	0.451	0.557
Middle Temporal Gyrus	-0.373	0.984	4.268	0.995	-0.444	0.935	0.063	0.854
Parahippocampal Gyrus	-0.257	0.984	0.824	0.995	0.008	0.935	-0.019	0.557
Precuneus	0.269	0.984	0.577	0.995	0.001	0.935	0.005	0.557
Rostral Middle Frontal	-0.266	0.984	1.151	0.995	1.071	0.935	-0.366	0.557
Superior Frontal Gyrus	30.518	0.87	-9.218	0.995	0.708	0.935	-0.401	0.557
Superior Parietal Lobule	-4.051	0.984	12.846	0.995	0.557	0.935	-0.266	0.631
Superior Temporal Gyrus	4.909	0.87	0.02	0.995	-0.134	0.935	-0.272	0.567
Temporal Pole	1.761	0.87	-0.558	0.995	-0.069	0.935	0.136	0.557

Note. Significant uncorrected parameters are bolded. * $p < .05$.

Total Gray Matter Volume.

To ensure specificity of any ROI findings, co-development of total gray matter volume with latent episodic memory was examined. Fit for both the younger (RMSEA = 0.028 (0.000 - 0.042)) and older cohort (RMSEA = 0.070 (0.045 - 0.093)) were acceptable. Results showed that total gray matter volume does not co-develop with latent episodic memory in either cohort ($ps > .47$); Table 28). Additionally, initial gray matter volume does not predict memory development ($ps > .43$), and initial memory volumes do not predict total gray matter development ($ps > 0.22$). These findings suggest that associations between the growth parameters of each ROI and growth parameters of the latent episodic memory variable are region specific.

Table 28

Associations of Memory with Total Gray Matter Volume Parameters

Co-development of Memory with Total Gray Matter Volume.	ROI Int. WITH Memory Int.		ROI Int WITH Memory Slope		ROI Slope WITH Memory Int.		ROI Slope WITH Memory Slope	
	Estimate	<i>p</i>	Estimate	<i>p</i>	Estimate	<i>p</i>	Estimate	<i>p</i>
Younger Cohort	-0.709	0.911	-4.376	0.43	2.629	0.223	-0.704	0.589
Older Cohort	12.608	0.224	4.508	0.488	-1.848	0.362	-0.778	0.474

Aim 2: Preliminary Findings

Initial outlier analysis revealed that there were no extreme outliers greater than 3 standard deviations above the grand mean. Next, preliminary analyses examined differences in age, sex, and average 24-hour sleep duration as a function of nap habituality (i.e., nappers, intermediate nappers, and non-nappers). Findings demonstrated that there was no significant differences in age $F(2, 41) = 1.782, p = .18$, ICV, $F(2, 41) = .772, p = .47$ nor sex $\chi^2 (1, N = 44) = .749$ based on nap status. Thus, age and sex will not be included as covariates in any of the Aim 2 analyses.

In contrast, results demonstrated a significant difference in average 24-hour sleep duration based on nap status $F(2,28) = 4.09$ $p = .03$. Specifically, nappers spent significantly more time sleeping during an average 24-hour period than non-nappers ($p = 0.02$). There were no other significant group differences were found ($ps > .13$). As a result, ROIs that significantly differ based on nap habituality were regressed with 24-hour sleep duration. If associations were significant, average 24-hour sleep duration was included as a covariate in the final analysis.

Aim 2: Associations between Nap Habituality and the Cortex

Selection of ROIs.

For this analysis, all ROIs that demonstrated significant co-development with the latent episodic memory variable were examined. This included cortical thickness from bilateral and left ERC along with surface area from bilateral ERC, bilateral lingual gyrus, left ACC, left ERC, left IPS, left superior temporal gyrus, and right lingual gyrus. Additionally, ROIs that demonstrated an association between initial ROI volume at age 4 and the slope of the latent episodic memory variable from 4 to 6 years were explored. For cortical thickness, this included, bilateral IFG, bilateral LOC, bilateral lingual gyrus, bilateral middle temporal gyrus, bilateral precuneus, bilateral SPL, left IFG, left IPL, left LOC, left lingual gyrus, left mOFC, left precuneus, left SPL, left superior temporal gyrus, left temporal pole, right IFG, right middle temporal gyrus, right precuneus, right superior frontal gyrus, and right SPL (Table 29). For surface area, this included right lateralized precuneus and right lateralized PHG (Table 30). Importantly, regions that only demonstrated an association between initial ROI volume at age 6 and the slope of episodic memory from 6 to 8 years were not examined because the sample for Aim 2 is aged 3 to 5 years. Therefore, regions that are only implicated later in development are not relevant to this investigation. Finally, differences in the lateral occipital cortex and lingual gyrus were also

assessed, regardless of their fit with the previous guidelines. This is due to their connection with the neuropsychological of memory (Amlien et al., 2018; Karanian & Slotnick, 2015; Rosen et al., 2018; Schommartz et al., 2023; Wing et al., 2015).

Associations between Cortical Thickness of Selected ROIs and Nap Habituality.

Results revealed that right IFG differed significantly based on nap habituality, $F(2,41) = 3.395, p = .04$ (Table 29). Follow-up analysis revealed that this region was not associated with 24-hour sleep duration ($p = .35$). Therefore, it will not be included as a covariate. Post hoc testing revealed that there were no significant differences between any of the groups, however, there were marginal differences between nappers and both intermediate nappers ($p = 0.07$) and non-nappers ($p = 0.08$). Specifically, habitual nappers demonstrated a marginally thicker IFG than children who napped less frequently (Figure 10). This difference between the F test and the post hoc test is likely due to the conservative nature of Tukey's HSD and the small sample in Study 2. Specifically, a post hoc power analysis in G*Power version 3.1.9.6 (Faul et al., 2007) showed that the present sample ($N = 44$) only had a 28% chance of detecting a moderate effect (i.e., $f = .25$) with an $\alpha = .05$. Furthermore, findings demonstrated that a sample of 155 would be required to detect a moderate effect. In sum, these findings could suggest that non-nappers and intermediate nappers demonstrated a more 'mature' IFG than habitual nappers. However, this association did not survive correction for multiple comparisons. No other regions differed significantly based on nap status ($p > .09$; see Table 29).

Table 29

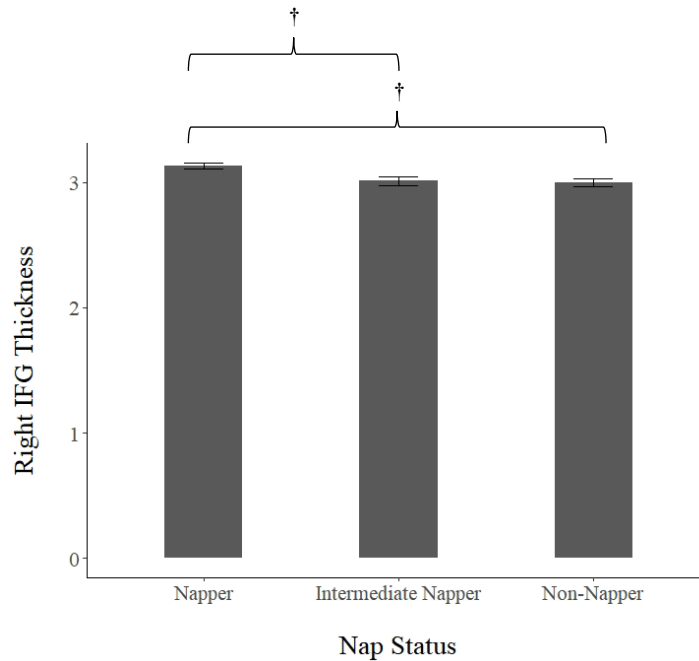
Differences in Cortical Thickness of Selected ROIs Based on Nap Status

ROIs	Bilateral Thickness			Left Thickness			Right Thickness		
	Fstat	p	Adj. p	Fstat	p	Adj. p	Fstat	p	Adj. p
Entorhinal Cortex	0.022	0.978	0.978	--	--	--	0.25	0.78	0.806
Inferior Frontal Gyrus	2.015	0.146	0.593	1.586	0.217	0.660	3.395*	0.043	0.389
Inferior Parietal Sulcus	--	--	--	1.468	0.242	0.660	--	--	--
Lateral Occipital Cortex	0.621	0.543	0.593	0.596	0.556	0.660	0.642	0.531	0.797
Lingual Gyrus	1.263	0.293	0.593	1.133	0.332	0.660	0.907	0.412	0.742
Medial Orbitofrontal	--	--	--	2.521	0.0928	0.660	--	--	--
Middle Temporal Gyrus	1.109	0.339	0.593	--	--	--	1.639	0.207	0.621
Posterior Parietal Cortex	--	--	--	--	--	--	0.217	0.806	0.806
Precuneus	1.382	0.262	0.593	0.541	0.587	0.660	1.883	0.165	0.621
Superior Frontal Gyrus	--	--	--	0.735	0.486	0.660	1.273	0.291	0.655
Superior Parietal Lobule	0.777	0.466	0.593	0.974	0.386	0.660	0.416	0.663	0.806
Temporal Pole	--	--	--	0.07	0.932	0.932	--	--	--

Note. Significant parameters, uncorrected for multiple comparisons, are bolded. * $p < .05$.

Figure 12

Differences in Inferior Frontal Gyrus Based on Nap Status



Note. Depicts findings uncorrected for multiple comparisons. † $p < .1$ uncorrected.

Associations between Surface Area of Selected ROIs and Nap Habituality.

Results revealed that bilateral LOC differed significantly based on nap habituality $F(2,41) = 4.272, p = .02$. Follow-up analysis revealed that LOC was not associated with 24-hour sleep duration ($p = .42$). Thus, it will not be included as a covariate. Post hoc testing revealed there were significant differences between nappers and both intermediate nappers ($p = 0.04$) and non-nappers ($p = 0.04$). Specifically, habitual nappers demonstrated a significantly less surface area in the LOC than children who napped less frequently (see Figure 11). Additionally, these findings generalized to both the left ($p = 0.04$) and right ($p = 0.04$) LOC (see Table 30). However, this association did not survive correction for multiple comparisons.

Table 30

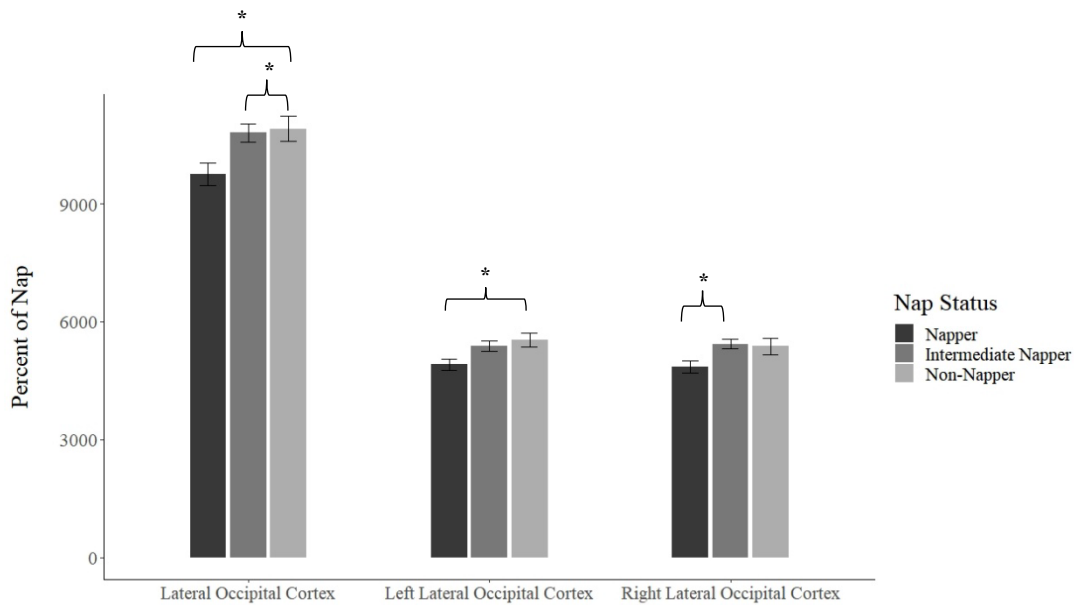
Differences in Surface Area of Selected ROIs Based on Nap Status

ROIs	Bilateral Thickness			Left Thickness			Right Thickness		
	Fstat	p	Adj. p	Fstat	p	Adj. p	Fstat	p	Adj. p
Anterior Cingulate Cortex	--	--	--	0.085	0.918	0.953	--	--	--
Entorhinal Cortex	0.237	0.79	0.790	0.262	0.771	0.953	--	--	--
Inferior Parietal Sulcus	--	--	--	0.048	0.953	0.953	--	--	--
Lingual Gyrus	0.307	0.738	0.790	0.859	0.431	0.953	3.161	0.0529	0.106
Lateral Occipital Cortex	4.272*	0.0206	0.082	3.419*	0.0424	0.254	3.508*	0.0392	0.106
Parahippocampal Gyrus	--	--	--	--	--	--	0.407	0.668	0.520
Precuneus	0.237	0.79	0.790	--	--	--	0.965	0.39	0.668
Superior Temporal Gyrus	--	--	--	0.088	0.916	0.953	--	--	--

Note. Significant parameters, uncorrected for multiple comparisons, are bolded. * $p < .05$.

Figure 13

Differences in Lateral Occipital Cortex Based on Nap Status



Note. Depicts findings uncorrected for multiple comparisons. * $p < .05$ uncorrected.

Whole Brain Associations with Nap Habituality.

Findings from the whole brain analysis revealed that there were no associations between average naps per week and cortical thickness that survived correction for multiple comparisons.

There were also no associations cortical surface area that survived Monte Carlo simulations.

Specificity of ROI Findings.

To ensure that all effects were region-specific, differences in total gray matter volume as a function of nap status were explored. Results revealed that there were no significant differences in total gray matter volume based on nap habituality, $F(2,41) = .131$ $p = .88$. Therefore, associations are likely region specific. Additionally, to explore whether associations between nap

status and significant regions of interest (i.e., LOC and IFG) were attributed to downstream effects from the hippocampus, relations between these regions and hippocampal volumes were investigated. Results revealed that there were no associations between ROIs (i.e., LOC and IFG) and total hippocampal volume when controlling for ICV ($p > .14$), suggesting that ROI findings are not attributed to a common association with the hippocampus.

Chapter 6: Discussion

The purpose of this dissertation was to 1) characterize the longitudinal development of cortical regions that support memory in early to middle childhood, 2) examine cross-sectional and longitudinal associations between episodic memory and the cortex during childhood, and 3) explore individual differences in cortical regions that co-develop with memory based on nap habituality.

Findings demonstrated that early to middle childhood is marked by gradual linear thinning in cortical thickness of most regions (i.e., IFG, PCC, ACC, IPS, LOC, lingual gyrus, middle temporal gyrus, PHG, precuneus, rostral middle frontal gyrus, superior frontal gyrus, SPL, and superior temporal gyrus). Results also showed that surface area demonstrates early increases in most ROIs (i.e., IFG, PCC, ACC, ERC, IPS, LOC, lingual gyrus, middle temporal gyrus, PHG, precuneus, rostral middle frontal gyrus, superior frontal gyrus, SPL, superior temporal gyrus, temporal pole) that slow and eventually give way to later decreases in some regions (i.e., IPS, LOC, lingual gyrus, middle temporal gyrus, mOFC, precuneus, and SPL). Importantly, this work builds upon existing literature that suggests there are developmental distinctions between cortical thickness and surface area (Hutton et al., 2009; Li et al., 2013; Lyall et al., 2015; Wierenga et al., 2014).

Furthermore, findings revealed that there were both concurrent and longitudinal associations between structural cortical measures and memory performance for regions within the episodic memory network. Although, the specific region and measure implicated varied by analysis. For example, cross-sectional analysis in 4- to 8-year-old children suggested more prevalent associations between memory and cortical surface area ($N=6$ regions) than cortical thickness ($n=1$ region). For longitudinal analyses, findings were additionally dependent on the

cohort. Specifically, cortical thickness in multiple regions (i.e., IFG, IPS, LOC, lingual gyrus, mOFC, middle temporal gyrus, precuneus, SPL, superior frontal gyrus, superior temporal gyrus, and temporal pole) at 4 years of age predicted improvements in memory from age 4 to 6 years. Additionally, changes in cortical surface area of lingual gyrus, ERC, ACC, IPS, and superior temporal gyrus predicted improvements in memory performance from age 6 to 8 years. These findings expand on previous work that suggests cross-sectional and longitudinal analyses may implicate different ROIs (Keresztes et al., 2022) and provide evidence that surface area and cortical thickness contribute differently to memory development. However, neither cross-sectional nor longitudinal findings survived correction for multiple comparisons.

Finally, results demonstrated that individual differences in nap habituality predicted structural measures of cortical regions that support memory development across childhood. Namely, non-nappers had significantly greater surface area in LOC and a marginally thinner IFG when compared to their habitual nappers. Additionally, intermediate nappers demonstrated significantly less surface area in LOC than non-nappers and marginally thinner IFG than habitual nappers. Given that cortical thickness decreases with age, these results may suggest that non-nappers demonstrate greater maturity in regions that support memory performance. However, future work is needed to verify these results, as they did not survive correction for multiple comparisons. In the following section I further expand upon these results and their relation to existing literature. In each subsection I first discuss cortical thickness then cortical surface area.

Cross-Sectional Associations

Hypothesis 1A predicted that there would be significant negative associations between the cortical thickness and surface area of ROIs and a composite memory variable such that a thinner cortex and less surface area would be associated with better memory performance.

Consistent with this hypothesis, results demonstrated a negative association between the cortical thickness of right ACC and the composite memory variable. This finding is similar to past work that demonstrated a cross-sectional association between right rostral ACC and memory integration abilities in 5 to 8-year-old children (Bauer et al., 2019). However, in contrast to previous work utilizing similar samples (i.e., childhood samples aged 4 to 8 years), there were no other associations between cortical thickness and the composite memory variable (Schommartz et al., 2023).

There are several potential reasons for differences between the present study and past work. The first is that cross-sectional associations with cortical thickness in other regions could be task specific. Notably, this study utilized a composite memory variable that accounted for a battery of memory tasks. Whereas other studies have only examined associations with one task at a time (Bauer et al., 2019; Chad-Friedman et al., 2021; Schommartz et al., 2023). The second potential reason is differences in samples. Specifically, this study utilized a cross-sectional sample that covered a 4-year developmental period. In contrast, previous studies have only covered 2 (e.g., 5 to 7 years; Schommartz et al., 2023) or 3 year development periods (i.e., 5 to 8 years; Bauer et al., 2019; Chad-Friedman et al., 2021). This is critical because certain associations may be specific to younger or older children. As a result, a lack of association in younger children could mask effects in older children and vice versa. Importantly, there is evidence of developmental differences in our current sample. Specifically, the bivariate growth curve models demonstrated that cortical thickness in ACC was negatively associated with the latent episodic memory performance at age 4, but not age 6 suggesting that findings are specific to the younger cohort.

Importantly, growth models also uncovered an additional cross-sectional association at age 6 that was not found in the 4 to 8 year old cross-sectional sample. Specifically, left mOFC was associated with latent episodic memory performance at 6 years, but not 4 years. This is similar to findings from Otsby and colleagues (2021) who found a significant negative association between left mOFC and a visual-spatial memory task in participants aged 8 to 19 years. However, findings were in the opposite direction of Otsby (2021), such that greater thickness in mOFC was associated with better memory performance. This is likely attributed to the much larger age range included in that sample. In sum, cross-sectional associations between cortical thickness and memory performance associations are variable across studies and may be highly age dependent or more sensitive to individual differences.

Consistent with Hypothesis 1A, there were also associations between the composite memory variable and cortical surface area in medial orbital frontal gyrus, rostral middle frontal gyrus, ACC, SPL, and precuneus. However, associations were in the opposite direction of cortical thickness, such that greater surface area was associated with better memory performance. This is likely accounted for by the development trajectory of surface area during this developmental period. Specifically, unlike cortical thickness, cortical surface area in these regions demonstrate increases from 4 to 8 years. These findings are novel because previous studies have not examined associations between episodic memory and cortical surface area during early to middle childhood. However, these results do expand upon previous work in late adolescence that demonstrated associations between memory performance and cortical surface area in anterior right middle frontal gyrus and inferior frontal gyrus (Lyle et al., 2017). Together with cortical thickness findings, these results provide evidence that cortical surface area demonstrates distinct associations with memory performance that do not yield associations in the

same regions as cortical thickness. This may also suggest that surface area in the episodic memory network is a better predictor of cross-sectional associations with memory performance in 4- to 8-year-olds.

Longitudinal Development of the Episodic Memory Network.

Hypothesis 1B predicted that there would be age-related changes in cortical thickness and surface area from ages 4 to 8 years. Additionally, Research Question 1A aimed to assess whether changes were linear or non-linear from ages 4 to 8 years.

Consistent with Hypothesis 1B, findings demonstrated wide-spread linear cortical thinning in the episodic memory network from ages 4 to 8 years, with few regional exceptions. Specifically, only ERC, temporal pole, left LOC, and left PHG demonstrated no significant thinning from 4 to 8 years. Importantly, this may suggest that these regions are early developing and that changes may precede the developmental period examined. Additionally, only left mOFC demonstrated significant thickening, indicating that this region may be later developing. These findings add to a substantial body of literature that suggests early to middle childhood is marked by gradual, mostly, linear decreases in cortical thickness (Ducharme et al., 2016; Fjell et al., 2015; Mills et al., 2014; Mutlu et al., 2013; Remer et al., 2017; Sowell et al., 2004; Tamnes et al., 2017; Wierenga et al., 2014; Zielinski et al., 2014). However, findings are contrary to a handful of other studies that suggest an inverted U-shape trajectory in samples examining early childhood through adulthood (e.g., ages 3 to 30 years). Specifically, these study found that increases across early childhood giving way to later decreases following later childhood (Raznahan et al., 2011; Shaw et al., 2007).

Findings from surface area further supported hypothesis 1B by demonstrating marked increases from age 4 to 6 years. However, developmental trajectories of surface area from 6 to 8

years varied widely between regions with some demonstrating decelerating increases, some demonstrating a plateau, and others decreasing. As a result, most regions showed a non-linear surface area trajectory across early to middle childhood. These findings expand on previous literature that demonstrates early increases across early to middle childhood give way to later decreases (Ducharme et al., 2015; Mills et al., 2014; Raznahan et al., 2011; Remer et al., 2017; Wierenga et al., 2014). Importantly, this literature also shows that surface area peaks during middle to late childhood. Specifically, Wierenga used a longitudinal sample of 135 7- to 23-year-olds to show that the earliest peaks in surface area occurred around age 8 years for boys and around age 9 years in girls. However, findings from the present investigation suggest that some regions may reach peak cortical surface area between ages 6 and 8 years. Differences between these investigations are likely driven by the age range of the samples included. Specifically, Wierenga's sample may not have included children young enough to identify a peak prior to age 8 years.

In addition to Hypothesis 1B and Research Question 1A, this dissertation was able to investigate lateralization of developmental trajectories in cortical thickness and surface. Although, no priori hypotheses were made. Findings indicate that the development of cortical thickness is largely similar across hemispheres, with only a handful of exceptions. Specifically, mOFC and PHG both demonstrated asymmetric lateralized development from age 4 to 6 years. Whereas IPS and mOFC demonstrated significant asymmetric lateralized development from 6 to 8 years. In the younger cohort, findings deviate from past work that demonstrated leftward asymmetry in ACC along with rightward asymmetry in precuneus and LOC from age 1 to 6 years, but no differences in mOFC or PHG (Remer et al., 2017). In the older cohort, comparisons with previous literature are more difficult because lateralization from ages 6- to 8-years is under

investigated. Thus, future work should aim to understand whether findings presented here generalize across samples.

In contrast, cortical surface area demonstrates widespread lateralization, especially in the older cohort. In the younger cohort, ACC, ERC, and superior temporal gyrus exhibited asymmetric left lateralized development, whereas rostral middle frontal exhibited asymmetric right lateralized development. These findings are distinct from previous work examining surface area lateralization in a sample of 1- to 6-year-olds, that found left lateralization in superior frontal gyrus and mOFC. Though, consistent with the present investigation, there have been previous findings demonstrating left lateralization in ERC and superior temporal sulcus (Remer et al., 2017). Differences with Remer (2017) for both thickness and surface area may be attributed to the younger sample included in Remer (2017), as findings from that investigation largely overlap with findings in younger samples (Li et al., 2014, 2015; Nie et al., 2014; Remer et al., 2017). In the older cohort, IFG, ACC, middle temporal, and precuneus showed asymmetric left lateralized development. While IPS and LOC showed asymmetric right lateralized development. Like cortical thickness, investigations of lateralization in this age group are rare. Thus, future work should aim to better understand cortical lateralization in older children.

Another post hoc question that this dissertation assessed was whether there were similarities in developmental trajectories between cortical thickness and surface area. Past work on this topic suggests that the developmental trajectories of cortical thickness and surface area are dissimilar due to distinct underlying developmental mechanisms (Hutton et al., 2009; Li et al., 2013; Lyall et al., 2015; Wierenga et al., 2014). Specifically, changes in thickness are thought to reflect synaptogenesis and synaptic pruning (Huttenlocher, 1979), whereas changes in surface

are thought to reflect the division of neural stem cells and apoptosis (Cafiero et al., 2019; Rakic, 2009). Consistent with this literature, there were no associations between the developmental slopes of cortical thickness and surface area in 4 to 6 year olds. However, there was a negative association between developmental slopes of cortical thickness and surface area in both the left SPL and the left superior temporal gyrus from age 6 to 8 years, such that decreases in cortical thickness were associated with decreases in surface. This association provides preliminary evidence that there may be some similarities in developmental trajectories that might reflect overlapping mechanisms that contribute to the development of both cortical thickness and surface area. However, these overlapping developmental mechanisms may be specific to the developmental period being examined.

Memory Development

Consistent with Hypothesis 1C and Canada et al., (2021), the latent episodic memory variable demonstrated linear increases from 4 to 8 years old when employing a second-order latent growth model. These findings demonstrate that changes in the latent episodic memory variable translate from piecewise models to simpler latent growth models. As a result, these models allowed for the examination of co-development with cortical thickness and surface area in the episodic memory network.

Co-development of Latent Episodic Memory with the Episodic Memory Network

Associations between the Slope of ROIs with the Slope of Latent Episodic Memory.

Hypothesis 1D predicted that longitudinal changes in cortical thickness and surface area would be associated with longitudinal changes in memory performance. Consistent with this

hypothesis, ERC demonstrated negative co-development with memory performance from age 4 to 6 years, but positive co-development from age 6 to 8 years. In other words, improvements in memory performance were associated with thinning in the ERC from 4 to 6 years, but a thickening of the ERC from 6 to 8 years. This suggests that for younger children smaller is superior, but for older children bigger is better. These findings are somewhat surprising given that there were no significant changes in ERC in either the older or the younger cohort. Therefore, associations may be driven by individual differences. For example, some children may be experiencing significant thinning while others are not. Interestingly, similar trends have been demonstrated with hippocampus (Canada et al., 2019), suggesting that association between regions in the hippocampal complex and memory performance may differ as a function of age.

Importantly, ERC does play a very definitive role in supporting memory performance. Specifically, it is the final region in the “what” and “where” pathways that carries item and context-related information from sensory regions to the hippocampus. Thus, it synapses directly onto the hippocampus and plays a role in object and context memory (Eichenbaum et al., 2012; Wixted & Squire, 2011). Furthermore, previous work has demonstrated cross-sectional associations between ERC thickness and memory performance in 5 to 7 year old children (Schommartz et al., 2023). Together these findings could suggest that changes ERC support changes in memory performance across childhood.

In cortical surface area, changes in multiple regions were associated with memory performance from age 6 to 8 years. Specifically, there were negative associations of change in bilateral ERC, left ERC, and left superior temporal gyrus with change in latent episodic memory abilities. These findings suggest that decreases in surface area of these regions predict improvements in latent episodic memory performance. Furthermore, there were positive

associations between change in bilateral lingual gyrus, left ACC, left IPL, and right lingual gyrus with change in latent episodic memory during this same developmental period, such that increases in the surface area of these regions was related to increases memory performance.

Importantly, findings for left ACC are consistent with its observed developmental trajectory, indicating that surface area maturation of ACC predicts memory improvements. However, in all other regions (i.e., ERC, lingual gyrus, left IPS, and left superior temporal gyrus), findings are inconsistent with developmental trajectories. Specifically, left ERC and left superior temporal gyrus are undergoing significant increases during this developmental period even though they demonstrate negative associations with change in latent episodic memory performance. Furthermore, left IPS and right lingual gyrus are undergoing significant decreases during this developmental period even though they demonstrate positive associations with change in latent episodic memory performance. Thus, in these regions, findings suggest that regional maturation in surface area is associated with poorer memory performance. Though this seems unlikely.

One potential cause for this discrepancy is non-linearity within the older cohort. For example, surface area in the older cohort could demonstrate decreases from 6 to 7 years, then increases from 7 to 8, creating a U-shape trajectory. This type of trajectory has a negative slope from age 6 to 7 years that could be driving negative associations, though, the overall slope from 6 to 8 years can still be positive. Based on the freely estimated loading from the developmental models, this may be the case with left IPS and right lingual gyrus given that both regions feature negative freely estimate loadings, indicating non-linearity. As a result, negative associations may be driven by decreasing surface area from 6 to 7 years or from 7 to 8 years. Thus, future work

examining associations with surface area during this development period should consider using piecewise models if data allows.

Another potential cause is individual differences in development trajectories, such that some children demonstrate developmental increases and others demonstrate developmental decreases from 6 to 8 years. Given that left ERC and left superior temporal gyrus do not show evidence of extreme non-linearity within the older cohort, individual differences may be driving discrepancies between the co-development findings and the observed developmental trajectories in these regions. For example, some children could demonstrate early peaks in cortical surface area of these regions leading to decrease across middle childhood and thus negative associations with change in the latent episodic memory variable.

Associations Between the ROI Intercept and the Slope of Latent Episodic Memory.

Hypothesis 1E predicted that cortical thickness and surface area at age 4 and 6 years would predict longitudinal changes in memory performance across the following 2 years. Consistent with this hypothesis, results showed a negative association between the cortical thickness of ROIs at age 4 and improvements in memory performance across the following two years, such that a thinner (or more mature) cortex at age 4 predicted more rapid improvements in memory abilities. Furthermore, these effects were broad, spanning regions that overlap the DMN (i.e., precuneus, middle temporal gyrus, left temporal pole and right superior frontal gyrus), the DAN (i.e., SPL, left IPS), and the frontal control network (i.e., IFG). Additionally, there were associations with both the lingual gyrus and LOC, occipital regions that have previously been associated with the “replay” of encoded patterns during retrieval in adults (Alvarez & Squire, 1994; Karanian & Slotnick, 2015; Norman & O’Reilly, 2003; Wing et al., 2015). Importantly, all of these regions have previously been associated with memory performance cross-sectionally

during childhood or adolescence (Amlien et al., 2018; Bauer et al., 2019; Chad-Friedman et al., 2021; Fjell et al., 2019; Guillery-Girard et al., 2013; Klijn et al., 2016; Østby et al., 2012; Rosen et al., 2018; Schommartz et al., 2023; Sowell et al., 2001; Yu, McCall, et al., 2018). The widespread nature of these effects throughout the episodic memory network could indicate that memory development from age 4 to 6 years is supported by retrieval abilities associated with the DMN (Amlien et al., 2018; Miotto et al., 2020; Yu et al., 2018), encoding abilities associated with the DAN and frontal parietal control network (Miotto et al., 2020; Tang et al., 2018; Wendelken et al., 2011), and consolidation abilities associated with occipital regions (Alvarez & Squire, 1994; Karanian & Slotnick, 2015; Norman & O'Reilly, 2003; Wing et al., 2015) at age 4 years. Importantly, none of these findings survived correction for multiple comparison, although the majority did remain marginal.

In the older cohort, there were also associations between cortical thickness of ROIs at age 6 and improvements in memory performance across the following two years. However, findings were in the opposite direction, such that a thicker (or less mature) cortex at age 6 predicted more rapid improvements in memory abilities. Furthermore, these effects were far less broad compared to the younger cohort, only overlapping one region in the DMN (i.e., middle temporal gyrus) and one in the DAN (i.e., right IPS). Together these findings are likely indicative of individual differences at age 6 years. For example, children who have a less mature cortex in these regions at age 6 may undergo rapid improvements in memory development, from age 6 to 8 years as a form of cognitive catch-up. In contrast, average developmental trajectories beyond age 8 years could feature additional thickening and these children could be earlier developers. Unfortunately, the precise mechanism behind these associations is unclear. Thus, further work should aim to explore these relations in middle childhood.

For cortical surface area, similar results were observed in the younger cohort. However, the findings were far less broad. Specifically, only the surface area of right precuneus and right PHG at age 4 predicted better memory performance in the following 2 years. Importantly, with right precuneus suggested that greater surface area at age 4 predicted more rapid improvements in memory. In contrast, associations with right PHG suggest that less surface area at age 6 predicted more rapid improvements in memory performance. Interestingly, findings did not generalize to the older cohort. In comparison to cross-sectional findings, these results suggest that cortical thickness in the episodic memory network at age 4 may be a better predictor than surface area of change memory performance across the following 2-year developmental period. Whereas surface area in the episodic memory network may be a better predictor than cortical thickness of memory performance at the same timepoint in 4- to 8-year-old children.

Importantly, these findings are consistent with past work demonstrating that early assessments of the brain can predict later cognitive performance. Specifically, previous studies have found similar findings with IQ (Schnack et al., 2015), numerical abilities (Evans et al., 2015; Supekar et al., 2021), reading abilities (Hoeft et al., 2011), and memory (Canada et al., 2021; Geng et al., 2021). For example, Canada and colleagues (2021) found that hippocampal subfield volumes at age 6 predicted improvements in source memory performance from age 6 to 7 years. Similarly, Geng and colleagues (2021) demonstrated that hippocampal connectivity at 6 predicted better memory performance at age 8 years. However, no previous examination has found that brain structure or function at age 4 predicts memory performance from age 4 to 6 years. In sum, this literature and findings from the present dissertation could suggest that certain levels of brain maturation are required to “set the stage” for cognitive development.

In combination with findings from Hypothesis 1A (i.e., cross-sectional associations) and Hypothesis 1D (i.e., associations between slopes), these findings demonstrate that most regions do not demonstrate both cross-sectional and longitudinal associations with memory performance. One notable exception is the surface area of right precuneus. Specifically, surface area in right precuneus at timepoint 1 predicted memory performance at the same timepoint (i.e., cross-sectionally) in 4- to 8-year-olds and change in memory performance over a two-year period in 4- to 6-year-olds (i.e., longitudinally). These findings suggest that associations with between precuneus and memory performance are robust to cross-sectional and longitudinal associations. However, five other ROIs that demonstrated cross-sectional association did not demonstrate longitudinal associations (i.e., surface area in left mOFC, right ACC, left SPL, right rostral middle frontal gyrus, and thickness in right ACC). Additionally, several other regions demonstrated longitudinal associations, but not cross-sectional associations. These findings expand on previous work that suggests cross-sectional and longitudinal analyses sometimes implicate different ROIs (Keresztes et al., 2022). This phenomenon is likely driven by nuances in developmental trajectories and individual differences.

Associations Between the Latent Episodic Memory Intercept and the Slope of ROI.

This dissertation did not make an a priori hypothesis about the association between initial memory performance and changes in ROIs. Thus, the analyses are exploratory. Findings demonstrated that better memory performance at age 4 predicted less cortical thinning in two regions (i.e., right mOFC and right rostral middle frontal) from age 4 to 6 years. In older children, findings were in the opposite direction. Specifically, better memory performance was associated with more thinning in bilateral ERC from age 6 to 8 years. Together, findings demonstrated that better memory performance at timepoint 1 predicted changes in the episodic

memory network over the following two years in both the younger and older cohort.

Furthermore, there were no such associations with surface area suggesting that findings are specific to cortical thickness. Though, no association with cortical thickness survived correction for multiple comparisons.

These findings are somewhat consistent with past work examining associations between initial memory performance and later brain development (Geng et al., 2021). Specifically, Geng et al., (2021) found that memory performance at age 4 and age 6 predicted hippocampal connectivity 1 year later. The authors interpreted this to mean that there is a bi-directional association between memory and brain development. Specifically, it was proposed that early memory experiences lead to later changes in brain function which ultimately results in greater change in memory performance. Findings from the present investigation demonstrate a similar pattern of results in older children. Specifically, in this dissertation, latent episodic memory performance at age 6 predicted greater maturation in cortical thickness of ERC across the following two years. This phenomenon may suggest that better memory performance readies the brain to be shaped by experience leading to long term differences in brain structure and function.

In contrast, this literature may not explain results in 4- to 6-year-old children. Specifically, in contrast to the older cohort, associations were positive in the younger cohort, such that better memory performance at age 4 was associated with less cortical thinning from 4 to 6 years. However, surface area is undergoing significant increases during this development period. One possible explanation is that in early childhood, children who have less mature memory abilities may experience neuronal catch up in these regions. However, this is speculative and future work should aim to untangle these complex associations.

What do these associations mean for memory?

Importantly these findings have implications for our understanding of memory development. Specifically, all regions used in this investigation can be organized into several categories based on underlying memory abilities. For example, some regions are associated with memory encoding (e.g., regions belonging to the frontal control network and DAN), some with retrieval (e.g., regions belonging to the DMN), some with consolidation (e.g., lingual gyrus and LOC), and some directly support the hippocampus with memory binding (e.g., ERC and PHG). Thus, associations between these regions and changes in episodic memory abilities during early to middle childhood may provide insight into the underlying memory processes that support memory development. In this section, I will discuss how these findings can inform the field's view of memory development in early to middle childhood.

In the younger cohort, greater structural maturity (i.e., a thinner cortex) in regions that support encoding, retrieval, and consolidation at age 4 was associated with better latent episodic memory improvements from age 4 to 6 years. These findings suggest that initial encoding, retrieval, and consolidation abilities, that are developed prior to age 4 years, may support improvements in memory performance during early childhood. However, these results do not provide evidence that improvements in encoding, retrieval, and consolidation abilities actively drive improvements in memory during early childhood. To support this assumption, there would have to be evidence that changes in these regions are associated with changes in memory performance.

One region that did meet this assumption was ERC. Specifically, changes in cortical thickness and surface area of the right ERC were associated with changes in episodic memory during early childhood. Notably, ERC carries item and context-related information to the

hippocampus making it critical to memory binding (i.e., the gluing together of event-related contextual details). Furthermore, ERC has previously been directly associated with memory-binding abilities (Schommartz et al., 2023). Thus, these findings might suggest that brain development in regions that support encoding, retrieval, and consolidation prior to age 4 set the stage for memory development during early childhood, but binding abilities supported by the hippocampal complex actively drive changes in memory performance from 4 to 6 years.

In contrast, findings from the older cohort showed that regions that support encoding (e.g., IPS), retrieval (e.g., ACC), consolidation (e.g., lingual gyrus), and binding (e.g., ERC) all demonstrate significant associations between ROI and memory slopes. In other words, changes in these regions were associated with changes in episodic memory performance from age 6 to 8 years. These results provide evidence that changes in encoding, retrieval, consolidation, and binding abilities during middle childhood are supporting improvements in episodic memory abilities during middle childhood.

Taken together, findings from younger and older cohorts could suggest that improvements in episodic memory performance during early childhood are primarily driven by binding abilities subserved via the MTL, whereas improvements in episodic memory performance during middle childhood are driven by improvements in encoding, retrieval, consolidation, and binding abilities via changes in the broader cortical network. Importunately, these findings build on previous theoretical work suggesting that improvements in memory during middle childhood are mainly driven by increases in top-down strategic encoding and retrieval abilities that are supported by regions in the PFC (Ghetti & Fandakova, 2020; Miotto et al., 2020; Shing et al., 2010; Yu, McCall, et al., 2018). Furthermore, these results also build on

this idea by demonstrating that early childhood changes are mainly supported by increasing associative abilities that are supported by the MTL (e.g., ERC).

Specificity of Regional Findings

The present study utilized total gray matter volume to assess whether associations between ROIs and episodic memory were region specific. In the cross-sectional sample, findings between the composite memory variable and total gray matter volume were significant. However, after controlling for age and sex, relations disappeared. This suggests that associations of memory with total gray matter are indirect and accounted for by a common associations with age-related maturation. Furthermore, when age-related changes are included as a competent of model design, results demonstrate no significant associations between memory and total gray matter volume. Specifically, when using bivariate growth models, total gray matter volume does not predict memory performance cross-sectionally or longitudinally. Together these findings suggest that associations between memory and ROIs in the episodic memory network are region specific.

Differences in Cortical Thickness and Surface Area Based on Nap Habituality

Hypothesis 2A predicted that there would be differences in cortical thickness and surface area of ROIs that longitudinally co-development with memory performance based on nap status during early childhood. As hypothesized, there were differences in two regions whose initial structure predicted change in memory performance from age 4 to 6 years. Specifically, non-nappers demonstrated significantly greater cortical surface area in LOC than both intermediate nappers and non-nappers. Additionally, non-nappers and intermediate nappers demonstrated significantly thinner cortical thickness in IFG than habitual nappers. Importantly, follow up

analysis with total gray matter volume revealed that these associations are region specific, as total gray matter volume does not differ based on nap habituality. Furthermore, there were no associations between these ROIs and hippocampal volumes suggesting that differences based on nap status are not attributed to a common association with hippocampal structure. However, findings in the ROI analysis and the whole brain analysis did not survive correction for multiple comparisons.

In combination with findings from Hypothesis 1B that assessed developmental trajectories of cortical thickness and surface area, these results could suggest that nappers demonstrate significantly less mature measures of LOC and IFG compared with non-nappers. This pattern of results is consistent with previous findings in hippocampal subregions (Allard et al., Under Review) and hippocampal subfields (Riggins & Spencer, 2022), that demonstrate habitual napping is associated with variations in structures in the episodic memory network. Together, these findings suggest that nap habituality is associated with maturation of regions that support memory, and that these findings are likely region specific. Importantly, these results are novel as no study to date has investigated cortical differences based on nap habituality. Therefore, this investigation provides the first evidence that associations between nap habituality and the brain extend beyond the hippocampus. However, no significant findings survived correction for multiple correction. This was likely accounted for by the small sample size ($N = 44$). Thus, future analyses are required to confirm these highly exploratory findings.

Limitations

While Study 1 allowed for the longitudinal assessment of co-development between memory and the brain, the sample only included 1 overlapping timepoint between cohorts. This makes it especially difficult to characterize the nature of change (e.g., linear, quadratic, cubic, or

even discontinuous) and may reduce the ability to properly characterize nuance in developmental trajectories. Additionally, Study 1 only included 3 timepoints within each cohort. This severely limits longitudinal modeling capabilities, especially when 2nd order variables are not theoretically supported. Specifically, samples with at least 4 timepoints allow for the assessment of non-linearity within cohorts, result in better estimates of growth parameters, have improved model fit, and have greater statistical power (Duncan & Duncan, 2009; Whittaker & Khojasteh, 2017). Thus, future studies examining co-development should strive to utilize a cohort sequential design with least two overlapping timepoints between cohorts and 4 or more time points within cohorts. Although this may be challenging, increasing the number of measured timepoints and overlapping timepoints will result in a better understanding of developmental change.

For Study 2, the sample size was small and resulted in limited power to detect structural differences based on nap habitually. Additionally, the sample used in Study 2 was cross-sectional. Therefore, the direction of effects cannot be determined. Specifically, it is unclear whether changes in the brain precede changes in nap habits or if changes in nap habits precede changes in the brain. Thus, future work should utilize a larger longitudinal sample to assess these questions.

Future directions

There are several future directions for this work. In this section, three are highlighted. First, future studies should utilize piecewise models to better examine associations between memory and brain development. These models estimate multiple slopes within a single cohort allowing for assessment from 4 to 5 years, 5 to 6 years, 6 to 7 years, and so on. As a result, piecewise models would allow for a more nuanced understanding of co-development between memory and the brain. Second, future investigations of nap habituality should aim to examine

longitudinal associations between nap habits, memory, and the brain to establish the direction of effects. Specifically, findings from this investigation suggest that nap habituality in 3- to 5-year-olds is associated with brain regions that predict change in memory performance across early childhood. Unfortunately, any interpretation of these findings must be tentative because it is unclear whether changes in the brain preempt changes in nap habituality or if changes in nap habituality preempt changes in the brain. A longitudinal investigation with a larger sample could determine the direction of these effects. Third, findings from aim 2 are the first step to understanding associations between the cortex and nap habits during early childhood. However, research in this area is still extremely lacking. While past work has examined other measures of brain structure (Allard et al., Under Review; Riggins & Spencer, 2020), there are currently no examinations of brain function or hippocampal connectivity. Thus, these are clear future directions for the field.

Conclusions

Findings from this investigation suggest that early childhood is a period marked by rapid developmental changes in the episodic memory network that support changes in episodic memory performance. Specifically, change in memory performance during early childhood was linked to structural assessments of the episodic memory network at age 4 and change in the episodic memory network from age 6 to 8 years. Additionally, findings show that associations are broad, spanning regions that have previously been implicated in encoding, retrieval, and consolidation. However, none of these co-development findings survived correction for multiple comparisons. Furthermore, results suggest that individual differences are important. We explored one individual difference, specifically nap habits, and found that nap habituality may be associated with structural differences in regions that support change in memory performance.

Namely, non-nappers demonstrate more mature patterns of cortical thickness and surface area in right IFG and LOC when compared to habitual nappers. However, again, no findings survived correction for multiple comparisons. Thus, future work is needed to verify these results and explore other individual differences that may contribute to variability between children. In summary, findings from this dissertation provide new insight about the role of the cortical episodic memory network in memory development and identifies potential individual differences (i.e., nap habituality) that are associated with these brain structures.

Appendix A: Supplementary Table(s)

Table 31

Hemispheric Differences in Cortical Thickness and Surface Area of Selected ROIs

Hemispheric Differences in ROI Slopes	Cortical Thickness		Surface Area	
	Younger Cohort	Older Cohort	Younger Cohort	Older Cohort
Inferior Frontal Gyrus	-0.101	0.282	0.714	-1.599
Posterior Cingulate Cortex	-1.408	0.141	-0.044	0.315
Anterior Cingulate Cortex	-0.118	0.248	-1.320	-3.787***
Entorhinal Cortex	0.646	0.490	-1.664*	-1.192
Inferior Parietal Sulcus	0.781	-1.768*	0.746	-3.835***
Lateral Occipital Cortex	-0.564	-0.300	-2.371*	-2.033*
Lingual Gyrus	-0.514	1.193	0.038	-0.438
Medial Orbitofrontal Cortex	-3.969***	-2.325*	0.171	-2.112*
Middle Temporal	0.141	-0.808	0.173	-2.588**
Parahippocampal Gyrus	-2.049*	-0.941	-0.446	-0.569
Precuneus	0.000	-0.849	0.079	2.694**
Rostral Middle Frontal	-1.035	0.901	1.776*	3.043**
Superior Frontal Gyrus	-0.404	-1.302	0.580	-0.323
Superior Parietal Lobule	0.000	-0.943	-0.472	-0.836
Superior Temporal Gyrus	0.424	0.000	-2.846**	-2.543**
Temporal Pole	0.542	0.712	0.516	0.338

Note. Degrees of freedom were 162 for the younger cohort and 198 for the older cohort. Significant parameters are bolded. * $p < .05$. ** $p < .01$. *** $p < .001$.

Appendix B: Literature Review

Across childhood and into early adulthood there are marked improvements in episodic memory performance (Allard et al., 2023; Canada et al., 2020, 2022; Lee et al., 2016; Picard et al., 2012; Riggins, 2014; Yim et al., 2013) that are supported by hippocampal development (Botdorf et al., 2022; Canada et al., 2021; Lee et al., 2020). However, work in adults has demonstrated that episodic memory is *also* supported by a broad network of cortical subregions in the medial temporal lobe (MTL), prefrontal cortex (PFC), parietal cortex, and occipital cortex (e.g., Brewer et al., 1998; Buckner et al., 1999; Davachi et al., 2003; Lavenex & Amaral, 2000; Nyberg et al., 2000; Stern et al., 1996; Stevens et al., 2008; Wagner et al., 1998). Like the hippocampus, these regions undergo protracted structural development during childhood (Frangou et al., 2022; Gogtay et al., 2004; Lenroot et al., 2007; Raznahan et al., 2011; Tamnes et al., 2017; Y. Wang et al., 2019) and may also play a role in the development of episodic memory abilities. Although limited, some work in developing populations has supported this notion by demonstrating cross-sectional relations between memory performance and these extrahippocampal cortical regions during childhood (Ghetti & Bunge, 2012; Østby et al., 2012; Schommartz et al., 2023). For example, Schommartz and colleagues (2023) recently found an association between better episodic memory performance and thinner cortical thickness in 12 extrahippocampal regions in an early childhood sample of 5 to 7-year-olds. All 12 of the regions have previously been implicated in memory in adults, suggesting that the broad memory network supporting adult-like memory may be present during childhood. The thesis of this review is that development of these regions contributes to variations in memory improvement across childhood.

During early childhood, just before children demonstrate significant changes in memory and brain development, they also transition from biphasic sleep to monophasic sleep (i.e., cessation of the afternoon nap; Staton et al., 2020). These developmental shifts in memory, sleep, and the brain are likely related. Specifically, some have theorized that habitual nappers may require more regular sleep due to an inefficiency in sleep-dependent memory consolidation caused by an immature episodic memory network (Lokhandwala & Spencer, 2022; Mason et al., 2021; Spencer & Riggins, 2022). Empirical evidence in early childhood supports this hypothesis. For example, habitual nappers do worse on an episodic memory task following a wake period compared to their non-napping counterparts (Kurdziel et al., 2013) and they demonstrate variations in hippocampal maturity (Riggins & Spencer, 2020).

The purpose of this review is twofold. The first aim is to summarize literature examining associations between episodic memory performance and the structural development of brain regions that support memory during childhood. The second is to summarize literature examining associations between nap habitually, memory, and brain development during early childhood.

Before moving forward, I will define the scope of this review, with the aim of motivating the specific questions posed in my dissertation. Specifically, I will first describe the developmental trajectory of episodic memory performance, structural brain maturation, and sleep habits across childhood (e.g., age 3 to 12 years). Importantly, studies with adults will be used to provide theoretical context, however, investigations of infancy, toddlerhood, and adolescence are beyond the scope of this review (see instead Galván, 2020; Johnson et al., 2020; Kopasz et al., 2010; Mason et al., 2021).

Next, I will examine associations between memory and structural measures of brain maturation, including cortical thickness and surface area. These measures are thought to reflect

synaptogenesis, gyrification, and myelination in cortical grey matter (Cafiero et al., 2019; Huttenlocher, 1979; Rakic, 2009). Therefore, they are proxies for neural development that are often regionally associated with memory performance during childhood (e.g., Schommartz et al., 2023). Importantly, this review will not directly focus on measures of brain function because literature using these methods is quite limited in this age range. This may be because functional examinations of memory are challenging to acquire in childhood, and nearly impossible to acquire in early childhood (e.g., ages 3 to 6 years). Furthermore, brain structure and function develop on distinct time scales for most brain regions (Gilmore et al., 2018). As a result, developmental associations that exist with functional MRI may not appear with structural MRI and vice versa. Thus, as the questions posed in my dissertation focus on brain structure, that was the focus here (for a review of functional work see Ghetti & Bunge, 2012 in children & Spaniol et al., 2009 adults).

Finally, I will review relations between sleep habits, memory, and the brain. Specifically, sleep behaviors will include the effects of an afternoon nap, nap habitually, and sleep duration due to previously demonstrated associations between these variables, memory, and memory-related brain regions (see Cheng et al., 2021; Esterline & Gómez, 2021; Hansen et al., 2022; Kurdziel et al., 2013; Kurdziel et al., 2018; Riggins & Spencer, 2020; Taki et al., 2012). It is worth noting that there is a burgeoning body of work examining the association between memory development, the brain, and sleep physiology (e.g., sleep architecture and microstructure). However, these measures are beyond the scope of this review and will not be covered (see Lokhandwala & Spencer, 2022). I will conclude by summarizing gaps in the literature and providing recommendations for future research.

Development of Episodic Memory:

Fifty years ago, Tulving (1972) suggested that the ability to remember past events is a distinct form of declarative memory. Specifically, he noted that episodic memory is different from semantic memory due to its auto-noetic quality that requires a person to perform mental time travel (Tulving, 1984, 2002). Thus, episodic memories, unlike semantic memories, are specific and detailed, including information about event content (i.e., what), location (i.e., where), and timing (i.e., when). Neuroimaging evidence in adults supports these claims and demonstrates that episodic memories have separable neural correlates from other forms of memory (see Eichenbaum et al., 2007; Tulving, 2002).

Episodic memory also has a distinct order of operation. First, sensory information is encoded to short-term or working memory through focused attention. Then, rehearsal of that information results in consolidation to long term storage until it is either forgotten or retrieved (Atkinson & Shiffrin, 1968). Importantly, early literature in memory development proposed that infant and childhood amnesia were a result of an inability to recall objects and events (Piaget, 1952). However, developmental science research dispelled this myth by demonstrating that children as young as 9 months of age can retrieve events on a delayed imitation task (Meltzoff, 1988). Further, more recent work suggests that all steps of the Atkinson & Shiffrin model are in place during early childhood, with improvements in memory being driven by developments in multiple aspects of memory including encoding, consolidation, and recall (see Lukowski & Bauer, 2013 for an in depth review of this topics). These developmental processes are relevant because research demonstrates that memory-related brain regions uniquely contribute to each stage of this memory model (e.g., Eichenbaum, 2017; Ghetti & Fandakova, 2020).

Development of Underlying Episodic Memory Abilities

Episodic memory, unlike other forms of memory (e.g., semantic memory and implicit memory), is thought to be a recent evolutionary development. As a result, it is comparatively late developing, early deteriorating, and more sensitive to neural deficits (Tulving, 2002). Both cross-sectional and longitudinal research has demonstrated that early to late childhood is a time of marked improvements in episodic memory abilities (see Ghetti & Lee, 2011 and Shing & Lindenberger, 2011 for review). Specifically, past work shows that across childhood, children are able to remember more event related details (Ghetti & Bauer, 2012). However, episodic memory, like other cognitive processes (e.g., executive functioning), cannot be measured directly because no cognitive task can fully capture the complexity of underlying abilities that support episodic memory. Thus, studies of episodic memory development often aim to measure one or more component parts. For example, episodic memory tasks often assess feature binding (i.e., the ability to glue together contextual event details), temporal order memory (i.e., memory for event order), and pattern separation (i.e., the ability to separate between similar events or items). Previous studies have demonstrated improvement in all of these memory abilities across childhood (Allard et al., 2023s; Canada et al., 2019, 2020, 2022; Cheke & Clayton, 2015; Drummey & Newcombe, 2002; Lee et al., 2016; Lloyd et al., 2009; Lorscheid & Reimer, 2005; Ngo et al., 2017; Riggins, 2014; Sluzenski et al., 2006).

Importantly, not all episodic memory abilities develop at the same rate. Some demonstrate linear trajectories (i.e., temporal order memory; Canada et al., 2020), whereas others show non-linear trajectories (i.e., feature binding; Allard et al., 2023). Furthermore, different developmental trajectories can be observed within the same task depending on the variable assessed (Allard et al., 2023; Lee et al., 2020). For example, one study found that hit rates (i.e.,

the ability to accurately identify a previous connection as old) on a binding task are relatively early developing, while false alarm rates (i.e., the inability to recognize a novel connection as new) are relatively late developing (Allard et al., 2023). Although these tasks are all thought to reflect the same underlying memory process, these findings show that there is no one-to-one correlation between a task and the episodic memory system. As a result, varying demands from multiple other cognitive (or memory-specific) systems may lead to conflicting developmental trajectories. For example, these tasks might also reflect attention, executive functions, verbal, and spatial abilities that may exert a unique effect on developmental trajectories.

Development of Latent Episodic Memory

One solution to the variability introduced by differing memory tasks is to use data reduction techniques to create a latent variable from a variety of assessments. This is a technique that allows the experimenter to isolate shared variance across tasks resulting in a variable that is closer to a pure measure of episodic memory than any single task on its own. In children, a handful of studies have successfully utilized these techniques to assess associations between latent episodic memory and age (Canada et al., 2022; Cheke & Clayton, 2015). These studies have demonstrated that a battery of episodic memory tasks featuring surface differences can be used to create a latent measure of episodic memory in children as young as 4 years (Canada et al., 2022). Further, results from a longitudinal study show that these latent measures of episodic memory undergo linear age-related increases from early to middle childhood (i.e., 4 to 8 years; Canada et al., 2022).

Development of brain regions that support memory:

Several regions, both cortical and subcortical (e.g., hippocampus, MTL, parietal cortex, PFC, and occipital cortex), support aspects of memory performance across the lifespan (i.e., Ghetti & Bunge, 2012; Schommartz et al., 2023). Further, previous research demonstrates that all of these regions undergo some form of change across childhood, with varying trajectories (Canada et al., 2020, 2021; Frangou et al., 2022; Gogtay et al., 2004; Wang et al., 2019). Some work has even suggested that changes in brain morphology may also support changes in memory performance (e.g., Canada et al., 2021). To better understand associations between these regions and changes in memory performance, I will review changes in brain maturation from early childhood through adulthood.

Changes in Cortical Maturation

Cortical gray matter is responsible for most neuronal function because it contains the brain's neural synapses and cell bodies. Unsurprisingly, total gray matter undergoes extensive maturation across early to late childhood. Specifically, past work demonstrates that total gray matter volume increases until age 7 years and then decreases into adulthood (Giedd et al., 1999; Sowell et al., 2001). That said, gray matter volume is the product of two distinct and commonly used measures, cortical thickness and cortical surface area, that have been implicated in both the memory and sleep literature. These two measures play a distinct role in development and are phenotypically unique (Winkler et al., 2010). Specifically, early increases in gray matter are thought to reflect synaptogenesis, while cortical thinning following the onset of puberty is thought to reflect synaptic pruning (Huttenlocher, 1979). In contrast, changes in cortical surface area are thought to reflect folding and gyrification due to the division of neural stem cells and intra-cortical myelination (Cafiero et al., 2019; Rakic, 2009). As result, measures of cortical thickness and

surface area do not co-vary (Cafiero et al., 2019; Im et al., 2008) and they demonstrate unique developmental trajectories (Hutton et al., 2009; Lyall et al., 2015; Wierenga et al., 2014).

Research has demonstrated non-linear changes in cortical thickness across childhood with rapid early increases, followed by gradual thinning into adulthood (Lenroot et al., 2007; Ofen et al., 2007; Raznahan et al., 2011; Tamnes et al., 2017). However, it is debated when cortical thickness reaches its peaks (Tamnes et al., 2017; Walhovd et al., 2017). Specifically, some suggest that cortical thickness peaks around latter childhood (e.g., 8 to 10 years; Lenroot et al., 2007; Raznahan et al., 2011; Shaw et al., 2007). While, others demonstrate regional thinning during early to middle childhood (Ducharme et al., 2016; Gogtay et al., 2004; Mills et al., 2014; Wierenga et al., 2014). This is further complicated by the fact that development of cortical thickness is nonuniform. In other words, regions demonstrate maturation at distinct rates. Specifically, cortical thinning begins in the dorsal parietal cortices and then spreads rostrally, caudally, and laterally over the frontal, occipital, and temporal cortices (Gogtay et al., 2004). Importantly, while a consensus about when cortical thickness peaks has not been reached, a meta-analysis including over 17,000 subjects aged 3 to 90 years demonstrated that most cortical regions appear to exhibit the greatest thickness during childhood (e.g., 3 to 10 years) except for the entorhinal cortex, the temporopolar cortex, and the anterior cingulate cortices which peak in middle adulthood (Frangou et al., 2022; Wang et al., 2019).

Like cortical thickness, cortical surface area demonstrates a nonlinear trend, with early childhood increases into middle-late childhood eventually giving way to later adolescent decreases (Ducharme et al., 2015; Mills et al., 2014; Raznahan et al., 2011; Wierenga et al., 2014). However, unlike cortical thickness, most agree that cortical surface area peaks in late childhood (i.e., age 8 to 10 years). Several studies have also demonstrated that surface area consistently peaks later than

cortical thickness (Raznahan et al., 2011; Wierenga et al., 2014). Importantly, cortical surface area, like cortical thickness, demonstrates non-uniform subregion development (Ducharme et al., 2015; Ofen et al., 2007; Raznahan et al., 2011; Tamnes et al., 2017; Wierenga et al., 2014). For example, one longitudinal investigation of early childhood (i.e., 5 years) through young adulthood (i.e., 22 years) found that many regions demonstrate no change in surface area across childhood. Further, of the regions that did demonstrate change, some (e.g., right temporal lobe, right rostral middle frontal cortex, right frontal old, right OFC, right PCC, and left pars triangularis) showed non-symmetric cubic trajectories, some (e.g., dlPFCm lateral and medial frontal lobe, and ACC) showed bi-lateral quadratic trajectories., and some (e.g., superior temporal gyrus, precuneus, and occipital lobe; Ducharme et al., 2015) showed linear decreases. That said, most regions peak between late childhood and early adolescence (i.e., 8 to 13 years; Ducharme et al., 2015; Wierenga et al., 2014).

Importantly, nonlinear trajectories in cortical thickness and surface area could impact brain-behavior relations in complex ways. Specifically, greater cortical thickness or surface area may be associated with better memory performance in younger children. Whereas cortical thinning or smaller surface area may be associated with better memory performance in older children. Moreover, the age when this shift occurs could differ based on the brain region and measure used. Thus, in the same child, better memory performance may be associated with both thinner parietal regions and thicker prefrontal regions, depending on their developmental age.

Sex Differences. Past work has demonstrated that there are sex-based differences in the development trajectories of cortical thickness and cortical surface area (Lenroot et al., 2007; Raznahan et al., 2011; Wierenga et al., 2014; see Kaczkurkin et al., 2019 for review). For example, both males and females show a U-shaped curve in the development of cortical

thickness from early childhood to adulthood. However, females demonstrate comparatively accelerated growth, peaking approximately 1 to 2 years before males (Lenroot et al., 2007). Findings in cortical surface area demonstrate a similar trend, with surface area peaking approximately 1 year earlier in females (Raznahan et al., 2011). However, these effects are not equal across cortical measures. Specifically, Wierenga (2014) found that cortical surface area is more susceptible to sex differences than cortical thickness.

Importantly, many have suggested that sex-based differences in cortical development derive from hormonal shifts during puberty (Kaczurkin et al., 2019; Lenroot et al., 2007kar). However, studies have suggest that sex-based differences in cortical thickness and cortical surface area appear during early childhood suggesting that these differences are present prior to the onset of puberty (Kaczurkin et al., 2019; Raznahan et al., 2011; Wierenga et al., 2014). Thus, although puberty likely plays a role in sex-based differences in developmental cortical trajectories, it cannot account for all differences as brain-behavior associations may also differ based on sex during early childhood.

Changes in Hippocampal Development

The hippocampus undergoes developmental changes across early childhood. This has been demonstrated in both longitudinal (Canada, Botdorf, et al., 2020; Canada et al., 2021; Dick et al., 2022; Østby et al., 2012; Tamnes et al., 2013) and cross-sectional studies (DeMaster et al., 2014; Tamnes et al., 2018), though findings using these two methodologies have been shown to be in conflict (Canada et al., 2021; Keresztes et al., 2022; Riggins et al., 2018). Past work has demonstrated contradictory trajectories in total hippocampus with some studies demonstrating longitudinal decreases (Tamnes et al., 2013) and others demonstrating longitudinal increases in volume after age 4 (Østby et al., 2012). That said, these relations may be accounted for by a more

complex developmental trajectory. Specifically, several cross-sectional and longitudinal studies suggest that the developmental trajectory of total hippocampus is actually cubic, with volumes peaking between late childhood and early adolescence, then decreasing into adulthood (Brown & Jernigan, 2012; Canada, Botdorf, et al., 2020; Dick et al., 2022; Tamnes et al., 2018).

Hippocampal Subregions. Importantly, the hippocampus is a non-homogeneous structure that can be divided using multiple methods. One of the most common methods is to divide the hippocampus along the longitudinal axis to create subregions (i.e., head, body, and tail; Poppenk et al., 2013). These subregions are thought to have functional significance that differ based on their structural connections with other cortical brain regions (see Amaral & Lavenex, 2007; Duvernoy, 2005; Poppenk et al., 2013; Poppenk & Moscovitch, 2011; Small, 2002; Strange et al., 2014). Furthermore, past work has suggested that these subregions show distinct developmental trajectories (Canada, Botdorf, et al., 2020; DeMaster et al., 2014; Riggins et al., 2018; Tamnes et al., 2018). Thus, this section will aim to characterize change in hippocampal subregions across childhood.

In hippocampal head, several cross-sectional studies have demonstrated that age is a significant predictor of volume (DeMaster et al., 2014; Lee et al., 2020; Riggins et al., 2018; Schlichting et al., 2017). Specifically, findings suggest that there are age-related increases in hippocampal head volumes from age 4 through adolescence. Furthermore, Schlichting and colleagues (2017) demonstrated that these associations are likely non-linear, with hippocampal volumes peaking in adolescence and then decreasing into young adulthood, similar to total hippocampus. This is consistent with longitudinal accounts of 4 to 8-year-old children that show non-linear increases in hippocampal head volumes that slow with age and eventually decrease

(Canada, Botdorf, et al., 2020; Lee et al., 2020). In sum, all studies agree that hippocampal head volumes increase from early to late childhood.

In the hippocampal body, cross-sectional and longitudinal findings are conflicting in early to middle childhood samples. Specifically, a cross-sectional study in children aged 4 to 8 years showed no age-related associations between age and volumes (Riggins et al., 2018). In contrast, a longitudinal examination of the same sample demonstrated that hippocampal body volumes show a slight, but significant increase from 4 to 8 years (Canada et al., 2020). Together these findings could suggest that cross-sectional studies are not always sensitive enough to detect increases in hippocampal body volumes during early to middle childhood. In samples of older children (8 – 12 years), the hippocampal body shows quadratic effects from childhood into adulthood, increasing until late childhood and then decreasing into adolescents (Daugherty et al., 2016; Lee et al., 2020; Schlichting et al., 2017). Thus, hippocampal body volumes increase from early to late childhood and then decrease into adolescence.

In the hippocampal tail, Riggins et al., 2018 and Canada et al., 2020 demonstrates a similar pattern of results to the hippocampal body during early to middle childhood. Specifically, cross-sectional findings demonstrate no age-related change from 4 to 8 years (Riggins et al., 2018), whereas longitudinal findings demonstrate slight but significant increases. This could suggest that hippocampal tail volumes are undergoing increases during early childhood that are not detectable by cross-sectional examinations. That said, a longitudinal study in older children (i.e., aged 8 to 11 years) found no development changes from 8 to 11 years (Lee et al., 2020). In combination, these studies provide evidence that hippocampal tail volumes increase from early to middle childhood and then stagnate.

Together these findings not only demonstrate that hippocampal subregions develop at distinct rates and trajectories, but also that cross-sectional and longitudinal investigations often yield unique results. These findings are consistent with a recent study that demonstrated disparate age-associations between cross-sectional and longitudinal analysis in the hippocampus. Specifically, cross-sectional findings were less like to reflect differences where longitudinal analysis demonstrated change (Keresztes et al., 2022). Thus, longitudinal studies of brain development are generally preferred. However, it may also be beneficial to examine both cross-sectional and longitudinal findings in the same sample to better understand how findings differ (Keresztes et al., 2022).

Hippocampal Subfields. Another common method for hippocampal division is via functional subunits that are defined by unique cell types and layers. They are dentate gyrus (DG) cornu ammonis (CA1-CA4), and subiculum (Lavenex & Banta Lavenex, 2013; Yassa & Stark, 2011). These regions provide additional insight into the role of the hippocampus in memory development across childhood. However, due to the high-resolution scan needed to examine these subfields and concerns with motion, most research in this area has compared older children to adults (Daugherty et al., 2016, 2017; Schlichting et al., 2017; Tamnes et al., 2018). Fortunately, a smaller number of recent studies have also examined early childhood (Canada et al., 2021; Riggins et al., 2018). As a result, this section aims to characterize changes in hippocampal subfields from early to late childhood.

Hippocampal subfields are unequally distributed along the longitudinal (i.e., anterior-posterior) axis of the hippocampus. Thus, these regions are often examined separately within subregions (Canada et al., 2021; Riggins et al., 2018; Schlichting et al., 2017). Though previous studies have also collapsed across the head and body (Tamnes et al., 2018) or examined a few

slices in the body only (Daugherty et al., 2016, 2017). This is because subfields are difficult to delineate in the hippocampal head and tail (Yushkevich et al., 2015). This results in conflicting developmental trajectories within subfields. Therefore, I will distinguish between these types of examinations.

In studies that delineate subfields based on subregions, findings across studies are consistent, with a few exceptions. Specifically, cross-sectional and longitudinal studies agree that in the hippocampal head, CA1 increases through early childhood, then decreases into adulthood (Canada et al., 2021; Daugherty et al., 2016; Riggins et al., 2018; Schlichting et al., 2017). In the hippocampal body, subiculum appears to demonstrate a similar trajectory, increasing through early childhood and then decreasing into adulthood (Canada et al., 2021; Schlichting et al., 2017). However, some conflicting *cross-sectional* evidence suggests that subiculum does not demonstrate age related differences after age 4 (Daugherty et al., 2016; Riggins et al., 2018).

In contrast to CA1 and subiculum, dentate gyrus (DG) is later developing and therefore exhibits more protracted growth. Specifically, in the hippocampal body, a longitudinal study found that CA2-4/DG volume increases from age 5 to 7 years (Canada et al., 2021). Similarly, a cross-section study examining associations between age and CA2-4/DG volume in the body found a positive association in subjects aged 6 to 30 years (Schlichting et al., 2017) suggesting that volumes increase across childhood and into adulthood. Importantly, Daugherty and colleagues (2016) found a conflicting negative association in subjects aged 8 to 82 years. However, this study used a different segmentation, including only CA3 (not CA2) in the calculation of DG. Furthermore, these findings could be partially accounted for by effects in the older subjects.

Sex Differences. Hippocampal volumes differ based on sex regardless of ICV, with males demonstrating a larger hippocampus on average than females (see Kaczurkin et al., 2019). Further, these differences are present in childhood (Canada, Botdorf, et al., 2020; Riggins et al., 2018; Tamnes et al., 2018). Specifically, studies have demonstrated sex-based differences in total hippocampus (Canada et al., 2021; Tamnes et al., 2018), hippocampal head (Canada, Botdorf, et al., 2020), and CA2-4/DG (Riggins et al., 2018) in developing populations. Thus, sex is an important covariate when examining the hippocampus.

Development of Sleep Habits

The main variable of interest in my dissertation is nap habituality. This is because past work suggests that nap habituality is thought to reflect maturity in sleep habits that may be relevant to memory consolidation. Specifically, habitual nappers likely require more regular sleep to ‘offload’ memories from the hippocampus to the cortex due to immature neural networks (Lokhandwala & Spencer, 2022; Mason et al., 2021). Therefore, ideally, this review would aim to better understand these mechanisms by only covering studies that examine nap secession during childhood. However, only a small number of studies have investigated associations between memory and nap habituality (e.g., Kurdziel et al., 2013; Kurdziel et al., 2018; Lokhandwala & Spencer, 2021). Further, an even smaller number have investigated associations between nap habituality and the hippocampus (Allard et al., Under Review; Riggins & Spencer, 2020), and no studies have investigated associations with cortical thickness. Therefore, this review will also cover 24-hour sleep duration, which has previously been associated with memory and brain development in childhood (see Dutil et al., 2018 for review). This measure may capture some variability contributed by nap habituality because it accounts for both overnight sleep and daily naps. All other measures of sleep are outside the

scope of this review (see instead Lokhandwala & Spencer, 2022 and Mason et al., 2021 for associations of cognition, the brain, and development with other measures of sleep).

Changes in Sleep Habits

The average amount of sleep children get over a 24-hour period changes with age (Bathory & Tomopoulos, 2017; Iglowstein et al., 2003). According to a meta-analysis conducted by Galland et al., (2012), infants sleep an average of 12.7 hours per day. This decreases to 12 hours per day for children aged 2 to 3 years, and to 11.5 hours per day for preschool-aged children (aged 4 to 5 years; Bathory & Tomopoulos, 2017). Further, overall 24 hour sleep time continues to decrease by approximately 5.9 minutes each year until age 12 years (Galland et al., 2012).

From infancy through early childhood, children also take progressively fewer naps and the duration of naps decreases (Iglowstein et al., 2003; Kurth et al., 2016; Ohayon et al., 2004; Weissbluth, 1995). For example, infants take upwards of 4 naps per day lasting an average of 3 hours per nap. In contrast, children over 5 years who still nap take only one nap per day lasting an average of 1 hour per nap (Galland et al., 2012; Iglowstein et al., 2003; Staton et al., 2020; Weissbluth, 1995). Eventually, these trends lead to nap cessation sometime in early childhood. Specifically, while 97% of children under 2 years nap at least one day per week, by age 3 years, 33% of children have stopped napping and by age 6 years, 94% of children have stopped napping (Staton et al., 2020).

Associations between the Brain and Episodic Memory

In adults, episodic memory abilities are supported by a broad network of neocortical regions including several subregions in medial temporal (i.e., parahippocampal gyrus, entorhinal cortex, and perirhinal cortex), prefrontal (i.e., medial prefrontal, others), parietal (i.e., precuneus,

posterior parietal, and inferior parietal sulcus), and occipital cortices (lateral occipital and lingual gyrus; see Brewer et al., 1998; Buckner et al., 1999; Davachi et al., 2003; Lavenex & Amaral, 2000; Nyberg et al., 2000; Stern et al., 1996; Stevens et al., 2008; Wagner et al., 1998).

Furthermore, the majority of these regions are anatomically connected with the hippocampus (H. Duvernoy et al., 2013) and functionally connected to the default mode network (DMN) which is commonly implicated in memory consolidation and retrieval (see Kaefer et al., 2022). However, this is still an emerging area of interest in the childhood literature, with most studies surfacing in the last decade (e.g., Amlien et al., 2018; Botdorf et al., 2022; Chad-Friedman et al., 2021; Guillery-Girard et al., 2013; Keresztes et al., 2018; Miotto et al., 2020; Østby et al., 2012; Schommartz et al., 2023; Squeglia et al., 2013; Tang et al., 2018; Yu et al., 2018). The purpose of this section is to examine the specific role of each cortical subregion in memory process and then to review literature connecting episodic memory and structural measures of these cortical regions in children.

Medial Temporal Lobe

The medial temporal lobe (MTL) is the brain region most often associated with episodic memory performance. This is because MTL is home to the hippocampus, the memory powerhouse of the brain. The first evidence illuminating the role of the hippocampus in episodic memory came from patient HM, who demonstrated an inability to form new episodic memories after their hippocampus was fully resected (Scoville & Milner, 1957). Since then, research has demonstrated that the hippocampus receives and binds sensory information from two distinct neural pathways (i.e., what and where) that are thought to provide unique event-related details (Eichenbaum et al., 2012; Wixted & Squire, 2011).

The “what” pathway carries item-related information from visual regions through the perirhinal cortex (PRC) and the lateral entorhinal cortex (ERC) to the hippocampus. In contrast, the “where” pathway carries contextual information from the parietal cortex and retrosplenial cortex through the parahippocampal gyrus (PHG) and medial ERC to the hippocampus (see Eichenbaum et al., 2012). Thus, the hippocampus serves as the glue for the unique episodic information provided by the other regions in the MTL that include the PHG, PRC, and the ERC (see Wixted & Squire, 2011 for review).

Structural Association in Total Hippocampus during Childhood. Extensive research has demonstrated that the hippocampus plays a critical role in supporting the formation and consolidation of episodic memories (Davachi et al., 2003; Lavenex & Banta Lavenex, 2013; Scoville & Milner, 1957). In children, cross section studies have demonstrated that structural measures of the hippocampus are related to episodic memory (see Botdorf et al., 2022 for review). Moreover, a handful of longitudinal studies have demonstrated that volumetric changes in the hippocampus across childhood are associated with improvements in episodic memory performance (Canada et al., 2021; Lee et al., 2020). However, findings in total hippocampus are conflicting with some studies demonstrating a positive association (Bauer et al., 2019; Chaddock et al., 2010; Østby et al., 2012; Yu, Daugherty, et al., 2018), some a negative association (Raffington et al., 2019; Schlichting et al., 2017; Willoughby et al., 2008), and some no association at all (Daugherty et al., 2017; DeMaster et al., 2014; Hill et al., 2004; Riggins et al., 2015).

A recent meta-analysis aiming to rectify these conflicting effects found that larger total hippocampal volumes are associated with better memory performance across childhood and into adolescents (Botdorf et al., 2022). Importantly, these findings conflict with a previous meta-

analysis demonstrating the opposite effect. However, this older meta-analysis only included two childhood samples younger than 17 years (Van Petten, 2004). Unfortunately, Botdorf and colleagues (2022) were unable to directly assess associations between memory and hippocampal subfields or subregions because a much smaller sample of studies has examined these associations in childhood. Therefore, in this section, I will discuss the findings from these studies.

Structural Association in Hippocampal Subregions during Childhood. Across early to late childhood (e.g., aged 4 to 12 years), evidence suggests a positive association between hippocampal head volumes and episodic memory performance (Daugherty et al., 2017; Lee et al., 2020; Riggins et al., 2015; Schommartz et al., 2023). Furthermore, Schommartz (2023), demonstrated that findings hold for both short (1 day) and long delays (2 weeks). One notable exception is Schlichting and colleagues (2017) that demonstrated a negative association. That said, the childhood sample was small ($n = 18$) and the associative inference task is notably distinct from other episodic memory tasks. In older children (e.g., aged 6 to 12 years) multiple studies have also demonstrated a positive association between hippocampal body and tail with episodic memory performance (Daugherty et al., 2017; DeMaster et al., 2014; Lee et al., 2020; Schommartz et al., 2023). In sum, these findings demonstrate a reliance on the hippocampal head in early childhood that shifts to include the hippocampal body and tail in later childhood.

Importantly, it should be noted that associations between subregions and episodic memory are highly dependent on the task assessed (Lee et al., 2020). For example, a longitudinal study of 7 to 12-year-olds found that development trajectories of item-item, item-time, and item-space performance were not associated with each other and that they each demonstrated unique relations with subregions development (Lee et al., 2020). Specifically, Item-Item and Item-time

performance were associated with head and body volumes, while item-space performance was associated with tail volumes.

Structural Association in Hippocampal Subfields during Childhood. Evidence in hippocampal subfields demonstrates that associations between volumetric measures and episodic memory differ based on age and subregion assessed. For example, in early childhood (e.g., ages 4 to 6 years), studies agree that improved episodic memory is associated with increased CA1 volume in the head (Canada et al., 2021; Riggins et al., 2018), increased DG/CA2-4 and subiculum volumes in the body (Canada et al., 2019; Riggins et al., 2018), and decreased CA1 volumes in the body (Riggins et al., 2018). In contrast, findings in middle to late childhood (e.g., 6 to 12 years) are less consistent. Specifically, some findings show that improved episodic memory is associated with decreased CA1 volume in the head (Riggins et al., 2018; Schlichting et al., 2017), while other studies demonstrate that improved episodic memory the opposite association (Schommartz et al., 2023). Similarly, in DG/CA2-4 body, some studies show a negative association (Daugherty et al., 2017), while others demonstrate a positive association (Lee et al., 2014; Riggins et al., 2018; Schommartz et al., 2023). Furthermore, Schommartz and colleagues (2023) suggest that increased subiculum in both head and body are associated with memory retention, while other studies have not demonstrated any relation in older childhood. There are several explanations that could explain these differences including the task, ages included, and the subfield delineation used. Future research should aim to rectify these conflicting associations in middle and later childhood.

Structural Association in Extrahippocampal MTL Regions during Childhood. While some functional work has demonstrated associations of PHG, PRC, and ERC with relational memory in children (Fandakova et al., 2019; Golarai et al., 2007; Staresina & Davachi, 2008;

Wang et al., 2013), surprisingly limited research has investigated associations between episodic memory performance and structural measures of PHG, PRC, or ERC during childhood. One notable exception is Schommartz and colleagues (2023) who found a significant negative association between performance on an item location binding task and ERC thickness, but not PHG thickness (Schommartz et al., 2023). This work suggests that ERC, but not its upstream pathways, play a role in memory binding. Moreover, evidence in children has also demonstrated associations between visual spatial memory, verbal memory, and temporal order memory performance with cortical thinning of other regions in the MTL across middle childhood into adulthood (Guillery-Girard et al., 2013; Sowell et al., 2001). These regions include the superior and middle temporal gyrus, and the anterior temporal pole (Guillery-Girard et al., 2013). Together, these findings could suggest that MTL regions surrounding the hippocampal complex assist with memory encoding during childhood. Furthermore, developmental thinning in these regions may work with protracted increases in hippocampus to support improvements in memory performance. However, longitudinal studies are required to test this hypothesis. In this dissertation, I was able to examine this hypothesis.

Prefrontal Cortex

Another region commonly implicated in episodic memory performance is the prefrontal cortex (PFC), which is thought to play a top-down role in memory recall. Specifically, Mill & Cohen (2001) suggested that the hippocampus is responsible for “laying down tracks” while the PFC is responsible for “switching between different tracks.” In other words, the hippocampus binds detailed aspects of an event to form memories, while the prefrontal cortex influences which connections are retrieved. Specifically, PFC is thought to regulate context-appropriate recall by suppressing inappropriate or unrelated memories (Eichenbaum, 2017). Evidence for

this notion is demonstrated by lesion studies that show individuals with PFC damage don't demonstrate episodic memory deficits unless some form of interference is applied after encoding (Shimamura, 1995). Moreover, individuals with PFC lesions demonstrate invasion of irrelevant memories on selective attention tasks (Anderson et al., 2016) suggesting that the PFC inhibits irrelevant episodic information during recall.

Importantly, there are two theories about the role of PFC subregions in the development of episodic memory (Ghetti & Fandakova, 2020). Specifically, one line of work suggests that specific subregions of the PFC that align with the DMN, including bilateral medial orbital frontal, medial frontal pole, and anterior cingulate gyrus (ACC), support strategies that are related to memory recall (Miotto et al., 2020; Yu, Daugherty, et al., 2018). For example, Yu and colleagues (2018) found that semantic clustering during recall on a verbal learning task, a measure indicative of recall strategies, is negatively associated with PFC volumes in a sample of 5 to 25-year-olds. Furthermore, findings demonstrated semantic clustering increases across early childhood and into adulthood (Yu, Daugherty, et al., 2018) suggesting that pruning of these PFC subregions during development lead to increases in memory-related strategy use (Petanjek et al., 2011). However, another study demonstrated that these processes only contribute to memory performance after childhood, in adolescence (Miotto et al., 2020). Thus, there is conflicting evidence about whether improvements in strategies contribute to memory performance during childhood.

A second line of work coming from functional MRI suggests that activation of lateral regions, including dorsolateral PFC (dlPFC) in the rostral middle frontal gyrus and ventrolateral PFC (vlPFC) in the inferior frontal gyrus (IFG), support improved attention during encoding (Miotto et al., 2020; Tang et al., 2018; Wendelken et al., 2011). For example, Tang and

colleagues (2018) found that lateral PFC activation was associated with subsequent memory effects for encoded visual scenes in children, adolescents, and adults. Furthermore, this association between memory and region-specific activation increases with age (Tang et al., 2018), suggesting memory encoding increasingly relies on these frontal regions. Relatedly, Wendelken (2011) found similar effects in middle to late childhood. Specifically, lateral PFC's selective encoding of task-relevant versus task-irrelevant information differed between children and adults. Importantly, both studies demonstrate a role for attention-related PFC activity in older children suggesting that these processes are at play in developing populations. However, we are not aware of research that has examined these associations in early childhood.

In summary, these two lines of work suggest that subregions of the PFC play distinct but complementary roles in memory development. Specifically, regions overlapping the DMN, like the medial frontal gyrus and ACC, contribute to improved memory strategies during recall. In contrast, lateral PFC regions, like the rostral middle frontal gyrus and IFG, contribute to enhanced attention during encoding.

Structural Association in PFC Subregions during Childhood. Past work demonstrates that these dynamics can be detected in structural measures of the brain during childhood. Specifically, research has shown that a thinner medial orbitofrontal cortex (Bauer et al., 2019; Østby et al., 2012; Schommartz et al., 2023), ACC (Bauer et al., 2019), IFG (Guillery-Girard et al., 2013; Klijn et al., 2016; Schommartz et al., 2023), and rostromedial cortex (Guillery-Girard et al., 2013; Schommartz et al., 2023) are associated with better memory performance on a variety of memory tasks that assess temporal order and feature binding in early, middle, and late childhood samples. Furthermore, Klijn et al., 2016 found that left IFG cortical thickness mediates age-related differences in verbal memory performance between children (10-12),

adolescents (18), and adults (25 to 32) suggesting that age-related differences in prefrontal cortex underlie age-related differences in memory performance. Together these findings demonstrate that there are associations between episodic memory and structural measures of PFC subregions implicated in both memory recall strategies and enhanced memory encoding during childhood.

Parietal and the Posterior Medial Cortices

The parietal cortex is believed to play a central role in attention, visual-spatial, and sensorimotor functions (Corbetta & Shulman, 2011; Kastner & Ungerleider, 2001). However, evidence from the last two decades also suggests that it also plays a central role in memory (see Cabeza et al., 2008; Sestieri et al., 2017). Further, functional studies in adults demonstrate that it is one of the most active regions during episodic retrieval (Cabeza et al., 2001). Importantly, findings linking the parietal cortex to memory fall into two categories. Some studies have demonstrated that the parietal cortex plays a role in episodic-like memory (i.e., recollection) that includes highly contextual information about past events (Buckner et al., 1998; Konishi et al., 2000). While other studies have demonstrated an additional role for the parietal cortex in memory familiarity that allows the brain to make judgments about “oldness” without recalling accurate information about the item or relevant context (Kahn et al., 2004; Wheeler & Buckner, 2003). Thus, a variety of theories have been proposed to explain associations between the parietal cortex and these diverse memory processes (Cabeza et al., 2008; Sestieri et al., 2017; Wagner et al., 2005).

Three of the earliest theories include the output buffer hypothesis, the mnemonic accumulator hypothesis, and the attention to internal representations hypothesis (Wagner et al., 2005). Each of these hypotheses suggests a different role for parietal cortex in memory. For example, the *output buffer hypothesis* suggests that parietal regions are responsible for holding

information relevant to decision-making about information familiarity. Whereas the *mnemonic accumulator hypothesis* proposes that the parietal cortex doesn't hold actual memories. Instead, it integrates memory-strength signals coming from surrounding brain regions allowing for an understanding of familiarity without making accurate judgments. Both hypotheses explain associations between the parietal cortex and memory familiarity, but they don't explain associations with detailed episodic memory retrieval. In contrast, the *attention to internal representation hypothesis* explains this association by suggesting that parietal regions shift attention based on the intention to remember information. However, it does not explain parietal associations with memory familiarity when detailed memories are forgotten. In sum, none of these hypotheses perfectly account for existing evidence suggesting a role for the parietal cortex in both recollection and familiarity.

In 2008, Cabeza and colleagues proposed the *Attention to Memory Model (AtoM)*. It addresses the pitfalls of other theories by suggesting that the parietal cortex plays a multifaceted attentional role in episodic memory encoding (Cabeza et al., 2008). Specifically, the dorsal parietal cortex (DPC); i.e., the superior parietal lobule and the precuneus) play a top-down role in attention that helps to maintain retrieval goals, while the ventral parietal cortex (VPC; i.e., the supramarginal and angular gyri) plays a bottom-up role in attention that is captured by relevant memory cues. Importantly, this model explains differential activation in familiarity vs. recollections. Specifically, bottom-up processing in VPC allows for the spontaneous retrieval of episodic details during recollection. Whereas top-down processing in DPC engages decision-making about old vs. new information to produce a sense of familiarity (Cabeza et al., 2008). Cabeza goes on to say that these attention processes are distinct constructs that interact closely, suggesting that both bottom-up and top-down mechanisms can play a role in episodic memory

performance. As a result, there may be overlapping contributions of DPC and VPC to episodic retrieval.

Importantly, more recent accounts suggest that the parietal cortex is highly modular and that subregions play a distinct role in episodic memory based on their connections with larger brain-wide networks resulting in a functional anatomical parcellation (Hutchinson et al., 2009; Sestieri et al., 2010, 2017). In contrast to the AtoM model, this notion proposes that there are three types of functional subregions in the parietal cortex that are divided based on their inclusion in the Dorsal Attention Network (DAN), Frontal-Parietal Control Network, and the Default Mode Network (DMN). Specifically, parietal regions overlapping with the DAN, namely the posterior intraparietal sulcus (pIPS) and superior parietal lobule (SPL), are involved in perceptual attention. Whereas, parietal regions overlapping with the Frontal-Parietal Control Network, namely the lateral IPS (lIPS), are involved with memory familiarity and parietal regions overlapping with the Default Mode Network, namely the angular gyrus (AG), co-active with the MTL to recall past events.

The E/R Flip in the Posteromedial Cortex. In the posteromedial cortex, findings have demonstrated a similar pattern of results to the functional anatomical parcellation model proposed Sestieri (2010, 2017). Specifically, the posterior medial cortex is a series of regions in the parietal and cingulate cortices that are functionally connected to the DMN (Buckner et al., 2008) with downstream connections to the hippocampus (Rolls, 2022). It includes the precuneus extending into the posterior cingulate cortex and retrosplenial cortex (Bzdok et al., 2015). Previous work shows that these regions deactivate during memory encoding (Daselaar et al., 2004), but activate during retrieval (Daselaar et al., 2004; Wagner et al., 2005), a phenomenon referred to as the encoding/retrieval or E/R flip. These findings are consistent with the role of the

DMN in memory which is thought to deactivate while attending to external stimuli but reactivate while orienting to internal stimuli like spontaneous memory retrieval (Buckner et al., 2008).

The handful of studies that have directly examined this process suggest that the magnitude of this functional modulation or the E/R flip are related to memory performance (Amlien et al., 2018; Huijbers et al., 2012; Vannini et al., 2011, 2013). Additionally, findings demonstrate that this functional modulation increases from early childhood through late adolescence (Amlien et al., 2018) and then declines into older adulthood (Vannini et al., 2013). Thus, developmental changes in the E/R flip could potentially account for some memory performance changes in developing and aging populations.

Structural Association in Parietal and PMC Regions during Childhood. In children, there is strong evidence connecting structural subregions of the parietal cortex and the posterior medial cortex to episodic memory performance (Amlien et al., 2018; Chad-Friedman et al., 2021; Schommartz et al., 2023; Squeglia et al., 2013). Specifically, studies have found that bilateral cortical thinning of the precuneus, PCC, and the inferior parietal cortex is associated with better performance on a variety of episodic memory tasks (e.g., an item location binding task, a source memory task, and a verbal memory task) in developing populations (Amlien et al., 2018; Schommartz et al., 2023; Squeglia et al., 2013). Further, one study found that white matter connectivity from the hippocampus to the IPS, but not medial PFC, significantly predicted children's memory performance during early childhood (Ngo et al., 2017). These findings suggest that parietal regions that overlap with DMN are associated with episodic memory performance in childhood.

There is also some evidence connecting the superior parietal cortex, located in the dorsal attention network, with performance in this age group (Chad-Friedman et al., 2021; Schommartz

et al., 2023; Squeglia et al., 2013). However, findings are conflicting. For example, two separate samples of 5 to 7-year-old children demonstrated an association between superior parietal cortex and performance on a feature-binding task (Chad-Friedman et al., 2021; Schommartz et al., 2023). However, one found a positive association (Chad-Friedman et al., 2021) and the other a negative association (Schommartz et al., 2023). Differences in these associations are likely attributed to varying task demands. Specifically, the delay in Schommartz (2023) was 1 day to 2 weeks whereas the delay in Chad-Friedman (2019) was only 1 hour. Further, Schommartz (2023) used an item location binding task containing more visual elements, whereas Chad-Friedman (2019) used a source memory paradigm containing more verbal elements. Thus, future research in this particular area could benefit from a latent approach to memory.

Occipital Cortex

The occipital cortex is most commonly associated with visual processing; therefore, it may seem unlikely that it would be related to memory performance. However, one theory does explain possible associations between the occipital cortex and episodic memory, the neurobiological model of memory. This model assumes that regions originally active during encoding are reactivated during recall due to the consolidation of memories from the hippocampus to the neocortex (e.g., Alvarez & Squire, 1994; Norman & O'Reilly, 2003).

Importantly, there is functional evidence for the neurobiological model of memory in a subregion of the occipital cortex called the lingual gyrus, located in the inferior medial occipital cortex. Beyond visual processing, this region is typically associated with word learning (Mechelli et al., 2000) and spatial navigation (Boccia et al., 2014; Teghil et al., 2021). However, Wing and colleagues (2015) also found an association with episodic memory performance on a verbal memory task. Specifically, findings demonstrated that the lingual gyrus shows a similar

pattern of activation during both an episodic memory encoding trial and a recall trial (Wing et al., 2015) suggesting that memories are reactivated in this region following a delay.

Another occipital region that is commonly implicated in memory performance is lateral occipital cortex (LOC), that is typically associated with shape processing and visual object recognition (Lucan et al., 2010). Past work in children aged 8 to 15 years demonstrates that activation of LOC during encoding is associated with the reinstatement of object-related information during retrieval (Fandakova et al., 2019; Karanian & Slotnick, 2015). For example, one study found that increased activation in the LOC during encoding was associated with better item location binding performance in a sample of 6 to 19-year-olds (Rosen et al., 2018). Furthermore, relations between age and memory performance were mediated by LOC activation. Together these findings suggest that LOC and lingual gyrus are implicated in memory processes and that early developmental changes in these regions may partially explain memory improvement. However, findings in each region are likely task-specific, with the lingual gyrus more commonly implicated in verbal memory and LOC more commonly implicated in spatial memory.

Structural Association in Occipital Regions during Childhood. In children, research examining structural associations between the occipital cortex and memory performance is limited. However, a handful of studies have connected cortical thinning of both LOC and lingual gyrus to episodic memory performance across early childhood (Amlien et al., 2018; Schommartz et al., 2023). Specifically, Amlien and colleagues (2018) used a vertex-wise whole brain analysis to examine associations between cortical thickness and memory performance on a source memory task. Findings demonstrated that cortical thickness in the lingual gyrus was negatively associated with source memory performance in a lifespan sample of 5 to 80-year-olds (Amlien et

al., 2018). Similarly, Schommartz and colleagues (2022) found a negative association between both memory performance on an item location binding task and cortical thickness of LOC in an early childhood sample of 5 to 7-year-olds.

Importantly, findings in each of these studies could be linked to the unique task utilized and the roles of each implicated occipital subregion. For example, source memory tasks tend to rely more heavily on verbal abilities while item location binding tasks assess spatial processing. This could explain why lingual gyrus was implicated in Amlien (2018), while LOC was implicated in Schommartz (2023). Together these findings suggest thinner lingual gyrus and LOC are associated with better memory performance across childhood. However, these regions may only be associated with memory performance on tasks that test unique sensory processing abilities related to each region.

Table 1*Peer-reviewed Articles that Examine Associations Between Memory and Cortical ROIs.*

Study	N	Mean Age (years)	Age Range (years)	ROI(s)	Memory Assessment(s)
Amlien et al (2018)	270	19.4	6 to 80	Isthmus Cingulate Lingual Gyrus Posterior Cingulate Cortex Precuneus	Source Memory
Bauer et al. (2019)	66	7.34	5 to 8	ACC mPFC Hippocampus	Self-Derivation through Integration (Stem Facts–Open Ended) Self-Derivation through Integration (Stem Facts–Total) Self-Derivation through Integration (Integration Facts–Open Ended) Self-Derivation through Integration (Integration Facts–Total)
Chad-Friedman et al (2021)	63	4.23 & 7.19	4 to 7	SPL	CMS Source Memory
Fjell et al (2019)	650	25.8	4 – 88	Hippocampus IPFC	CVLT-C Rey-Osterrieth Complex Figure test
Klijin et al., 2016	90	11.0 (child sample)	10 to 12, 18, & 25 to 32	IFG	Verbal Memory Task
Guillery-Girard et al (2013)	30	11.31	6 to 23	Anterior Middle Temporal Gyrus dlPFC Hippocampus Superior Temporal Cortex vlPFC	What-Where-When Paradigm
Kereteszes et al., (2017)	70	9.8	6 to 14	ERC Hippocampus	Source Memory MST
Ostby et al (2012)	107	13.9	8 to 19	Hippocampus mOFC	Rey-Osterrieth Complex Figure test
Schommartz et al., (2023)	63	6.37	5 to 7	Hippocampus ERC IFG Inferior Parietal Sulcus LOC Lateral Orbitofrontal Cortex Medial Orbitofrontal Cortex Precuneus Rostral Middle Frontal Cortex SPL	Object-Location Association Task
Sowell et al (2001)	35	N/A	7 to 16	Frontal Cortex MTL	CVLT-C Rey-Osterrieth Complex Figure test
Squeglia et al (2013)	185	N/A	12 to 14	IPS SPL	CVLT-C
Yu et al (2018)	120	13.6	5 to 25	dlPFC (Rostral Middle Frontal Cortex)	CVLT-C

Summary

In sum, episodic memory performance is supported by a broad network of cortical regions during childhood (e.g., Schommartz et al., 2023; see table 1). Moreover, these regions likely play unique roles in memory performance. For example, some regions in PFC and the parietal lobe overlap with DMN and are believed to play a role in memory recall. In contrast, other regions overlap with control networks that are thought to support memory encoding (Ghetti & Fandakova, 2020; Sestieri et al., 2010, 2017). Importantly, work in some of these regions show that developmental changes in cortical thickness support improvements in episodic memory (Canada et al., 2021; Lee et al., 2020). However, longitudinal studies are limited, thus, developmental questions are difficult to examine using the existing literature. Furthermore, region specific associations may be task specific. Therefore, future work should aim to examine these questions using a longitudinal design with a latent measure of episodic memory.

Associations between the Sleep Habits and Episodic Memory

24-hour sleep duration in middle to late childhood

In older children (aged 8 to 12 years), most research suggests little to no association between 24-hour sleep duration and memory performance. For example, a recent meta-analysis found an association with overall measures of cognitive ability, but not memory in middle to late childhood (Short et al., 2018). However, these findings reflect results from both working memory (i.e., digit span and n-back) and episodic memory (e.g., auditory verbal learning). Unfortunately, only 6 of the 14 studies included an episodic memory task that included some form of item binding, temporal ordering, or pattern separation (Biggs et al., 2010; Bruni et al., 2012; Carskadon et al., 1981; Randazzo et al., 1998; Vriend et al., 2012, 2013). Of these, all but

one demonstrated no association between episodic memory and 24-hour sleep duration or overnight sleep restriction (Biggs et al., 2010; Bruni et al., 2012; Carskadon et al., 1981; Vriend et al., 2012, 2013).

The one notable exception is Randazzo (1998) who did find an effect. However, it was in the opposite direction of expectations. Specifically, the experimental design in this study required some children to sleep 11 hours (control group), but others only 5 hours. Results showed that children in the control group did worse on an episodic memory task. Authors note that these effects were not driven by overall performance in the sleep restriction group, but rather by performance deficits in the control group suggesting that too much overnight sleep may have negative effects on memory performance.

Importantly, results from these 6 studies come with three important disclaimers. First, all of these studies contain relatively small samples ($Ns = 9 - 42$ children), including 3 samples with fewer than 20 participants. Thus, they may have been underpowered. Second, though the metaanalysis suggests that all of these studies provide some information about 24-hour sleep duration, many utilized sleep restriction techniques that did not properly assess daytime sleep. Therefore, some of these studies do not account for the unique effects of napping nor the interactive effect of napping with overnight sleep. Finally, the third restriction is that these samples were restricted to middle and late childhood (e.g., aged 8 to 12 years). Thus, early childhood was not examined in these findings.

24-hour sleep duration in early to middle childhood

In younger children (e.g., 3 to 8 years) both experimental and correlational studies have found associations between 24-hour sleep duration and episodic memory performance (Kurdziel

et al., 2018; Riggins & Spencer, 2020). For example, Kurdziel and colleagues (2018), tested whether an afternoon nap or an overnight sleep bout had an impact on memory performance across a 24-hour period. In the study, there were two conditions: the first was a nap condition and the second was a wake condition lasting the same duration. They found that there were memory deficits due to a missed afternoon nap, but only after an overnight sleep bout. Thus, the effects of an afternoon nap and overnight sleep have an interactive effect on memory performance. This is consistent with findings from Riggins and Spencer (2020) who found that 24-hour sleep duration is positively associated with source memory performance in 4 to 8-year-old children. Together, these findings could suggest that associations between 24-hour sleep duration and episodic memory are specific to early childhood and that nap habits could be a possible contributing mechanism (Blair et al., 2012; Galland et al., 2012; Iglowstein et al., 2003; Kurdziel et al., 2018).

The Unique Benefits of an Afternoon Nap

Past work suggests that napping provides an important contribution to memory performance above and beyond 24-hour sleep behaviors (Cousins et al., 2019). For example, one study showed that adolescents and young adults who got 5 hours of overnight sleep and 1.5 hours of daytime sleep performed better on a memory task than those who received equivalent overnight sleep. Thus, it is valuable to study the unique impacts of napping beyond 24-hour sleep duration.

In early childhood, strong evidence demonstrates that an afternoon nap has a positive impact on several memory abilities including emotion memory (Kurdziel et al., 2018), temporal order memory (Lokhandwala & Spencer, 2021); verbal memory (Esterline & Gómez, 2021; Giganti et al., 2014; Spanò et al., 2018; H. Wang et al., 2022; Williams & Horst, 2014) and

memory generalization (Sandoval et al., 2017; Wang et al., 2022). Furthermore, several studies have found that the memory benefit generated by an afternoon nap persists after overnight sleep (Kurdziel et al., 2018; Lokhandwala & Spencer, 2021; Sandoval et al., 2017; Spanò et al., 2018; Williams & Horst, 2014) suggesting that memories lost during an afternoon wake session cannot be regained at night. Moreover, one study found persisting effects of an afternoon nap 1 week later (Williams & Horst, 2014). In sum, an afternoon nap improves memory performance, with little evidence to the contrary, and the impacts of a nap are long-lasting.

Although few studies have examined these effects in middle and late childhood, there is evidence that these effects are not specific to early childhood. Specifically, a meta-analysis of 54 studies examining relations between cognition and afternoon naps in samples ranging from early childhood to mid-adulthood found that an afternoon nap has a positive effect on declarative memory and that these effects are not moderated by age (Leong et al., 2022). Thus, an afternoon nap is good for everyone, regardless of age.

Nap Habituality

Importantly, while past works does suggest that an afternoon nap is good for everyone, regardless of age, the effects of missing an afternoon nap are not. Past work demonstrates that habitual nappers who miss their afternoon nap display more extreme memory decay on episodic memory tasks than non-nappers (Esterline & Gómez, 2021; Kurdziel et al., 2013; Kurdziel et al., 2018). Further, these memory differences are still present 24 hours later (Kurdziel et al., 2013; Kurdziel et al., 2018). Moreover, one study in adults found that habitual nappers demonstrate a greater nap benefit following an afternoon nap compared to their non-napping peers (McDevitt et al., 2018). Similar trends can be observed in some childhood samples, though findings don't reach significance (Kurdziel et al., 2013). Only one study has demonstrated contrary findings,

showing no unique effects on an afternoon nap on memory due to nap habituality (Sandoval et al., 2017). However, this study used a memory generalization task instead of a traditional retrieval task. Therefore, this likely suggests that nap habituality does have an effect on episodic memory performance following a nap, but it does not have an effect on memory generalization.

Associations between the Brain and Sleep Habits

Associations between brain structure and sleep duration/disturbances

Shorter 24-hour sleep duration is consistently associated with reduced total gray matter volume, cortical surface area, and cortical thickness across childhood (Cheng et al., 2021; Hansen et al., 2022; Taki et al., 2012). Furthermore, there are regional associations between sleep duration and ROIs implicated in episodic memory. For example, past work has shown that shorter 24-hour sleep duration is associated with decreased cortical volumes and surface area in the orbital frontal region, superior and middle frontal gyri, inferior and middle temporal gyri, precuneus, posterior cingulate cortex, ventromedial prefrontal cortex, and dlPFC of the rostral medial prefrontal cortex across the middle and into late childhood (Cheng et al., 2021). Additionally, Hansen and colleagues (2022) showed that shorter sleep duration during weekdays is significantly associated with reduced cortical thickness in the left middle temporal gyrus, right postcentral, and right superior frontal cortices.

In the hippocampus, investigations of 24-hour sleep duration and individual differences in brain morphology are conflicting. Specifically, one study found that total hippocampal volume was positively associated with 24-hour sleep duration in a sample of healthy children aged 5 to 18 years (Taki et al., 2012). However, other studies have found no association between sleep

duration and total hippocampal volume during middle childhood (e.g., 7 years; Kocevka et al., 2017) and late childhood (e.g., 9 to 11 years; Cheng et al., 2021).

There are two possible contributing factors. The first possibility is variations in the age of the sample. Specifically, Taki (2012) covers a younger age range than Kocevka (2017) and Cheng (2021). This younger group may be more sensitive to differences in sleep duration. Notably, 24-hour sleep duration in this group is also more likely to be influenced by daytime napping due to changes in nap habitually. The second explanation is that total hippocampal volume is not specific enough to capture the effects of shorter sleep duration. Notably, none of these three studies investigated hippocampal subregions or subfields, which may be differentially affected by sleep loss. That said, Riggins and Spencer (2020) support both claims. They found that younger (aged 4 to 6 years), but not older children (aged 6 to 8 years), demonstrate a positive association between 24-hour sleep duration and ICV-adjusted volume of CA2-4/DG volumes.

Associations between brain structure and nap habituality

Research examining associations between brain structure and nap habituality during childhood is extremely limited. Furthermore, studies have only examined the hippocampus and not the larger network of cortical regions that support memory. In the hippocampus, studies have demonstrated significant differences between habitual nappers and non-nappers in both hippocampal subregions (Allard et al., Under Review) and hippocampal subfields (Riggins & Spencer, 2020). However, findings are in the opposite direction. Specifically, Allard and colleagues (in prep) found that hippocampal head volumes were significantly greater for habitual nappers aged 3 to 5 years. In contrast, Riggins and Spencer (2020) found that CA1 volumes in the hippocampal body are significantly smaller in habitual nappers aged 4 to 6 years.

There are several key factors that could account for these differences. First, these studies used different definitions of habitual napper. Specifically, Allard (in prep) described habitual nappers as napping greater than 5 days per week, while Riggins and Spencer (2020) considered habitual nappers to be those napping greater than 2 days per week. These are two drastically different definitions of napping that could easily influence results. Second, Riggins and Spencer (2020) used a slightly older sample than Allard (in prep) and utilized subfields instead of subregions. Given evidence that CA1 volumes in the hippocampal body peak prior to volumes in total hippocampal head, these conflicting findings could be described by differing developmental trajectories in the two subunits. Future work should continue to examine these dynamics to better understand how nap habituality contributes to brain development.

Gaps and Future Directions

There are several limitations to the existing literature. In this section, I will discuss these limitations and some potential future directions.

First, most studies examining memory development utilize a single task that assesses only one underlying episodic memory ability (e.g., feature binding or temporal order memory). However, these tasks are not process pure and likely assess additional cognitive systems. Latent memory models are a solution to this problem. Only two studies have utilized latent memory variables to assess episodic memory development during childhood (Canada et al., 2022; Cheke & Clayton, 2015). Furthermore, neither of these studies connected latent memory with brain development or sources of individual differences like sleep. To address this gap, future research should utilize latent memory variables to better understand associations between memory, the brain, and contextual factors underlying differences in developmental trajectories.

Second, very few studies have examined extrahippocampal structures and episodic memory in early to middle childhood (e.g., ages 4 to 8 years). Though there is significant evidence connecting the hippocampus and episodic memory during this period (see Botdorf et al., 2022). Although there are some notable exceptions (Amlien et al., 2018; Bauer et al., 2019; Chad-Friedman et al., 2021; Fjell et al., 2019; Guillery-Girard et al., 2013; Keresztes et al., 2018; Østby et al., 2012; Schommartz et al., 2023; Sowell et al., 2001; Squeglia et al., 2013; Yu, Daugherty, et al., 2018). Of these, several have very small samples of young children, focusing instead on older children and adults. Further, longitudinal studies examining these associations during childhood are non-existent. Given that past work has demonstrated differences in brain behavior relations based on cross-sectional vs. longitudinal designs (Canada et al., 2021; Keresztes et al., 2022; Riggins et al., 2018), future studies should aim to utilize longitudinal methods when examining extrahippocampal relations with memory.

Third, past work has demonstrated that there are differences in episodic performance based on nap habitually that are likely driven by changes in brain regions that support memory consolidation (Esterline & Gómez, 2021; Kurdziel et al., 2013; Kurdziel et al., 2018). Yet, only limited work has examined associations between nap habitually and the brain (Allard et al., Under Review; Riggins & Spencer, 2020). These two studies both demonstrated that nap secession is related to hippocampal maturation in early childhood. However, no work has examined associations between nap habitability and cortical regions outside the hippocampus that support episodic memory. Future work should expand these findings beyond the hippocampus as the cortex also plays a critical role in memory consolidation.

Conclusion

In summary, previous work demonstrates that episodic memory, brain regions that support memory, and sleep habits all undergo developmental changes across childhood. Further, these changes are likely entangled. Specifically, sleep supports consolidation of memory from the hippocampus to the cortex. Thus, changes in sleep habits are unsurprisingly related to both episodic memory and brain development of regions related to episodic memory during childhood. However, the current literature is missing two key components. The first is that there is currently no longitudinal investigation of extrahippocampal memory regions and episodic memory performance. The second is that there is very limited work investigating differences in brain structure based on nap habitually, and no work investigating these differences outside the hippocampus. My dissertation addresses both gaps.

Bibliography

- Acebo, C., Sadeh, A., Seifer, R., Tzischinsky, O., Hafer, A., & Carskadon, M. A. (2005). Sleep/Wake Patterns Derived from Activity Monitoring and Maternal Report for Healthy 1- to 5-Year-Old Children. *Sleep*, *28*(12), 1568–1577.
- Alden, J. T. (1994). *Development of memory for temporal order* [Ph.D., University of Minnesota].
<https://www.proquest.com/docview/304122280/abstract/44F8AED98BA345E2PQ/1>
- Allard, T. L., Canada, K. L., Botdorf, M., & Riggins, T. (2023). Longitudinal Exploration of Binding Ability across Early Childhood: The Differential Contribution of Hits and False Alarms. *The Journal of Genetic Psychology*, *0*(0), 1–14.
<https://doi.org/10.1080/00221325.2023.2213268>
- Allard, T. L., Lokhandwala, S., Spencer, R. M. C., & Riggins, T. (Under Review). *Associations between nap habituality, hippocampal volume, sleep physiology and memory in preschool-aged children.*
- Alvarez, P., & Squire, L. R. (1994). Memory consolidation and the medial temporal lobe: A simple network model. *Proceedings of the National Academy of Sciences*, *91*(15), 7041–7045. <https://doi.org/10.1073/pnas.91.15.7041>
- Amaral, D. G., & Lavenex, P. (2007). Hippocampal neuroanatomy. In P. Andersen, R. Morris, D. Amaral, T. Bliss, & J. O'Keefe (Eds.), *The hippocampus book*. Oxford University Press.
- Amlien, I. K., Sneve, M. H., Vidal-Piñeiro, D., Walhovd, K. B., & Fjell, A. M. (2018). The Lifespan Trajectory of the Encoding-Retrieval Flip: A Multimodal Examination of Medial Parietal Cortex Contributions to Episodic Memory. *The Journal of Neuroscience*:

The Official Journal of the Society for Neuroscience, 38(40), 8666–8679.

<https://doi.org/10.1523/JNEUROSCI.1702-17.2018>

- Anderson, M. C., Bunce, J. G., & Barbas, H. (2016). Prefrontal–hippocampal pathways underlying inhibitory control over memory. *Neurobiology of Learning and Memory*, 134, 145–161. <https://doi.org/10.1016/j.nlm.2015.11.008>
- Atkinson, R. C., & Shiffrin, R. M. (1968). Human Memory: A Proposed System and its Control Processes. In K. W. Spence & J. T. Spence (Eds.), *Psychology of Learning and Motivation* (Vol. 2, pp. 89–195). Academic Press. [https://doi.org/10.1016/S0079-7421\(08\)60422-3](https://doi.org/10.1016/S0079-7421(08)60422-3)
- Barnett, V., & Lewis, T. (1994). *Outliers in statistical data* (Vol. 3). Willey.
- Bathory, E., & Tomopoulos, S. (2017). Sleep Regulation, Physiology and Development, Sleep Duration and Patterns, and Sleep Hygiene in Infants, Toddlers, and Preschool-Age Children. *Current Problems in Pediatric and Adolescent Health Care*, 47(2), 29–42. <https://doi.org/10.1016/j.cppeds.2016.12.001>
- Bauer, P. J., Dikmen, S. S., Heaton, R. K., Mungas, D., Slotkin, J., & Beaumont, J. L. (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/10.1016/j.jecp.2012.06.007>
- Bauer, P. J., Dugan, J. A., Varga, N. L., & Riggins, T. (2019). Relations between neural structures and children’s self-derivation of new knowledge through memory integration. *Developmental Cognitive Neuroscience*, 36, 100611. <https://doi.org/10.1016/j.dcn.2018.12.009>

- Biggs, S. N., Bauer, K. M. M., Peters, J., Dorrian, J., Kennedy, J. D., Martin, A. J., & Lushington, K. (2010). Acute sleep restriction does not affect declarative memory in 10-year-old girls. *Sleep and Biological Rhythms*, 8(222–225), 222–225.
- Blair, P. S., Humphreys, J. S., Gringras, P., Taheri, S., Scott, N., Edmond, A., Henderson, J., & Peter J. Fleming. (2012). Childhood Sleep Duration and Associated Demographic Characteristics in an English Cohort. *Sleep*, 35(3), 353–360.
- Boccia, M., Nemmi, F., & Guariglia, C. (2014). Neuropsychology of Environmental Navigation in Humans: Review and Meta-Analysis of fMRI Studies in Healthy Participants. *Neuropsychology Review*, 24(2), 236–251. <https://doi.org/10.1007/s11065-014-9247-8>
- Botdorf, M., Canada, K. L., & Riggins, T. (2022). A meta-analysis of the relation between hippocampal volume and memory ability in typically developing children and adolescents. *Hippocampus*, 32(5), 386–400. <https://doi.org/10.1002/hipo.23414>
- Brewer, J. B., Zhao, Z., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. E. (1998). Making Memories: Brain Activity that Predicts How Well Visual Experience Will Be Remembered. *Science*, 281(5380), 1185–1187. <https://doi.org/10.1126/science.281.5380.1185>
- Brown, T. T., & Jernigan, T. L. (2012). Brain Development During the Preschool Years. *Neuropsychology Review*, 22(4), 313–333. <https://doi.org/10.1007/s11065-012-9214-1>
- Bruni, O., Kohler, M., Novelli, L., Kennedy, D., Lushington, K., Martin, J., & Ferri, R. (2012). The Role of NREM Sleep Instability in Child Cognitive Performance. *SLEEP*. <https://doi.org/10.5665/sleep.1824>

- Buckner, R. L., Andrews-Hanna, J. R., & Schacter, D. L. (2008). The Brain's Default Network. *Annals of the New York Academy of Sciences*, 1124(1), 1–38.
<https://doi.org/10.1196/annals.1440.011>
- Buckner, R. L., Kelley, W. M., & Petersen, S. E. (1999). Frontal cortex contributes to human memory formation. *Nature Neuroscience*, 2(4), Article 4. <https://doi.org/10.1038/7221>
- Buckner, R. L., Koutstaal, W., Schacter, D. L., Dale, A. M., Rotte, M., & Rosen, B. R. (1998). Functional–Anatomic Study of Episodic Retrieval: II. Selective Averaging of Event-Related fMRI Trials to Test the Retrieval Success Hypothesis. *NeuroImage*, 7(3), 163–175. <https://doi.org/10.1006/nimg.1998.0328>
- Bzdok, D., Heeger, A., Langner, R., Laird, A. R., Fox, P. T., Palomero-Gallagher, N., Vogt, B. A., Zilles, K., & Eickhoff, S. B. (2015). Subspecialization in the human posterior medial cortex. *NeuroImage*, 106, 55–71. <https://doi.org/10.1016/j.neuroimage.2014.11.009>
- Cabeza, R., Ciaramelli, E., Olson, I. R., & Moscovitch, M. (2008). The parietal cortex and episodic memory: An attentional account. *Nature Reviews Neuroscience*, 9(8), Article 8. <https://doi.org/10.1038/nrn2459>
- Cabeza, R., Rao, S. M., Wagner, A. D., Mayer, A. R., & Schacter, D. L. (2001). Can medial temporal lobe regions distinguish true from false? An event-related functional MRI study of veridical and illusory recognition memory. *Proceedings of the National Academy of Sciences*, 98(8), 4805–4810. <https://doi.org/10.1073/pnas.081082698>
- Cafiero, R., Brauer, J., Anwender, A., & Friederici, A. D. (2019). The Concurrence of Cortical Surface Area Expansion and White Matter Myelination in Human Brain Development. *Cerebral Cortex*, 29(2), 827–837. <https://doi.org/10.1093/cercor/bhy277>

- Canada, K. L., Botdorf, M., & Riggins, T. (2020). Longitudinal development of hippocampal subregions from early- to mid-childhood. *Hippocampus*, *30*(10), 1098–1111.
<https://doi.org/10.1002/hipo.23218>
- Canada, K. L., Hancock, G. R., & Riggins, T. (2021). Modeling longitudinal changes in hippocampal subfields and relations with memory from early- to mid-childhood. *Developmental Cognitive Neuroscience*, *48*, 100947.
<https://doi.org/10.1016/j.dcn.2021.100947>
- Canada, K. L., Hancock, G. R., & Riggins, T. (2022). Developmental changes in episodic memory across early- to mid-childhood: Insights from a latent longitudinal approach. *Memory*, *30*(3), 248–261. <https://doi.org/10.1080/09658211.2021.2006233>
- 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>
- Canada, K. L., Pathman, T., & Riggins, T. (2020). Longitudinal development of memory for temporal order in early to middle childhood. *The Journal of Genetic Psychology*, *181*(4), 237–254. <https://doi.org/10.1080/00221325.2020.1741504>
- Carskadon, M. A., Harvey, K., & Dement, W. C. (1981). Acute Restriction of Nocturnal Sleep in Children. *Perceptual and Motor Skills*, *53*(1), 103–112.
<https://doi.org/10.2466/pms.1981.53.1.103>
- Chaddock, L., Erickson, K. I., Prakash, R. S., Kim, J. S., Voss, M. W., VanPatter, M., Pontifex, M. B., Raine, L. B., Konkel, A., Hillman, C. H., Cohen, N. J., & Kramer, A. F. (2010). A neuroimaging investigation of the association between aerobic fitness, hippocampal

- volume, and memory performance in preadolescent children. *Brain Research*, 1358, 172–183. <https://doi.org/10.1016/j.brainres.2010.08.049>
- Chad-Friedman, E., Botdorf, M., Riggins, T., & Dougherty, L. R. (2021). Early childhood cumulative risk is associated with decreased global brain measures, cortical thickness, and cognitive functioning in school-age children. *Developmental Psychobiology*, 63(2), 192–205. <https://doi.org/10.1002/dev.21956>
- 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>
- Cheng, W., Rolls, E., Gong, W., Du, J., Zhang, J., Zhang, X.-Y., Li, F., & Feng, J. (2021). Sleep duration, brain structure, and psychiatric and cognitive problems in children. *Molecular Psychiatry*, 26(8), 3992–4003. <https://doi.org/10.1038/s41380-020-0663-2>
- Cohen, J. (1988). Set Correlation and Contingency Tables. *Applied Psychological Measurement*, 12(4), 425–434.
- Corbetta, M., & Shulman, G. L. (2011). Spatial Neglect and Attention Networks. *Annual Review of Neuroscience*, 34(1), 569–599. <https://doi.org/10.1146/annurev-neuro-061010-113731>
- Curran, P. J., & Hancock, G. R. (2021). The Challenge of Modeling Co-Developmental Processes over Time. *Child Development Perspectives*, 15(2), 67–75.
- Daselaar, S. M., Prince, S. E., & Cabeza, R. (2004). When less means more: Deactivations during encoding that predict subsequent memory. *NeuroImage*, 23(3), 921–927. <https://doi.org/10.1016/j.neuroimage.2004.07.031>

- Daugherty, A. M., Bender, A. R., Raz, N., & Ofen, N. (2016). Age differences in hippocampal subfield volumes from childhood to late adulthood: Lifespan Hippocampal Subfield Volumes. *Hippocampus*, *26*(2), 220–228. <https://doi.org/10.1002/hipo.22517>
- 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>
- 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 Sciences*, *100*(4), 2157–2162. <https://doi.org/10.1073/pnas.0337195100>
- 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>
- Desana Kocevaska, Ryan L. Muetzel, Annemarie I. Luik, Maartje P. C. M. Luijk, Vincent W. Jaddoe, Frank C. Verhulst, Tonya White, & Henning Tiemeier. (2017). The Developmental Course of Sleep Disturbances Across Childhood Relates to Brain Morphology at Age 7: The Generation R Study. *Sleep*, *40*(1). <https://doi.org/10.1093/sleep/zsw022>
- Desikan, R. S., Ségonne, F., Fischl, B., Quinn, B. T., Dickerson, B. C., Blacker, D., Buckner, R. L., Dale, A. M., Maguire, R. P., Hyman, B. T., Albert, M. S., & Killiany, R. J. (2006). An automated labeling system for subdividing the human cerebral cortex on MRI scans into gyral based regions of interest. *NeuroImage*, *31*(3), 968–980. <https://doi.org/10.1016/j.neuroimage.2006.01.021>

- Desrochers, P. C., Kurdziel, L. B. F., & Spencer, R. M. C. (2016). Delayed benefit of naps on motor learning in preschool children. *Experimental Brain Research*, 234(3), 763–772. <https://doi.org/10.1007/s00221-015-4506-3>
- Dick, A. S., Ralph, Y., Farrant, K., Reeb-Sutherland, B., Pruden, S., & Mattfeld, A. T. (2022). Volumetric development of hippocampal subfields and hippocampal white matter connectivity: Relationship with episodic memory. *Developmental Psychobiology*, 64(8). <https://doi.org/10.1002/dev.22333>
- Drummey, 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>
- Ducharme, S., Albaugh, M. D., Nguyen, T.-V., Hudziak, J. J., Mateos-Pérez, J. M., Labbe, A., Evan, A. C., Karama, S., & For the Brain Development Cooperative Group. (2016). Trajectories of cortical thickness maturation in normal brain development—The importance of quality control procedures—ScienceDirect. *NeuroImage*, 125, 267–279.
- Ducharme, S., Albaugh, M. D., Nguyen, T.-V., Hudziak, J. J., Mateos-Pérez, J. M., Labbe, A., Evans, A. C., & Karama, S. (2015). Trajectories of cortical surface area and cortical volume maturation in normal brain development. *Data in Brief*, 5, 929–938. <https://doi.org/10.1016/j.dib.2015.10.044>
- Duncan, S. C., Duncan, T. E., & Hops, H. (1996). Analysis of longitudinal data within accelerated longitudinal designs. *Psychological Methods*, 1, 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>

- Dutil, C., Walsh, J. J., Featherstone, R. B., Gunnell, K. E., Tremblay, M. S., Gruber, R., Weiss, S. K., Cote, K. A., Sampson, M., & Chaput, J.-P. (2018). Influence of sleep on developing brain functions and structures in children and adolescents: A systematic review. *Sleep Medicine Reviews, 42*, 184–201.
<https://doi.org/10.1016/j.smr.2018.08.003>
- Duvernoy, H., Cattin, F., & Risold, P.-Y. (2013). Structure, Functions, and Connections. In H. M. Duvernoy, F. Cattin, & P.-Y. Risold (Eds.), *The Human Hippocampus: Functional Anatomy, Vascularization and Serial Sections with MRI* (pp. 5–38). Springer.
https://doi.org/10.1007/978-3-642-33603-4_3
- Duvernoy, H. M. (2005). Sectional anatomy and magnetic resonance imaging. In *The hippocampus book: Functional anatomy, vascularization and serial sections with MRI* (3rd ed., pp. 129–217). Springer-Verlag.
- Eichenbaum, H. (2004). Hippocampus: Cognitive Processes and Neural Representations that Underlie Declarative Memory. *Neuron, 44*(1), 109–120.
<https://doi.org/10.1016/j.neuron.2004.08.028>
- Eichenbaum, H. (2017). Prefrontal–hippocampal interactions in episodic memory. *Nature Reviews Neuroscience, 18*(9), Article 9. <https://doi.org/10.1038/nrn.2017.74>
- Eichenbaum, H., Sauvage, M., Fortin, N., Komorowski, R., & Lipton, P. (2012). Towards a functional organization of episodic memory in the medial temporal lobe. *Neuroscience & Biobehavioral Reviews, 36*(7), 1597–1608.
<https://doi.org/10.1016/j.neubiorev.2011.07.006>

- Eichenbaum, H., Yonelinas, A. R., & Ranganath, C. (2007). The Medial Temporal Lobe and Recognition Memory. *Annual Review of Neuroscience*, *30*, 123–152.
<https://doi.org/10.1146/annurev.neuro.30.051606.094328>
- Esterline, K., & Gómez, R. L. (2021). The Role of Sleep in Retention of New Words in Habitually and Non-Habitually Napping Children. *Brain Sciences*, *11*(10), 1320.
<https://doi.org/10.3390/brainsci11101320>
- Evans, T. M., Kochalka, J., Ngoon, T. J., Wu, S. S., Qin, S., Battista, C., & Menon, V. (2015). Brain Structural Integrity and Intrinsic Functional Connectivity Forecast 6 Year Longitudinal Growth in Children’s Numerical Abilities. *Journal of Neuroscience*, *35*(33), 11743–11750. <https://doi.org/10.1523/JNEUROSCI.0216-15.2015>
- Ewell, A., Allard, T. L., Botdorf, M., & Riggins, T. (In Press). Emotion regulation and reactivity are associated with cortical thickness in early to mid-childhood. *Developmental Psychobiology*.
- Fandakova, Y., Leckey, S., Driver, C. C., Bunge, S. A., & Ghetti, S. (2019). Neural specificity of scene representations is related to memory performance in childhood. *NeuroImage*, *199*, 105–113. <https://doi.org/10.1016/j.neuroimage.2019.05.050>
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, *39*(2), 175–191. <https://doi.org/10.3758/BF03193146>
- Fischl, B. (2012). FreeSurfer. *NeuroImage*, *62*(2), 774–781.
<https://doi.org/10.1016/j.neuroimage.2012.01.021>
- Fischl, B., & Dale, A. M. (2000). Measuring the thickness of the human cerebral cortex from magnetic resonance images | PNAS. *Biological Sciences*, *97*(20), 11050–11055.

- Fischl, B., Salat, D. H., Busa, E., Albert, M., Dieterich, M., Haselgrove, C., van der Kouwe, A., Killiany, R., Kennedy, D., Klaveness, S., Montillo, A., Makris, N., Rosen, B., & Dale, A. M. (2002). Whole Brain Segmentation: Automated Labeling of Neuroanatomical Structures in the Human Brain. *Neuron*, 33(3), 341–355. [https://doi.org/10.1016/S0896-6273\(02\)00569-X](https://doi.org/10.1016/S0896-6273(02)00569-X)
- Fitter, M. H., Stern, J. A., Straske, M. D., Allard, T., Cassidy, J., & Riggins, T. (2022). Frontiers | Mothers' Attachment Representations and Children's Brain Structure. *Frontiers in Human Neuroscience*, 16. https://www.frontiersin.org/articles/10.3389/fnhum.2022.740195/full?&utm_source=Email_to_authors_&utm_medium=Email&utm_content=T1_11.5e1_author&utm_campaign=Email_publication&field=&journalName=Frontiers_in_Human_Neuroscience&id=740195
- Fjell, A. M., Grydeland, H., Krogstad, S. K., Amlien, I., Rohani, D. A., Ferschmann, L., Storsve, A. B., Tamnes, C. K., Sala-Llonch, R., Due-Tønnessen, P., Bjørnerud, A., Sølsnes, A. E., Håberg, A. K., Skranes, J., Bartsch, H., Chen, C.-H., Thompson, W. K., Panizzon, M. S., Kremen, W. S., ... Walhovd, K. B. (2015). Development and aging of cortical thickness correspond to genetic organization patterns. *Proceedings of the National Academy of Sciences*, 112(50), 15462–15467. <https://doi.org/10.1073/pnas.1508831112>
- Fjell, A. M., Sneve, M. H., Sederevicius, D., Sørensen, Ø., Krogstad, S. K., Mowinckel, A. M., & Walhovd, K. B. (2019). Volumetric and microstructural regional changes of the hippocampus underlying development of recall performance after extended retention intervals. *Developmental Cognitive Neuroscience*, 40, 100723. <https://doi.org/10.1016/j.dcn.2019.100723>

- Frangou, S., Modabbernia, A., Williams, S. C. R., Papachristou, E., Doucet, G. E., Agartz, I., Aghajani, M., Akudjedu, T. N., Albajes-Eizagirre, A., Alnæs, D., Alpert, K. I., Andersson, M., Andreasen, N. C., Andreassen, O. A., Asherson, P., Banaschewski, T., Bargallo, N., Baumeister, S., Baur-Streubel, R., ... Dima, D. (2022). Cortical thickness across the lifespan: Data from 17,075 healthy individuals aged 3–90 years. *Human Brain Mapping, 43*(1), 431–451. <https://doi.org/10.1002/hbm.25364>
- Galland, B. C., Taylor, B. J., Elder, D. E., & Herbison, P. (2012). Normal sleep patterns in infants and children: A systematic review of observational studies. *Sleep Medicine Reviews, 16*(3), 213–222. <https://doi.org/10.1016/j.smr.2011.06.001>
- Galván, A. (2020). The Need for Sleep in the Adolescent Brain. *Trends in Cognitive Sciences, 24*(1), 79–89. <https://doi.org/10.1016/j.tics.2019.11.002>
- Geng, F., Botdorf, M., & Riggins, T. (2021). How Behavior Shapes the Brain and the Brain Shapes Behavior: Insights from Memory Development. *Journal of Neuroscience, 41*(5), 981–990. <https://doi.org/10.1523/JNEUROSCI.2611-19.2020>
- Ghetti, S., & Bauer, P. J. (2012). *Origins and Development of Recollection: Perspectives from Psychology and Neuroscience*. Oxford University Press, USA.
- Ghetti, S., & Bunge, S. A. (2012). Neural changes underlying the development of episodic memory during middle childhood. *Developmental Cognitive Neuroscience, 2*(4), 381–395. <https://doi.org/10.1016/j.dcn.2012.05.002>
- Ghetti, S., & Fandakova, Y. (2020). Neural Development of Memory and Metamemory in Childhood and Adolescence: Toward an Integrative Model of the Development of Episodic Recollection. *Annual Review of Developmental Psychology, 2*(1), 365–388. <https://doi.org/10.1146/annurev-devpsych-060320-085634>

- Ghetti, S., & Lee, J. (2011). Children's episodic memory. *WIREs Cognitive Science*, 2(4), 365–373. <https://doi.org/10.1002/wcs.114>
- Giganti, F., Arzilli, C., Conte, F., Toselli, M., Viggiano, M. P., & Ficca, G. (2014). The Effect of a Daytime Nap on Priming and Recognition Tasks in Preschool Children. *Sleep*, 37(6), 1087–1093. <https://doi.org/10.5665/sleep.3766>
- Gilmore, A. W., Nelson, S. M., & McDermott, K. B. (2015). A parietal memory network revealed by multiple MRI methods. *Trends in Cognitive Sciences*, 19(9), 534–543. <https://doi.org/10.1016/j.tics.2015.07.004>
- Gogtay, N., Giedd, J. N., Lusk, L., Hayashi, K. M., Greenstein, D., Vaituzis, A. C., Nugent, T. F., Herman, D. H., Clasen, L. S., Toga, A. W., Rapoport, J. L., & Thompson, P. M. (2004). Dynamic mapping of human cortical development during childhood through early adulthood. *Proceedings of the National Academy of Sciences*, 101(21), 8174–8179. <https://doi.org/10.1073/pnas.0402680101>
- Golarai, G., Ghahremani, D. G., Whitfield-Gabrieli, S., Reiss, A., Eberhardt, J. L., Gabrieli, J. D. E., & Grill-Spector, K. (2007). Differential development of high-level visual cortex correlates with category-specific recognition memory. *Nature Neuroscience*, 10(4), Article 4. <https://doi.org/10.1038/nn1865>
- Guillery-Girard, B., Martins, S., Deshayes, S., Hertz-Pannier, L., Chiron, C., Jambaqué-Aubourg, I., Landeau, B., Clochon, P., Chetelat, G., & Eustache, F. (2013). Developmental Trajectories of Associative Memory from Childhood to Adulthood: A Behavioral and Neuroimaging Study. *Frontiers in Behavioral Neuroscience*, 7. <https://www.frontiersin.org/articles/10.3389/fnbeh.2013.00126>

- Hagler, D. J., Saygin, A. P., & Sereno, M. I. (2006). Smoothing and cluster thresholding for cortical surface-based group analysis of fMRI data. *NeuroImage*, 33(4), 1093–1103. <https://doi.org/10.1016/j.neuroimage.2006.07.036>
- Hansen, M., Simon, K. R., Strack, J., He, X., Noble, K. G., & Merz, E. C. (2022). Socioeconomic disparities in sleep duration are associated with cortical thickness in children. *Brain and Behavior*. <https://doi.org/10.1002/brb3.2859>
- Hill, D. E., Ciesielski, K. T., Hart, B. L., & Jung, R. E. (2004). MRI morphometric and neuropsychological correlates of long-term memory in survivors of childhood leukemia. *Pediatric Blood & Cancer*, 42(7), 611–617. <https://doi.org/10.1002/pbc.20004>
- Hoefl, F., McCandliss, B. D., Black, J. M., Gantman, A., Zakerani, N., Hulme, C., Lyytinen, H., Whitfield-Gabrieli, S., Glover, G. H., Reiss, A. L., & Gabrieli, J. D. E. (2011). Neural systems predicting long-term outcome in dyslexia. *Proceedings of the National Academy of Sciences*, 108(1), 361–366. <https://doi.org/10.1073/pnas.1008950108>
- Huijbers, W., Vannini, P., Sperling, R. A., C.m., P., Cabeza, R., & Daselaar, S. M. (2012). Explaining the encoding/retrieval flip: Memory-related deactivations and activations in the posteromedial cortex. *Neuropsychologia*, 50(14), 3764–3774. <https://doi.org/10.1016/j.neuropsychologia.2012.08.021>
- Hutchinson, J. B., Uncapher, M. R., & Wagner, A. D. (2009). Posterior parietal cortex and episodic retrieval: Convergent and divergent effects of attention and memory. *Learning & Memory*, 16(6), 343–356. <https://doi.org/10.1101/lm.919109>
- Huttenlocher, P. R. (1979). Synaptic density in human frontal cortex—Developmental changes and effects of aging. *Brain Research*, 163(2), 195–205. [https://doi.org/10.1016/0006-8993\(79\)90349-4](https://doi.org/10.1016/0006-8993(79)90349-4)

- Hutton, C., Draganski, B., Ashburner, J., & Weiskopf, N. (2009). A comparison between voxel-based cortical thickness and voxel-based morphometry in normal aging. *NeuroImage*, *48*(2), 371–380. <https://doi.org/10.1016/j.neuroimage.2009.06.043>
- Iglowstein, I., Jenni, O. G., Molinari, L., & Largo, R. H. (2003). Sleep Duration From Infancy to Adolescence: Reference Values and Generational Trends. *Pediatrics*, *111*(2), 302–307. <https://doi.org/10.1542/peds.111.2.302>
- Im, K., Lee, J.-M., Lyttelton, O., Kim, S. H., Evans, A. C., & Kim, S. I. (2008). Brain Size and Cortical Structure in the Adult Human Brain. *Cerebral Cortex*, *18*(9), 2181–2191. <https://doi.org/10.1093/cercor/bhm244>
- Jay N. Giedd, Jonathan Blumenthal, Neal O. Jeffries, F. X. Castellanos, Hong Liu, Alex Zijdenbos, Tomáš Paus, Alan C. Evans, & Judith L. Rapoport. (1999). Brain development during childhood and adolescence: A longitudinal MRI study | Nature Neuroscience. *Nature Neuroscience*, *2*, 861–863.
- Johnson, E. G., Prabhakar, J., Mooney, L. N., & Ghetti, S. (2020). Neuroimaging the sleeping brain: Insight on memory functioning in infants and toddlers. *Infant Behavior and Development*, *58*, 101427. <https://doi.org/10.1016/j.infbeh.2020.101427>
- Kaczurkin, A. N., Raznahan, A., & Satterthwaite, T. D. (2019). Sex differences in the developing brain: Insights from multimodal neuroimaging. *2019*, *44*, 71–85.
- Kaefer, K., Stella, F., McNaughton, B. L., & Battaglia, F. P. (2022). Replay, the default mode network and the cascaded memory systems model. *Nature Reviews Neuroscience*, *23*(10), Article 10. <https://doi.org/10.1038/s41583-022-00620-6>

- Kahn, I., Davachi, L., & Wagner, A. D. (2004). Functional-Neuroanatomic Correlates of Recollection: Implications for Models of Recognition Memory. *Journal of Neuroscience*, 24(17), 4172–4180. <https://doi.org/10.1523/JNEUROSCI.0624-04.2004>
- Karanian, J. M., & Slotnick, S. D. (2015). Memory for shape reactivates the lateral occipital complex. *Brain Research*, 1603, 124–132. <https://doi.org/10.1016/j.brainres.2015.01.024>
- Kastner, S., & Ungerleider, L. G. (2001). The neural basis of biased competition in human visual cortex. *Neuropsychologia*, 39(12), 1263–1276. [https://doi.org/10.1016/S0028-3932\(01\)00116-6](https://doi.org/10.1016/S0028-3932(01)00116-6)
- 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>
- Keresztes, A., Raffington, L., Bender, A. R., Bögl, K., Heim, C., & Shing, Y. L. (2022). Longitudinal developmental trajectories do not follow cross-sectional age associations in hippocampal subfield and memory development. *Developmental Cognitive Neuroscience*, 54, 101085. <https://doi.org/10.1016/j.dcn.2022.101085>
- Klijin, N., Müller, N., McQueen, J., & Fernández, G. (2016). Effects of Structural and Functional Prefrontal Cortex Maturation on Verbal Memory Development. *Nijmegen CNS*, 12(1).
- Konishi, S., Wheeler, M. E., Donaldson, D. I., & Buckner, R. L. (2000). Neural Correlates of Episodic Retrieval Success. *NeuroImage*, 12(3), 276–286. <https://doi.org/10.1006/nimg.2000.0614>
- Kopasz, M., Loessl, B., Hornyak, M., Riemann, D., Nissen, C., Piosczyk, H., & Voderholzer, U. (2010). Sleep and memory in healthy children and adolescents – A critical review. *Sleep Medicine Reviews*, 14(3), 167–177. <https://doi.org/10.1016/j.smr.2009.10.006>

- Kurdziel, L. B. F., Kent, J., & Spencer, R. M. C. (2018). Sleep-dependent enhancement of emotional memory in early childhood. *Scientific Reports*, 8(1), 12609. <https://doi.org/10.1038/s41598-018-30980-y>
- Kurdziel, L., Duclos, K., & Spencer, R. M. C. (2013). Sleep spindles in midday naps enhance learning in preschool children. *Proceedings of the National Academy of Sciences*, 110(43), 17267–17272. <https://doi.org/10.1073/pnas.1306418110>
- Kurth, S., Lassonde, J. M., Pierpoint, L. A., Rusterholz, T., Jenni, O. G., McClain, I. J., Achermann, P., & LeBourgeois, M. K. (2016). Development of nap neurophysiology: Preliminary insights into sleep regulation in early childhood. *Journal of Sleep Research*, 25(6), 646–654. <https://doi.org/10.1111/jsr.12427>
- Lavenex, P., & Amaral, D. G. (2000). Hippocampal-neocortical interaction: A hierarchy of associativity. *Hippocampus*, 10(4), 420–430. [https://doi.org/10.1002/1098-1063\(2000\)10:4<420::AID-HIPO8>3.0.CO;2-5](https://doi.org/10.1002/1098-1063(2000)10:4<420::AID-HIPO8>3.0.CO;2-5)
- 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>
- 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., Gogtay, N., Greenstein, D. K., Wells, E. M., Wallace, G. L., Clasen, L. S., Blumenthal, J. D., Lerch, J., Zijdenbos, A. P., Evans, A. C., Thompson, P. M., & Giedd, J. N. (2007). Sexual dimorphism of brain developmental trajectories during childhood and adolescence. *NeuroImage, 36*(4), 1065–1073. <https://doi.org/10.1016/j.neuroimage.2007.03.053>
- Leong, R. L. F., Lo, J. C., & Chee, M. W. L. (2022). Systematic review and meta-analyses on the effects of afternoon napping on cognition. *Sleep Medicine Reviews, 65*, 101666. <https://doi.org/10.1016/j.smr.2022.101666>
- Li, G., Lin, W., Gilmore, J. H., & Shen, D. (2015). Spatial Patterns, Longitudinal Development, and Hemispheric Asymmetries of Cortical Thickness in Infants from Birth to 2 Years of Age. *Journal of Neuroscience, 35*(24), 9150–9162. <https://doi.org/10.1523/JNEUROSCI.4107-14.2015>
- Li, G., Nie, J., Wang, L., Shi, F., Lin, W., Gilmore, J. H., & Shen, D. (2013). Mapping Region-Specific Longitudinal Cortical Surface Expansion from Birth to 2 Years of Age. *Cerebral Cortex, 23*(11), 2724–2733. <https://doi.org/10.1093/cercor/bhs265>
- Li, G., Nie, J., Wang, L., Shi, F., Lyall, A. E., Lin, W., Gilmore, J. H., & Shen, D. (2014). Mapping Longitudinal Hemispheric Structural Asymmetries of the Human Cerebral Cortex From Birth to 2 Years of Age. *Cerebral Cortex, 24*(5), 1289–1300. <https://doi.org/10.1093/cercor/bhs413>

- Lloyd, M. E., Doydum, A. O., & Newcombe, N. S. (2009). Memory Binding in Early Childhood: Evidence for a Retrieval Deficit. *Child Development, 80*(5), 1321–1328.
<https://doi.org/10.1111/j.1467-8624.2009.01353.x>
- Lokhandwala, S., & Spencer, R. M. C. (2021). Slow wave sleep in naps supports episodic memories in early childhood. *Developmental Science, 24*(2).
<https://doi.org/10.1111/desc.13035>
- Lokhandwala, S., & Spencer, R. M. C. (2022). Relations between sleep patterns early in life and brain development: A review. *Developmental Cognitive Neuroscience, 56*, 101130.
<https://doi.org/10.1016/j.dcn.2022.101130>
- Lorsbach, T. C., & Reimer, J. F. (2005). Feature Binding in Children and Young Adults. *The Journal of Genetic Psychology, 166*(3), 313–328.
<https://doi.org/10.3200/GNTP.166.3.313-328>
- Lucan, J. N., Foxe, J. J., Gomez-Ramirez, M., Sathian, K., & Molholm, S. (2010). Tactile shape discrimination recruits human lateral occipital complex during early perceptual processing. *Human Brain Mapping, 31*(11), 1813–1821.
<https://doi.org/10.1002/hbm.20983>
- Lukowski, A. F., & Bauer, P. J. (2013). Long-Term Memory in Infancy and Early Childhood. In *The Wiley Handbook on the Development of Children's Memory* (pp. 230–254). John Wiley & Sons, Ltd. <https://doi.org/10.1002/9781118597705.ch11>
- Lyall, A. E., Shi, F., Geng, X., Woolson, S., Li, G., Wang, L., Hamer, R. M., Shen, D., & Gilmore, J. H. (2015). Dynamic Development of Regional Cortical Thickness and Surface Area in Early Childhood. *Cerebral Cortex, 25*(8), 2204–2212.
<https://doi.org/10.1093/cercor/bhu027>

- MacCallum, R. C., Browne, M. W., & Cai, L. (2006). Testing differences between nested covariance structure models: Power analysis and null hypotheses. *Psychological Methods, 11*, 19–35. <https://doi.org/10.1037/1082-989X.11.1.19>
- Maret, S., Faraguna, U., Nelson, A. B., Cirelli, C., & Tononi, G. (2011). Sleep and wake modulate spine turnover in the adolescent mouse cortex. *Nature Neuroscience, 14*(11), 1418–1420. <https://doi.org/10.1038/nn.2934>
- Mason, G. M., Lokhandwala, S., Riggins, T., & Spencer, R. M. C. (2021). Sleep and human cognitive development. *Sleep Medicine Reviews, 57*, 101472. <https://doi.org/10.1016/j.smr.2021.101472>
- Mathews, M. E., & Fozard, J. L. (1970). Age differences in judgments of recency for short sequences of pictures. *Developmental Psychology, 3*, 208–217. <https://doi.org/10.1037/h0029582>
- Matricciani, L., Olds, T., & Petkov, J. (2012). In search of lost sleep: Secular trends in the sleep time of school-aged children and adolescents. *Sleep Medicine Reviews, 16*(3), 203–211. <https://doi.org/10.1016/j.smr.2011.03.005>
- 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>
- McDevitt, E. A., Sattari, N., Duggan, K. A., Cellini, N., Whitehurst, L. N., Perera, C., Reihanabad, N., Granados, S., Hernandez, L., & Mednick, S. C. (2018). The impact of frequent napping and nap practice on sleep-dependent memory in humans. *Scientific Reports, 8*(1), 15053. <https://doi.org/10.1038/s41598-018-33209-0>

- Mechelli, A., Humphreys, G. W., Mayall, K., Olson, A., & Price, C. J. (2000). Differential effects of word length and visual contrast in the fusiform and lingual gyri during. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 267(1455), 1909–1913. <https://doi.org/10.1098/rspb.2000.1229>
- Meltzoff, A. N. (1988). Infant Imitation and Memory: Nine-Month-Olds in Immediate and Deferred Tests. *Child Development*, 59(1), 217–225. <https://doi.org/10.2307/1130404>
- Mills, K. L., Lalonde, F., Clasen, L. S., Giedd, J. N., & Blakemore, S.-J. (2014). Developmental changes in the structure of the social brain in late childhood and adolescence. *Social Cognitive and Affective Neuroscience*, 9(1), 123–131. <https://doi.org/10.1093/scan/nss113>
- Miotto, E. C., Balardin, J. B., Martin, M. da G. M., Polanczyk, G. V., Savage, C. R., Miguel, E. C., & Batistuzzo, M. C. (2020). Effects of semantic categorization strategy training on episodic memory in children and adolescents. *PLOS ONE*, 15(2), e0228866. <https://doi.org/10.1371/journal.pone.0228866>
- Muthén, B., & Muthén, L. (2017). Mplus. In *Handbook of Item Response Theory*. Chapman and Hall/CRC.
- Muthén, L. K., & Muthén, B. O. (2002). How to Use a Monte Carlo Study to Decide on Sample Size and Determine Power. *Structural Equation Modeling: A Multidisciplinary Journal*, 9(4), 599–620. https://doi.org/10.1207/S15328007SEM0904_8
- Mutlu, A. K., Schneider, M., Debbané, M., Badoud, D., Eliez, S., & Schaer, M. (2013). Sex differences in thickness, and folding developments throughout the cortex. *NeuroImage*, 82, 200–207. <https://doi.org/10.1016/j.neuroimage.2013.05.076>

- Ngo, C. T., Alm, K. H., Metoki, A., Hampton, W., Riggins, T., Newcombe, N. S., & Olson, I. R. (2017). White matter structural connectivity and episodic memory in early childhood. *Developmental Cognitive Neuroscience, 28*, 41–53. <https://doi.org/10.1016/j.dcn.2017.11.001>
- Nie, J., Li, G., Wang, L., Shi, F., Lin, W., Gilmore, J. H., & Shen, D. (2014). Longitudinal development of cortical thickness, folding, and fiber density networks in the first 2 years of life. *Human Brain Mapping, 35*(8), 3726–3737. <https://doi.org/10.1002/hbm.22432>
- Norman, K. A., & O'Reilly, R. C. (2003). Modeling hippocampal and neocortical contributions to recognition memory: A complementary-learning-systems approach. *Psychological Review, 110*, 611–646. <https://doi.org/10.1037/0033-295X.110.4.611>
- Nyberg, L., Persson, J., Habib, R., Tulving, E., McIntosh, A. R., Cabeza, R., & Houle, S. (2000). Large Scale Neurocognitive Networks Underlying Episodic Memory. *Journal of Cognitive Neuroscience, 12*(1), 163–173. <https://doi.org/10.1162/089892900561805>
- 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), Article 9. <https://doi.org/10.1038/nn1950>
- Ohayon, M. M., Carskadon, M. A., Guilleminault, C., & Vitiello, M. V. (2004). Meta-Analysis of Quantitative Sleep Parameters From Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values Across the Human Lifespan. *Sleep, 27*(7), 1255–1273. <https://doi.org/10.1093/sleep/27.7.1255>
- Osborne, J. W., & Overbay, A. (2004). “The power of outliers (and why researchers should ALWAYS check for the” by Jason W. Osborne and Amy Overbay. *Practical Assessment, Research, and Evaluation, 9*(1). <https://scholarworks.umass.edu/pare/vol9/iss1/6/>

- Østby, Y., Tamnes, C. K., Fjell, A. M., & Walhovd, K. B. (2012). Dissociating memory processes in the developing brain: The role of hippocampal volume and cortical thickness in recall after minutes versus days. *Cerebral Cortex (New York, N.Y.: 1991)*, *22*(2), 381–390. <https://doi.org/10.1093/cercor/bhr116>
- Pathman, T., & Ghetti, S. (2014). The Eyes Know Time: A Novel Paradigm to Reveal the Development of Temporal Memory. *Child Development*, *85*(2), 792–807. <https://doi.org/10.1111/cdev.12152>
- Petanjek, Z., Judaš, M., Šimić, G., Rašin, M. R., Uylings, H. B. M., Rakic, P., & Kostović, I. (2011). Extraordinary neoteny of synaptic spines in the human prefrontal cortex. *Proceedings of the National Academy of Sciences*, *108*(32), 13281–13286. <https://doi.org/10.1073/pnas.1105108108>
- Piaget, J. (1952). *The origins of intelligence in children* (5th ed., 1–8). International Universities Press.
- Picard, L., Cousin, S., Guillery-Girard, B., Eustache, F., & Piolino, P. (2012). How Do the Different Components of Episodic Memory Develop? Role of Executive Functions and Short-Term Feature-Binding Abilities: How Does Episodic Memory Develop? *Child Development*, *83*(3), 1037–1050. <https://doi.org/10.1111/j.1467-8624.2012.01736.x>
- 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>
- Poppenk, J., & Moscovitch, M. (2011). A Hippocampal Marker of Recollection Memory Ability among Healthy Young Adults: Contributions of Posterior and Anterior Segments. *Neuron*, *72*(6), 931–937. <https://doi.org/10.1016/j.neuron.2011.10.014>

- Preacher, K. J., & Coffman, D. L. (2006). *Power and Sample Size using RMSEA* [Computer software]. <http://www.quantpsy.org/rmse/rmse.htm>
- Raffington, L., Czamara, D., Mohn, J. J., Falck, J., Schmall, V., Heim, C., Binder, E. B., & Shing, Y. L. (2019). Stable longitudinal associations of family income with children's hippocampal volume and memory persist after controlling for polygenic scores of educational attainment. *Developmental Cognitive Neuroscience, 40*, 100720. <https://doi.org/10.1016/j.dcn.2019.100720>
- Rakic, P. (2009). Evolution of the neocortex: A perspective from developmental biology. *Nature Reviews Neuroscience, 10*(10), 724–735. <https://doi.org/10.1038/nrn2719>
- Randazzo, A. C., Muehlbach, M. J., Schweitzer, P. K., & Walsh, J. K. (1998). Cognitive Function Following Acute Sleep Restriction in Children Ages 10–14. *Sleep*. <https://doi.org/10.1093/sleep/21.8.861>
- Rasch, B., & Born, J. (2013). About Sleep's Role in Memory. *Physiological Reviews, 93*(2), 681–766. <https://doi.org/10.1152/physrev.00032.2012>
- Raznahan, A., Shaw, P., Lalonde, F., Stockman, M., Wallace, G. L., Greenstein, D., Clasen, L., Gogtay, N., & Giedd, J. N. (2011). How Does Your Cortex Grow? *Journal of Neuroscience, 31*(19), 7174–7177. <https://doi.org/10.1523/JNEUROSCI.0054-11.2011>
- Remer, J., Croteau-Chonka, E., Dean, D. C., D'Arpino, S., Dirks, H., Whiley, D., & Deoni, S. C. L. (2017). Quantifying cortical development in typically developing toddlers and young children, 1–6 years of age. *NeuroImage, 153*, 246–261. <https://doi.org/10.1016/j.neuroimage.2017.04.010>

- Riggins, T. (2014). Longitudinal investigation of source memory reveals different developmental trajectories for item memory and binding. *Developmental Psychology, 50*, 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., 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., & Spencer, R. M. C. (2020). Habitual sleep is associated with both source memory and hippocampal subfield volume during early childhood. *Scientific Reports, 10*(1), 15304. <https://doi.org/10.1038/s41598-020-72231-z>
- Rolls, E. T. (2022). The hippocampus, ventromedial prefrontal cortex, and episodic and semantic memory. *Progress in Neurobiology, 217*, 102334.
<https://doi.org/10.1016/j.pneurobio.2022.102334>
- Rosen, M. L., Sheridan, M. A., Sambrook, K. A., Peverill, M. R., Meltzoff, A. N., & McLaughlin, K. A. (2018). The Role of Visual Association Cortex in Associative Memory Formation across Development. *Journal of Cognitive Neuroscience, 30*(3), 365–380. https://doi.org/10.1162/jocn_a_01202

- Sandoval, M., Leclerc, J. A., & Gómez, R. L. (2017). Words to Sleep On: Naps Facilitate Verb Generalization in Habitually and Nonhabitually Napping Preschoolers. *Child Development, 88*(5), 1615–1628. <https://doi.org/10.1111/cdev.12723>
- 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
- Schnack, H. G., van Haren, N. E. M., Brouwer, R. M., Evans, A., Durston, S., Boomsma, D. I., Kahn, R. S., & Hulshoff Pol, H. E. (2015). Changes in Thickness and Surface Area of the Human Cortex and Their Relationship with Intelligence. *Cerebral Cortex, 25*(6), 1608–1617. <https://doi.org/10.1093/cercor/bht357>
- Schommartz, I., Lembcke, P. F., Pupillo, F., Schuetz, H., de Chamorro, N. W., Bauer, M., Kaindl, A. M., Buss, C., & Shing, Y. L. (2023). Distinct multivariate structural brain profiles are related to variations in short- and long-delay memory consolidation across children and young adults. *Developmental Cognitive Neuroscience, 59*, 101192. <https://doi.org/10.1016/j.dcn.2022.101192>
- Scoville, W. B., & Milner, B. (1957). Loss of Recent Memory After Bilateral Hippocampal Lesions. *Journal of Neurology, Neurosurgery & Psychiatry, 20*(1), 11–21. <https://doi.org/10.1136/jnnp.20.1.11>
- Sestieri, C., Shulman, G. L., & Corbetta, M. (2010). Attention to Memory and the Environment: Functional Specialization and Dynamic Competition in Human Posterior Parietal Cortex. *Journal of Neuroscience, 30*(25), 8445–8456. <https://doi.org/10.1523/JNEUROSCI.4719-09.2010>

- Sestieri, C., Shulman, G. L., & Corbetta, M. (2017). The contribution of the human posterior parietal cortex to episodic memory. *Nature Reviews Neuroscience*, *18*(3), Article 3. <https://doi.org/10.1038/nrn.2017.6>
- Shaw, P., Eckstrand, K., Sharp, W., Blumenthal, J., Lerch, J. P., Greenstein, D., Clasen, L., Evans, A., Giedd, J., & Rapoport, J. L. (2007). Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proceedings of the National Academy of Sciences*, *104*(49), 19649–19654. <https://doi.org/10.1073/pnas.0707741104>
- Shimamura, A. P. (1995). Memory and frontal lobe function. In *The cognitive neurosciences* (pp. 803–813). The MIT Press.
- Shing, Y. L., & Lindenberger, U. (2011). The Development of Episodic Memory: Lifespan Lessons. *Child Development Perspectives*, *5*(2), 148–155. <https://doi.org/10.1111/j.1750-8606.2011.00170.x>
- Shing, Y. L., Werkle-Bergner, M., Brehmer, Y., Müller, V., Li, S.-C., & Lindenberger, U. (2010). Episodic memory across the lifespan: The contributions of associative and strategic components. *Neuroscience & Biobehavioral Reviews*, *34*(7), 1080–1091. <https://doi.org/10.1016/j.neubiorev.2009.11.002>
- Short, M. A., Blunden, S., Rigney, G., Matricciani, L., Coussens, S., M. Reynolds, C., & Galland, B. (2018). Cognition and objectively measured sleep duration in children: A systematic review and meta-analysis. *Sleep Health*, *4*(3), 292–300. <https://doi.org/10.1016/j.sleh.2018.02.004>
- Sluzenski, J., Newcombe, N. S., & Kovacs, S. L. (2006). Binding, relational memory, and recall of naturalistic events: A developmental perspective. *Journal of Experimental Psychology*:

- Learning, Memory, and Cognition*, 32, 89–100. <https://doi.org/10.1037/0278-7393.32.1.89>
- Small, S. A. (2002). The Longitudinal Axis of the Hippocampal Formation: Its Anatomy, Circuitry, and Role in Cognitive Function. *Reviews in the Neurosciences*, 13(2), 183–194. <https://doi.org/10.1515/REVNEURO.2002.13.2.183>
- Snodgrass, J. G., & Corwin, J. (1988). Pragmatics of measuring recognition memory: Applications to dementia and amnesia. *Journal of Experimental Psychology: General*, 117, 34–50. <https://doi.org/10.1037/0096-3445.117.1.34>
- Sowell, E. R., Delis, D., Stiles, J., & Jernigan, T. L. (2001). Improved memory functioning and frontal lobe maturation between childhood and adolescence: A structural MRI study. *Journal of the International Neuropsychological Society*, 7(3), 312–322. <https://doi.org/10.1017/S135561770173305X>
- Sowell, E. R., Thompson, P. M., Leonard, C. M., Welcome, S. E., Kan, E., & Toga, A. W. (2004). Longitudinal Mapping of Cortical Thickness and Brain Growth in Normal Children. *Journal of Neuroscience*, 24(38), 8223–8231. <https://doi.org/10.1523/JNEUROSCI.1798-04.2004>
- Spaniol, J., Davidson, P. S. R., Kim, A. S. N., Han, H., Moscovitch, M., & Grady, C. L. (2009). Event-related fMRI studies of episodic encoding and retrieval: Meta-analyses using activation likelihood estimation. *Neuropsychologia*, 47(8), 1765–1779. <https://doi.org/10.1016/j.neuropsychologia.2009.02.028>
- Spanò, G., Gómez, R. L., Demara, B. I., Alt, M., Cowen, S. L., & Edgin, J. O. (2018). REM sleep in naps differentially relates to memory consolidation in typical preschoolers and

- children with Down syndrome. *Proceedings of the National Academy of Sciences*, *115*(46), 11844–11849. <https://doi.org/10.1073/pnas.1811488115>
- Spencer, R. M. C., & Riggins, T. (2022). Contributions of memory and brain development to the bioregulation of naps and nap transitions in early childhood. *Proceedings of the National Academy of Sciences*, *119*(44), e2123415119. <https://doi.org/10.1073/pnas.2123415119>
- Squeglia, L. M., Jacobus, J., Sorg, S. F., Jernigan, T. L., & Tapert, S. F. (2013). Early Adolescent Cortical Thinning Is Related to Better Neuropsychological Performance. *Journal of the International Neuropsychological Society*, *19*(9), 962–970. <https://doi.org/10.1017/S1355617713000878>
- St. Laurent, C. W., Lokhandwala, S., Allard, T., Ji, A., Riggins, T., & Spencer, R. M. (2022). Influence of naps on sedentary time and physical activity in early childhood. *Scientific Reports*, *12*(1), 21198.
- Staresina, B. P., & Davachi, L. (2008). Selective and Shared Contributions of the Hippocampus and Perirhinal Cortex to Episodic Item and Associative Encoding. *Journal of Cognitive Neuroscience*, *20*(8), 1478–1489. <https://doi.org/10.1162/jocn.2008.20104>
- Staton, S., Rankin, P. S., Harding, M., Smith, S. S., Westwood, E., LeBourgeois, M. K., & Thorpe, K. J. (2020). Many naps, one nap, none: A systematic review and meta-analysis of napping patterns in children 0–12 years. *Sleep Medicine Reviews*, *50*, 101247. <https://doi.org/10.1016/j.smr.2019.101247>
- Stern, C. E., Corkin, S., González, R. G., Guimaraes, A. R., Baker, J. R., Jennings, P. J., Carr, C. A., Sugiura, R. M., Vedantham, V., & Rosen, B. R. (1996). The hippocampal formation participates in novel picture encoding: Evidence from functional magnetic resonance

- imaging. *Proceedings of the National Academy of Sciences*, 93(16), 8660–8665.
<https://doi.org/10.1073/pnas.93.16.8660>
- Stevens, W. D., Hasher, L., Chiew, K. S., & Grady, C. L. (2008). A Neural Mechanism Underlying Memory Failure in Older Adults. *Journal of Neuroscience*, 28(48), 12820–12824. <https://doi.org/10.1523/JNEUROSCI.2622-08.2008>
- 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), Article 10.
<https://doi.org/10.1038/nrn3785>
- Supekar, K., Chang, H., Mistry, P. K., Iuculano, T., & Menon, V. (2021). Neurocognitive modeling of latent memory processes reveals reorganization of hippocampal-cortical circuits underlying learning and efficient strategies. *Communications Biology*, 4(1), 405.
<https://doi.org/10.1038/s42003-021-01872-1>
- Szczepanski, S. M., Pinsk, M. A., Douglas, M. M., Kastner, S., & Saalman, Y. B. (2013). Functional and structural architecture of the human dorsal frontoparietal attention network. *Proceedings of the National Academy of Sciences*, 110(39), 15806–15811.
<https://doi.org/10.1073/pnas.1313903110>
- Taki, Y., Hashizume, H., Thyreau, B., Sassa, Y., Takeuchi, H., Wu, K., Kotozaki, Y., Nouchi, R., Asano, M., Asano, K., Fukuda, H., & Kawashima, R. (2012). Sleep duration during weekdays affects hippocampal gray matter volume in healthy children. *NeuroImage*, 60(1), 471–475. <https://doi.org/10.1016/j.neuroimage.2011.11.072>
- Tamnes, C. K., Bos, M. G. N., van de Kamp, F. C., Peters, S., & Crone, E. A. (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., Herting, M. M., Goddings, A.-L., Meuwese, R., Blakemore, S.-J., Dahl, R. E., Güroğlu, B., Raznahan, A., Sowell, E. R., Crone, E. A., & Mills, K. L. (2017). Development of the Cerebral Cortex across Adolescence: A Multisample Study of Inter-Related Longitudinal Changes in Cortical Volume, Surface Area, and Thickness. *The Journal of Neuroscience*, 37(12), 3402–3412. <https://doi.org/10.1523/JNEUROSCI.3302-16.2017>
- Tamnes, C. K., Walhovd, K. B., Dale, A. M., Østby, Y., Grydeland, H., Richardson, G., Westlye, L. T., Roddey, J. C., Hagler, D. J., Due-Tønnessen, P., Holland, D., & Fjell, A. M. (2013). Brain development and aging: Overlapping and unique patterns of change. *NeuroImage*, 68, 63–74. <https://doi.org/10.1016/j.neuroimage.2012.11.039>
- Tang, L., Shafer, A. T., & Ofen, N. (2018). Prefrontal Cortex Contributions to the Development of Memory Formation. *Cerebral Cortex*, 28(9), 3295–3308.
<https://doi.org/10.1093/cercor/bhx200>
- Teghil, A., Bonavita, A., Guariglia, C., & Boccia, M. (2021). Commonalities and specificities between environmental navigation and autobiographical memory: A synthesis and a theoretical perspective. *Neuroscience & Biobehavioral Reviews*, 127, 928–945.
<https://doi.org/10.1016/j.neubiorev.2021.06.012>
- Tononi, G., & Cirelli, C. (2014). Sleep and the Price of Plasticity: From Synaptic and Cellular Homeostasis to Memory Consolidation and Integration. *Neuron*, 81(1), 12–34.
<https://doi.org/10.1016/j.neuron.2013.12.025>

- Tulving, E. (1972). Episodic and semantic memory. In *Organization of memory* (pp. xiii, 423–xiii, 423). Academic Press.
- Tulving, E. (1984). Précis of Elements of episodic memory | Behavioral and Brain Sciences | Cambridge Core. *Behavioral and Brain Sciences*, 7(2), 223–238.
- 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>
- Van Petten, C. (2004). Relationship between hippocampal volume and memory ability in healthy individuals across the lifespan: Review and meta-analysis. *Neuropsychologia*, 42(10), 1394–1413. <https://doi.org/10.1016/j.neuropsychologia.2004.04.006>
- Vannini, P., Hedden, T., Huijbers, W., Ward, A., Johnson, K. A., & Sperling, R. A. (2013). The Ups and Downs of the Posteromedial Cortex: Age- and Amyloid-Related Functional Alterations of the Encoding/Retrieval Flip in Cognitively Normal Older Adults. *Cerebral Cortex*, 23(6), 1317–1328. <https://doi.org/10.1093/cercor/bhs108>
- Vannini, P., O'Brien, J., O'Keefe, K., Pihlajamäki, M., LaViolette, P., & Sperling, R. A. (2011). What Goes Down Must Come Up: Role of the Posteromedial Cortices in Encoding and Retrieval. *Cerebral Cortex*, 21(1), 22–34. <https://doi.org/10.1093/cercor/bhq051>
- Vriend, J. L., Davidson, F. D., Corkum, P. V., Rusak, B., Chambers, C. T., & McLaughlin, E. N. (2013). Manipulating Sleep Duration Alters Emotional Functioning and Cognitive Performance in Children. *Journal of Pediatric Psychology*, 38(10), 1058–1069. <https://doi.org/10.1093/jpepsy/jst033>
- Vriend, J. L., Davidson, F. D., Corkum, P. V., Rusak, B., McLaughlin, E. N., & Chambers, C. T. (2012). Sleep Quantity and Quality in Relation to Daytime Functioning in Children. *Children's Health Care*, 41(3), 204–222. <https://doi.org/10.1080/02739615.2012.685039>

- Wagner, A. D., Schacter, D. L., Rotte, M., Koutstaal, W., Maril, A., Dale, A. M., Rosen, B. R., & Buckner, R. L. (1998). Building Memories: Remembering and Forgetting of Verbal Experiences as Predicted by Brain Activity. *Science*, *281*(5380), 1188–1191.
<https://doi.org/10.1126/science.281.5380.1188>
- Wagner, A. D., Shannon, B. J., Kahn, I., & Buckner, R. L. (2005). Parietal lobe contributions to episodic memory retrieval. *Trends in Cognitive Sciences*, *9*(9), 445–453.
<https://doi.org/10.1016/j.tics.2005.07.001>
- Walhovd, K. B., Fjell, A. M., Giedd, J., Dale, A. M., & Brown, T. T. (2017). Through Thick and Thin: A Need to Reconcile Contradictory Results on Trajectories in Human Cortical Development. *Cerebral Cortex*, *27*(2), bhv301. <https://doi.org/10.1093/cercor/bhv301>
- Wang, H., Nation, K., Gaskell, M. G., Robidoux, S., Weighall, A., & Castles, A. (2022). Nap effects on preschool children’s learning of letter-sound mappings. *Child Development*, *93*(4), 1145–1153. <https://doi.org/10.1111/cdev.13753>
- Wang, W.-C., Yonelinas, A. P., & Ranganath, C. (2013). Dissociable neural correlates of item and context retrieval in the medial temporal lobes. *Behavioural Brain Research*, *254*, 102–107. <https://doi.org/10.1016/j.bbr.2013.05.029>
- Wang, Y., Hao, L., Zhang, Y., Zuo, C., & Wang, D. (2019). Entorhinal cortex volume, thickness, surface area and curvature trajectories over the adult lifespan. *Psychiatry Research: Neuroimaging*, *292*, 47–53. <https://doi.org/10.1016/j.psychresns.2019.09.002>
- Weissbluth, M. (1995). Naps in Children: 6 Months– 7 Years. *Sleep*, *18*(2), 82–87.
<https://doi.org/10.1093/sleep/18.2.82>

- Wendelken, C., Baym, C. L., Gazzaley, A., & Bunge, S. A. (2011). Neural indices of improved attentional modulation over middle childhood. *Developmental Cognitive Neuroscience*, *1*(2), 175–186. <https://doi.org/10.1016/j.dcn.2010.11.001>
- Wheeler, M. E., & Buckner, R. L. (2003). Functional Dissociation among Components of Remembering: Control, Perceived Oldness, and Content. *Journal of Neuroscience*, *23*(9), 3869–3880. <https://doi.org/10.1523/JNEUROSCI.23-09-03869.2003>
- Whittaker, T. A., & Khojasteh, J. (2017). Detecting Appropriate Trajectories of Growth in Latent Growth Models: The Performance of Information-Based Criteria. *The Journal of Experimental Education*, *85*(2), 215–230. <https://doi.org/10.1080/00220973.2015.1123669>
- Wierenga, L. M., Langen, M., Oranje, B., & Durston, S. (2014). Unique developmental trajectories of cortical thickness and surface area. *NeuroImage*, *87*, 120–126. <https://doi.org/10.1016/j.neuroimage.2013.11.010>
- Williams, S. E., & Horst, J. S. (2014). Goodnight book: Sleep consolidation improves word learning via storybooks. *Frontiers in Psychology*, *5*. <https://doi.org/10.3389/fpsyg.2014.00184>
- Willoughby, K. A., Sheard, E. D., Nash, K., & Rovet, J. (2008). Effects of prenatal alcohol exposure on hippocampal volume, verbal learning, and verbal and spatial recall in late childhood. *Journal of the International Neuropsychological Society*, *14*(6), 1022–1033.
- Wing, E. A., Ritchey, M., & Cabeza, R. (2015). Reinstatement of Individual Past Events Revealed by the Similarity of Distributed Activation Patterns during Encoding and Retrieval. *Journal of Cognitive Neuroscience*, *27*(4), 679–691. https://doi.org/10.1162/jocn_a_00740

- Winkler, A. M., Kochunov, P., Blangero, J., Almasy, L., Zilles, K., Fox, P. T., Duggirala, R., & Glahn, D. C. (2010). Cortical Thickness or Grey Matter Volume? The Importance of Selecting the Phenotype for Imaging Genetics Studies. *NeuroImage*, *53*(3), 1135–1146. <https://doi.org/10.1016/j.neuroimage.2009.12.028>
- Wixted, J. T., & Squire, L. R. (2011). The medial temporal lobe and the attributes of memory. *Trends in Cognitive Sciences*, *15*(5), 210–217. <https://doi.org/10.1016/j.tics.2011.03.005>
- Yang, G., & Gan, W.-B. (2012). Sleep contributes to dendritic spine formation and elimination in the developing mouse somatosensory cortex. *Developmental Neurobiology*, *72*(11), 1391–1398. <https://doi.org/10.1002/dneu.20996>
- Yassa, M. A., & Stark, C. E. L. (2011). Pattern separation in the hippocampus. *Trends in Neurosciences*, *34*(10), 515–525. <https://doi.org/10.1016/j.tins.2011.06.006>
- Yim, H., Dennis, S. J., & Sloutsky, V. M. (2013). The Development of Episodic Memory. *Psychological Science*, *24*(11), 2163–2172.
- Yu, Q., Daugherty, A. M., Anderson, D. M., Nishimura, M., Brush, D., Hardwick, A., Lacey, W., Raz, S., & Ofen, N. (2018). Socioeconomic status and hippocampal volume in children and young adults. *Developmental Science*, *21*(3), e12561. <https://doi.org/10.1111/desc.12561>
- Yu, Q., McCall, D. M., Homayouni, R., Tang, L., Chen, Z., Schoff, D., Nishimura, M., Raz, S., & Ofen, N. (2018). Age-associated increase in mnemonic strategy use is linked to prefrontal cortex development. *NeuroImage*, *181*, 162–169. <https://doi.org/10.1016/j.neuroimage.2018.07.008>
- Yushkevich, P. A., Pluta, J. B., Wang, H., Xie, L., Ding, S.-L., Gertje, E. C., Mancuso, L., Klot, D., Das, S. R., & Wolk, D. A. (2015). 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>

Zielinski, B. A., Prigge, M. B. D., Nielsen, J. A., Froehlich, A. L., Abildskov, T. J., Anderson, J. S., Fletcher, P. T., Zygmont, K. M., Travers, B. G., Lange, N., Alexander, A. L., Bigler, E. D., & Lainhart, J. E. (2014). Longitudinal changes in cortical thickness in autism and typical development. *Brain*, 137(6), 1799–1812. <https://doi.org/10.1093/brain/awu083>