Neuroscience and Biobehavioral Reviews 34 (2010) 1080–1091

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review

Episodic memory across the lifespan: The contributions of associative and strategic components

Yee Lee Shing [a,b,*], Markus Werkle-Bergner [a], Yvonne Brehmer [a,c], Viktor Müller [a], Shu-Chen Li [a], Ulman Lindenberger [a]

[a] Max Planck Institute for Human Development, Center for Lifespan Psychology, Berlin, Germany
[b] Humboldt University, Department of Psychology, Berlin, Germany
[c] Aging Research Center, Karolinska Institute, Stockholm, Sweden

ARTICLE INFO

Keywords:
Episodic memory
Binding
Strategy use
Lifespan development
Aging
Child development

ABSTRACT

The structural and functional brain circuitries supporting episodic memory undergo profound reorganization in childhood and old age. We propose a two-component framework that combines and integrates evidence from child development and aging. It posits that episodic memory builds on two interacting components: (a) the strategic component, which refers to memory control operations, and (b) the associative component, which refers to mechanisms that bind different features of a memory episode into a compound representation. We hypothesize that: (a) children's difficulties in episodic memory primarily originate from low levels of strategic operations, and reflect the protracted development of the prefrontal cortex (PFC); (b) deficits in episodic memory performance among older adults originate from impairments in both strategic and associative components, reflecting senescent changes in the PFC and the medio-temporal lobes (MTL). Initial behavioral and neural evidence is consistent with both hypotheses. The two-component framework highlights the specificities of episodic memory in different age periods, helps to identify and dissociate its components, and contributes to understanding the interplay among maturation, learning, and senescence.

Contents

1. Introduction

Remembering past events is a universal human experience. A core part of our identity formation is rooted in the ability to mentally travel back in time and re-experience past events. Episodic memory (Tulving, 1972) refers to the unique feature of

* Corresponding author at: Max Planck Institute for Human Development, Lentzeallee 94, 14195 Berlin, Germany. Tel.: +49 30 82406 436; fax: +49 30 8249939.
E-mail address: yshing@mpib-berlin.mpg.de (Y.L. Shing).

0149-7634/$ – see front matter © 2009 Elsevier Ltd. All rights reserved.
doi:10.1016/j.neubiorev.2009.11.002

human memory as the conscious remembrance of events that are situated in time and place. It follows that episodic memory, as a form of explicit memory, involves the encoding, consolidation, as well as retrieval of events (e.g., Underwood, 1969). Elements belonging to the same event have to be bound and at the same time separated from other features belonging to different events (O'Reilly and Norman, 2002; Treisman, 1996; Zimmer et al., 2006). Therefore, the ability to integrate the individual features of an event into a distinctive and cohesive memory representation is critical for the functioning of episodic memory.

Throughout the lifespan, episodic memory functions undergo profound and continuous changes. In general, episodic memory performance has been shown to increase rapidly during childhood (Schneider and Pressley, 1997), and to decrease during adulthood (Kausler, 1994), with accelerated decline in very old age (Singer et al., 2003). At the same time, the structural and functional brain circuitries supporting episodic memory also undergo profound reorganization in childhood (Johnson, 2001; Nelson, 2001) and old age (Buckner, 2004; Cabeza et al., 2004). It is important to point out that researchers working either in the field of child development or aging have initiated fruitful lines of research to better understand how memory develops and changes within confined age periods (see reviews in Kausler, 1994; Schneider and Pressley, 1997). However, remarkably little effort has been invested in directly examining and integrating the mechanisms underlying memory changes across the lifespan (but see Craik and Bialystok, 2006).

In this light, the present review, along with the framework we propose herein, seeks to foster and integrate knowledge about the differences, commonalities, and dependencies among the mechanisms that regulate episodic memory development across the various life periods. Furthermore, we aim to achieve a more comprehensive understanding of the mutual dependencies between neuronal and behavioral changes across the lifespan (Baltes et al., 2006b; Craik and Bialystok, 2006; Lindenberger et al., 2006).

The present review focused on the investigation of age-related differences in episodic memory from middle childhood (beginning at around six years of age) to old age. The reason for starting in middle childhood is linked to the autonoetic (i.e., capacity for mental time travel) and self-referential nature of episodic memory (Wheeler et al., 1997). While important changes take place in children's memory before 6 years, the ability to remember events as experiences emerges only after around this age (Perner and Ruffman, 1995).

We propose a two-component framework of episodic memory development that combines and integrates neuronal and behavioral evidence. According to this framework, the ontogeny of episodic memory builds on the interaction between two components: (1) The *strategic component*, which refers to control processes that aid and regulate memory functions at both encoding and retrieval. These processes may include elaboration and organization of memory content at encoding, and specification, verification, monitoring, and evaluation of relevant information at retrieval (e.g., Simons and Spiers, 2003). (2) The *associative component*, on the other hand, refers to mechanisms of binding together different features of the memory content into coherent representations (Treisman, 1996; Zimmer et al., 2006).

In the following sections, we will unfold the theoretical assumptions and empirical implications of the framework. We begin by briefly outlining some major behavioral findings concerning episodic memory changes across child development and aging. We then introduce the two-component framework of episodic memory and the associated neural correlates in some detail. Following this, we review the available evidence on age-related differences in the neural correlates of strategic and associative components. In the final section, we propose three possible lines of future research that can be derived from the two-component framework. In doing so, we hope to demonstrate how the proposed two-component framework of episodic memory, which is grounded in general assumptions about lifespan changes in cognition (Baltes et al., 2006b; Lindenberger et al., 2006), accounts for existing findings, and, more importantly, leads to novel predictions.

2. Lifespan differences in episodic memory: assembling findings and concepts from research on child development and aging

The following review selectively focuses on commonalities and differences in findings on episodic memory across different age periods. To facilitate this comparison, we proceed along four general research themes that are common to both child developmental and aging research.

First, differential patterns of age differences in memory have been noted on the *automatic/effortful* dimension (cf. Schneider and Shiffrin, 1977). Behavioral evidence indicates that mnemonic abilities develop from childhood, through adolescence, and into young adulthood (Schneider and Pressley, 1997). However, the patterns of differences vary depending on task characteristics. A general observation is that forms of memory requiring less conscious control develop earlier than forms of memory that require considerable degrees of strategy use (e.g., Gathercole, 1998). For example, experimental variations of retrieval conditions reveal differential rates of progression, with smaller age differences being found in recognition memory than in cued or free recall (e.g., Perlmutter and Lange, 1978). Similarly, memory change during adulthood can be organized along the automatic/effortful dimension. Older adults show poorer performance than younger adults on explicit memory tasks, with the difference in performance being largest in free recall, followed by cued recall and recognition (e.g., Craik and McDowd, 1987). These findings are consistent with the effortful/automatic distinction, as recall generally requires more self-initiated processing, thereby putting higher demands on strategy use and cognitive control compared to recognition. They also fit the distinction between "environmental support" versus "self-initiated activities" proposed by Craik (1986), which accounts for the benefits that older adults derive from retrieval cues and paradigms such as recognition memory relative to free recall. The basic idea is that older adults are less capable of self-initiating appropriate retrieval processes, but can organize and execute such processes when supported by adequate input from the external context.

Second, both younger children and older adults have been shown to encounter difficulties in *source monitoring* relative to younger adults. Source monitoring refers to the ability to specify contextual information surrounding memory traces (Johnson et al., 1993). For example, children below the age of 6 years seem to have difficulties in judging whether they performed or imagined an action (Foley and Johnson, 1985; Lindsay et al., 1991). Accordingly, several researchers proposed that preschoolers (3–5-year-olds) are more susceptible to false suggestions in eyewitness testimony because they easily confuse the sources of events (Ackil and Zaragoza, 1995; Poole and Lindsay, 1995). At the other end of the lifespan, older adults also show pronounced difficulties in memory for context (Spencer and Raz, 1995). Furthermore, it has been suggested that age-related deficits in source memory, and episodic memory in general, involve failures to adequately associate target items with other items, or target items with their contexts (see review in Old and Naveh-Benjamin, 2008), an observation termed the associative deficit hypothesis (Naveh-Benjamin, 2000).

The third common theme refers to the *differential reliance on familiarity and recollection* retrieval processes across the lifespan.

According to the dual-process account (Yonelinas, 2002), remembering past events can be based on retrieval accompanied by specific contextual details (i.e., recollection) or on the feeling of knowing that an event is old or new without necessarily recollecting specific details (i.e., familiarity). Despite difficulties faced by developmental researchers to study mental experiences in children, emerging evidence suggests that the development of recollection extends into adolescence, whereas familiarity matures earlier during childhood (e.g., Brainerd and Reyna, 2004; Ghetti and Angelini, 2008). At the other end of the lifespan, older adults have been found to rely more on familiarity processes during retrieval as they face difficulties in recollecting details of memory episodes (Healy et al., 2005; Jacoby and Hay, 1998).

Finally, the fourth common research theme refers to changes in the *organization of memory representations* in childhood and old age (Craik and Bialystok, 2006). Representational systems are postulated to be hierarchically organized, ranging from specific episodic instances to general, context-free concepts (Craik and Lockhart, 1972). According to Craik and Bialystok (2006), access to the different levels develops asymmetrically over the lifespan. During development, children have good access to the lower-level episodic instances and gradually build concepts at the higher levels. Older adults, on the other hand, retain access to higher conceptual levels but progressively lose access to the lower levels, such as the details of memory episodes. In a similar vein, the fuzzy-trace theory by Brainerd and Reyna (1990) posits that memory representations can be aligned on a continuum ranging from literal, verbatim traces (corresponding to representations constrained to the surface form of memory content) to fuzzy, gist-like traces (corresponding to generalized representations including semantic meaning of memory content). In terms of developmental differences, during the preschool and early elementary-school-years an initial improvement in verbatim memory can be noted. Gist memory, on the other hand, tends to lag behind in development. Overall, children experience a shift from relying on context-specific to gist-like representations over developmental periods (Brainerd and Reyna, 2004). The higher interference susceptibility of verbatim traces may thereby contribute to lower memory performance in younger children. In connection to the developmental progression of familiarity/recollection, fuzzy-trace theory posits that true recollective experiences are by-products of the retrieval of verbatim traces (Brainerd et al., 1999). Therefore, recollection, in parallel to accessibility to verbatim trace, increases with age (Brainerd and Reyna, 2004; Paz-Alonso et al., 2008).

At the other end of the lifespan, older adults tend to retain good access to high-level conceptual representations, as demonstrated by the prolonged maintenance and potential for growth of skills that are highly dependent on biographically acquired bodies of knowledge (Lindenberger, 2001). At the same time, their access to the details of specific memory episodes is deteriorating, in line with general impairments in fluid abilities. As a consequence, older adults are more likely to rely on semantic gist than younger adults. For instance, older adults show a more pronounced increase than younger adults in the erroneous retrieval of lures after studying semantically related items in the encoding list (i.e., the Deese-Roediger-McDermott paradigm; Norman and Schacter, 1997).

Taken together, despite a lack of integration across child developmental and aging research, we note some similarities among research themes pursued in the two fields. To some extent, children and older adults may exhibit similar behavioral patterns, especially along the automatic/effortful dimension, in that both age groups have difficulties in effectively engaging in memory control processes. In addition, both younger children and older adults have difficulty in monitoring the source of a memory event. However, this ability develops relatively quickly during childhood; children beyond preschool age no longer show pronounced source monitoring difficulties in comparison to young adults. In terms of memory retrieval, both younger children and older adults tend to rely more on familiarity processes. However, they differ in the creation of memory representations, such that younger children tend to create verbatim traces that are more susceptible to interference, while older adults tend to create more gist-like traces that may be more susceptible to pronounced intrusion errors.

3. Two-component framework of episodic memory: dissociating associative and strategic aspects

In the following, we suggest a two-component framework for the study of episodic memory that attempts to bridge the gap between research on childhood and research on aging. The distinction between associative and strategic components of episodic memory is at the core of this framework. In our view, many age-related changes in memory across the lifespan can be conceptualized as reflecting changes in one or both of the components as well as in their interactions.

The two-component framework proposed here is by no means a radical departure from existing theories of episodic memory. Rather, it contains threads of established conceptions of episodic memory, especially those that distinguish between associative-binding and strategic-control components and their neural correlates (Eichenbaum, 2002; Moscovitch, 1992; O'Reilly and Norman, 2002; Prull et al., 2000; Simons and Spiers, 2003). What we see as a novel contribution of our framework is to link these conceptions to a lifespan perspective, with, as we hope, mutual benefits for the understanding of lifespan development and the functioning of episodic memory (Brehmer et al., 2007; Shing et al., 2008; Werkle-Bergner et al., 2006).

3.1. Differential behavioral expressions of associative and strategic components

The associative component of episodic memory refers to mechanisms during encoding, storage, and retrieval that bind different aspects of an event into a cohesive memory episode (Treisman, 1996; Zimmer et al., 2006). Binding mechanisms can operate either among different features of a given memory item (e.g., Chalfonte and Johnson, 1996), between different memory items (e.g., Naveh-Benjamin, 2000), or between a given memory episode and its context (e.g., Spencer and Raz, 1995). Therefore, binding mechanisms refer to a set of cognitive processes that associate features within a memory trace or several memory traces among each other (Zimmer et al., 2006). Furthermore, mechanisms of association formation, consolidation, and retrieval exist at different "levels of binding", ranging from perceptual-feature binding to the formation of higher-order memory, concepts, and ideas (Murre et al., 2006).

Alongside the associative component, the strategic component contributes to memory functions through cognitive control operations that support the encoding of discrete memory traces, and the subsequent strategic search, retrieval, and evaluation of stored representations (Rugg and Wilding, 2000; Simons and Spiers, 2003). Strategic encoding may involve organization and elaboration of the memory features, presumably by making use of one's existing semantic knowledge to incorporate specific relational aspects between them. At retrieval, dependent on the task at hand, internally generated or externally provided retrieval cues may be specified and used to strategically search among stored representations. During retrieval of a memory representation, it needs to be monitored and verified before a mnemonic decision can be made. At the same time, given the vast amount of information available in memory, momentarily competing, irrelevant memory traces can be co-activated; this calls for controlled

processes to resolve the conflict. In contrast to the associative component that is employed relatively automatically, strategic processes appear effortful and may be self-activated spontaneously, or elicited by instruction. This notion of strategic activity is reminiscent of the levels-of-processing theory (cf. Craik and Lockhart, 1972), as both accounts postulate that successful encoding and retrieval are facilitated by 'self-initiated processing.'

3.2. Distinct, but interdependent neural correlates of associative and strategic components

Early neuronally informed models of episodic memory (e.g., Moscovitch, 1992) postulated that the strategic component depends primarily on the frontal cortex, whereas the associative component relies mostly on the medial temporal region (especially the hippocampus). This distinction has gained empirical support in recent years, as numerous studies, including case studies with amnesic patients and animal models, indicate that the medial temporal lobe (MTL) and the prefrontal cortex (PFC) contribute to memory in critical and separable ways (Simons and Spiers, 2003; Werkle-Bergner et al., 2006).

Reviews of animal models and human brain-lesion studies suggest that the hippocampal formation and associated structures contribute to the formation and maintenance of memories (Squire, 2004), and particularly to the establishment of associations between episodic features in memory, that is, binding (Cohen and Eichenbaum, 1993; Davachi and Wagner, 2002). Recent evidence from the neuroimaging literature has provided further specification of how distinct MTL regions contribute differentially to episodic memory formation. In particular, subsequent memory studies have demonstrated that the magnitude of activation in the hippocampus during encoding is critically associated with later objective and subjective indices of relational memory (see review in Davachi, 2006). While the hippocampus is clearly involved in the acquisition of new relational information, its particular role in the retrieval of past memories, especially remote memory, is less clear. For recent memory, the hippocampus is thought to be involved in the reinstatement of memory representations during retrieval. In particular, neuroimaging and animal studies support the hypothesis that familiarity and recollection rely on the hippocampus and parahippocampal region (e.g., perirhinal cortex), respectively (see Aggleton and Brown, 2006). For remote memory, the involvement of hippocampus is under debate. Current accounts of memory consolidation, such as the multiple-trace theory, suggest that the hippocampus is needed to re-experience detailed episodic memories no matter how old they are (e.g., Moscovitch et al., 2006). The standard consolidation model, on the other hand, suggests a prolonged process of consolidation in which the contribution of the hippocampus diminishes as consolidation proceeds, until the neocortex and other structures are ready to sustain the permanent memory trace (see review in Dudai, 2004). Supporting evidence has been reported for both models. Current conceptions speculate that the hippocampus may specifically be involved in retrieval of certain types of remote memories (e.g., vivid autobiographical memory), whereas new memory traces that get quickly independent of the hippocampus may be more easily integrated in an existing knowledge network or schema (see Fernandez and Tendolkar, 2009).

Compared to the early interest in the MTL, the importance of the frontal lobe regions for memory processes has been appreciated only recently. In an important early functional neuroimaging study, Fletcher et al. (1998) tested the hypothesis that activations within the left PFC reflect organizational processes necessary for optimal memory encoding. Participants were asked to study word lists that consisted of categorically related words. According to the encoding instruction, the word lists varied in the degree of organization the participants were required to perform in order to facilitate encoding. Results showed that the condition in which words were already organized produced the least degree of left PFC activity, whereas the condition that required participants to self-generate an organizational structure was associated with the highest activity in this region. Together with converging results from other studies, this finding revealed that the episodic memory system includes neocortical regions. In general, the PFC, particularly the lateral regions, serves strategic/cognitive control functions that are necessary for the creation of durable memory representations through organization and elaboration of the relevant stimuli (e.g., Cabeza and Nyberg, 2000; Kramer et al., 2005; Rajah and D'Esposito, 2005). In terms of retrieval, it is widely accepted that the PFC plays a key role in controlling episodic retrieval in general and resolving interference in particular (see review in Fletcher and Henson, 2001). The PFC has been shown to be activated by various component processes of retrieval control, including retrieval orientation, retrieval effort, and post-retrieval monitoring (e.g., Badre and Wagner, 2007; Rugg and Wilding, 2000). Dorsolateral PFC has been implicated in post-retrieval monitoring such as verification, monitoring, and evaluations of representations (e.g., Achim and Lepage, 2005). Ventrolateral PFC, on the other hand, seems to be involved in the strategic search of stored representations, maintenance of retrieved information, and selection of a specific memory among competing ones (Badre and Wagner, 2007).

Taken together, the ability to encode and retrieve episodic associations relies on a distributed functional network between MTL/hippocampal memory mechanisms (primarily for relational/binding processes) and PFC-mediated cognitive control processes (primarily for strategic encoding and controlled retrieval processes). However, the story does not end here, as a clear separation of associative and strategic components would not do justice to their interdependence, neither at the cognitive nor at the neural level of analysis. The neural networks for episodic memory are distributed across multiple brain regions, both at encoding (Otten and Rugg, 2001) and at retrieval (Buckner and Wheeler, 2001; Paller and Wagner, 2002). Analyses of neuroanatomical connections (e.g., Warrington and Weiskrantz, 1982) and functional connectivity point towards intricate interactions between PFC and MTL regions during encoding and retrieval (e.g., McIntosh et al., 1997). Furthermore, the PFC utilizes a wide range of learned associations to serve as control signals for the guidance of the dynamic flow of neural activity in response to different task demands (Miller and Cohen, 2001). In other words, on the one hand, the very processes supporting the strategic aspect of episodic memory may involve learned associations; and on the other hand, learned associations are likely to be more durable and distinctive when optimal strategic control is called into service.

In sum, the associative and strategic components are interdependent at neuronal and functional levels, posing a challenge for empirical dissociation. In this situation, a lifespan approach to the study of memory may help to disentangle the relative contributions of associative and strategic components as the two components change at different rates across life periods.

4. Towards a two-component framework for studying the lifespan development of episodic memory

We propose that the two components contribute to the lifespan trajectory of episodic memory functioning in dissociable ways. At the neural level, brain regions supporting memory functioning undergo substantial alterations across the lifespan (e.g., Buckner, 2004; Nelson, 2001). In the following, we will first assemble and integrate existing evidence on age differences in the neural correlates supporting the associative and the strategic components

of episodic memory, introduce two propositions that capture the hypothesized dissociation in the age trajectories of the two components, and finally present a recent study that aimed at a direct empirical test of this dissociation.

4.1. Differential developmental trajectories of PFC and MTL regions

In terms of brain development, anatomical and post-mortem studies show that the PFC in general, and its dorsolateral regions in particular, undergo profound maturational changes well into adolescence, while MTL regions mature at faster rates (e.g., Gogtay et al., 2006; Sowell et al., 2003). At the functional level, event-related potential (ERP) and functional magnetic resonance imaging (fMRI) studies support an association between the slower development of memory for context and details and the prolonged maturation of PFC networks (e.g., Cycowicz et al., 2001, 2003; Ofen et al., 2007). Thus, existing evidence suggests that the development of episodic memory functions is paralleled by an age-related increase in PFC integrity, whereas memory-related MTL functioning remains relatively constant across middle childhood.

With respect to brain aging, prefrontal regions show linear declines in cortical volume beginning in the mid-20s (Raz et al., 2005; Sowell et al., 2003). The frontal-lobe hypothesis of cognitive aging suggests that many age-related changes in cognition, including episodic memory, reflect, to a large extent, senescent structural and neurochemical changes of the frontal lobes (Buckner, 2004; West, 1996). In older adults, episodic memory function has been associated with increased bilaterality in PFC activation, sometimes resulting from increases in activation of the nondominant hemisphere, or reduced activation in the dominant hemisphere that is usually activated by the younger adults (see reviews in Cabeza, 2002; Park and Gutchess, 2005). At the same time, accelerated patterns of decline are also observed in the MTL regions, with a marked decline observed in the hippocampus and the entorhinal cortex (e.g., Raz et al., 2004, 2005). Functionally, age-associated impairments in these regions (the hippocampus particularly) have been linked to older adults' difficulties in forming new associations in working memory (Mitchell et al., 2000) and episodic memory (Daselaar et al., 2003; Grady et al., 2003), and in separating new associations from existing memory traces stored in long-term memory (e.g., Daselaar et al., 2006; Wilson et al., 2006).

Interestingly, a recent multivariate study (Raz et al., 2005) examining individual differences in rates of volume shrinkage has reported a strong correlation between volume losses in the hippocampus and losses in the white matter of the PFC, indicating that adults whose hippocampus shrinks more than average also tend to lose more white matter in the PFC (reliability-adjusted correlation, $r = .70$, $p < .0001$). This finding suggests that senescent changes in the PFC and the MTL are interdependent and may affect the functioning of the strategic and associative components in a coordinated rather than orthogonal manner.

4.2. Lifespan changes in episodic memory: two propositions based on the two-component framework

Based on the preceding behavioral and neural evidence on developmental changes in episodic memory functioning from childhood to old age, we now are in a position to formulate two propositions regarding its ontogeny. In terms of the two-component framework, the first proposition refers to the associative, and the second to the strategic component of episodic memory.

Proposition 1. *The associative component of episodic memory matures in middle childhood, and undergoes senescent decline in late adulthood and old age.*

Proposition 2. *The strategic component of episodic memory matures later, in approaching adolescence and young adulthood, and undergoes senescent decline in late adulthood and old age.*

In combination, the two propositions result in the prediction of a lifespan dissociation between associative and strategic components: Whereas the development of the strategic component trails behind the associative component in childhood, resulting in a clear lead-lag relation between the two components, both associative and strategic components decline in old age.

The results of a study on working memory by Cowan et al. (2006) support the lifespan dissociation within the associative component detailed by our first proposition. In a change-detection paradigm (Luck and Vogel, 1997), participants were asked to detect changes in either single features (different color of a square stimulus), or in feature conjunctions (different color and location), the latter requiring more binding-related processing. Older adults often failed to notice the feature-conjunction change, especially when single-feature and feature-conjunction trials were intermixed. The two child groups (aged 8–10 and 11–12 years) showed lower performance levels than younger adults on both kinds of trials. The age difference on the feature-conjunction trials was, however, not as disproportionately large as in the group of older adults. This finding lends additional support to the notion that the associative component of memory performance is relatively mature by middle childhood (see also Sluzenski et al., 2006), but undergoes substantial decline with aging.

Turning to the strategic component, direct lifespan comparisons are scarce (but see Shing et al., 2008, reported in detail below). In children, strategic encoding behavior in memory has been studied in sort-recall paradigms with lists of semantically-related pictures. Numerous studies have shown that memory strategies assessed in this manner develop rapidly throughout the elementary-school years (for a review, see Schneider and Pressley, 1997). High-level organizational strategies are typically not observed in children younger than six years of age, even after explicit instruction. Later kindergarten and early-grade school-children still do not spontaneously display strategic organizational behavior, but the reluctance to engage in strategic behavior can be overcome through instruction. During later elementary school years, children begin to display strategic behavior without being prompted to do so, and may go through a transitional phase in which they do not benefit immediately from its use. In summary, this line of research suggests that the full range of memory-related strategic behaviors is not mastered before the end of the elementary school years (Schneider and Pressley, 1997). In parallel to changes in strategic encoding behavior, various cognitive control processes undergo extended improvements with development and may contribute to progress in memory retrieval functions. These processes include the ability to inhibit attention to irrelevant stimuli, to remember the task stimulus set, and make selections in line with current goals, and to maintain and manipulate information in working memory (e.g., Crone et al., 2006; Davidson et al., 2006).

At the other end of the lifespan, older adults appear to engage in memory-relevant strategic processes less efficiently than younger adults (Dunlosky and Hertzog, 1998; Dunlosky et al., 2005; Kausler, 1994). Recent evidence indicates that strategic impairments are not confined to encoding. For instance, Naveh-Benjamin et al. (2007) showed that instructions to use appropriate strategies during encoding and retrieval led to a reduction of associative deficits in older adults relative to younger adults, more so than instructions restricted to encoding alone. In terms of self-reported strategy use, Dunlosky et al. (2005) found that aging-related deficits are substantial for the recall of mediators (e.g., images or sentences generated at encoding), small for decoding mediators

when recalled, and absent for producing mediators in the first place. Hence, older adults seem to have difficulties in engaging in effective strategic processes both at encoding and retrieval. Taken together, our postulation of lifespan changes in the strategic component of episodic memory is in agreement with the emphasis that Craik and Bialystok (2006) placed on cognitive control as a mechanism of change throughout the lifespan.

4.3. Initial empirical evidence in support of the two-component framework

The available evidence presented so far appears to be consistent with the lifespan dissociation of associative and strategic components predicted by the two-component framework. However, the two propositions were not yet tested directly in a lifespan sample within the same experiment. To overcome this, Shing et al. (2008) recently conducted a lifespan study that systematically varied the demands on associative and strategic components with an associative recognition memory task in a within-person repeated-measures design. Participants were presented with a list of word pairs, and were subsequently tested for their memory of either the single words (item recognition test), or the associations between the word pairs. The demands on the associative component were manipulated by using word pairs with (a) low and (b) high associative strength (i.e., German-German "GG" vs. German-Malay "GM" word pairs), and the demands on strategic component were manipulated by study instructions that emphasized (a) incidental item encoding, (b) intentional pair encoding, and (c) elaborative strategic encoding. A practice-based follow-up study for the GM condition was also conducted to induce further improvements in participants' performance in this condition.

The main findings were consistent with the two-component framework (see Fig. 1). First, in comparison to children, older adults initially performed slightly better in item- and pair-instruction sessions. This may have reflected older adults' ability to make use of their larger repertoire of knowledge and semantic memory acquired from life experience. However, children showed a higher performance gain from strategy instructions for the GG condition and also from practice for the GM condition. Thus, children improved more than older adults in forming associations between memory items when provided with a combination of instruction in a strategy tool and task-relevant practice, demonstrating their latent potential for associative binding. Older adults, in contrast, did not benefit as much as the children from instruction and practice in the mnemonic strategy. Their performance gain was especially small in the high associative-demand GM condition, supporting the hypothesis that older adults face an additional deficit in associative binding (see also Brehmer et al., 2007).

Teenagers and younger adults, who are supposedly close to their lifespan peaks in both the associative and the strategic component, showed earlier improvements in performance than children and older adults did. For instance, in the GG condition, they already started improving when asked to study the word pairs intentionally but before being told what strategy to use. This led to a magnification of age differences in performance after pair-encoding instructions between the "middle" lifespan age groups (i.e., teenagers and younger adults) and the extreme lifespan age groups (i.e., children and older adults). This finding parallels the notion that aging, in comparison to young adulthood, is associated with a decline in the ability to organize and execute self-initiated processing, probably due to a decreased efficiency of the strategic component. The same notion can be extended to child development, complementing the strategy deficiency literature. For the GM condition, there were differential patterns of change between the two age groups, such that younger adults showed improvements in performance mainly after strategy instruction, whereas teenagers showed improvement only after extensive practice. This might have been due to the high difficulty of the GM condition in the sense that even the high performing younger adults were unable to come up with an efficient strategy on their own. When provided with a strategy, younger adults apparently were immediately able to apply it as an aid for memory encoding. Teenagers, on the other hand, needed more extensive practice on putting the strategy to use. The close similarity in performance change between teenagers and younger adults in the GG condition partly supported the hypothesis that the strategic component is relatively mature in adolescence, corresponding to the burst in prefrontal cortex development after puberty (Sowell et al., 2003). However, in the more challenging GM condition, the difference between teenagers and younger adults revealed that the strategic

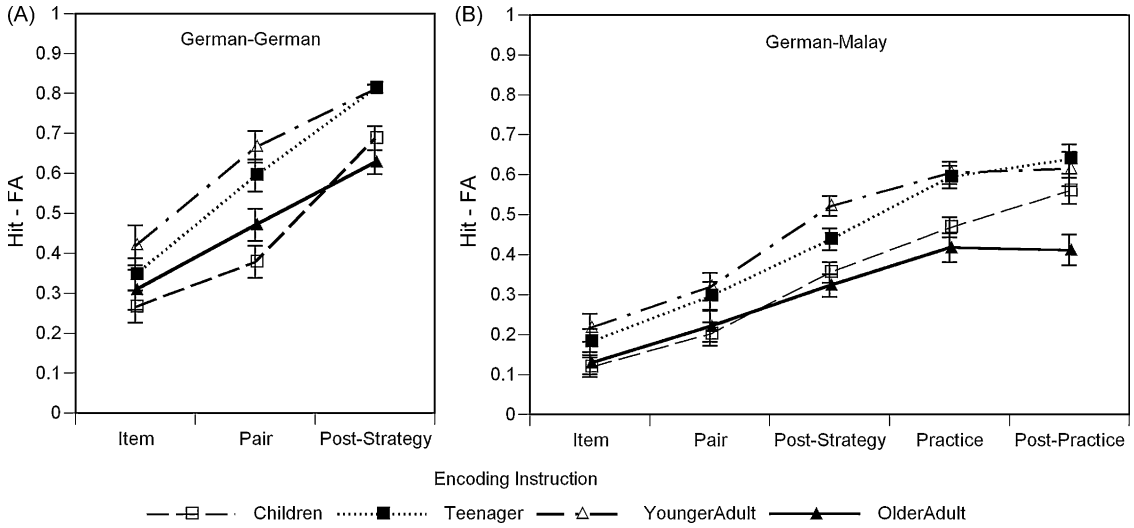

Fig. 1. Empirical support for the two-component framework of episodic memory development across the lifespan. In two multi-session experiments, memory for word pairs was probed with a recognition paradigm. Panel A displays data for the associatively less demanding German-German condition, Panel B for the associatively more demanding German-Malay condition. The strategic component was manipulated by instruction and subsequent practice. Memory performance refers to rate of correctly recognized pairs (hits) minus rate of erroneously recognized lure pairs (false alarms). Here, the lure pairs consist of words that had been separately presented during encoding. Adapted from Shing et al. (2008).

component continues to undergo protracted development until adulthood.

5. Directions for future research

The review above demonstrated that behavioral, neuroanatomical, and neurofunctional evidence supports the premise that episodic memory functioning requires the interactive operation of associative and strategic components, and that the two components of episodic memory follow different lifespan trajectories, leading to predictable patterns of age-related differences in episodic memory performance. However, the exact nature in which the components of the MTL-PFC network develop throughout the lifespan and how they interact to support memory functioning remains unclear.

A major task for future research is to comprehensively chart the ontogeny of the two components, testing and refining the guiding propositions of the framework. For the age periods of childhood and adolescence, the hypothesized lead–lag relation between the two components, with the strategic component trailing behind the associative component, has received some consistent support at both the behavioral and the neural level of analysis (e.g., Brehmer et al., 2007; Shing et al., 2008; Ofen et al., 2007). Turning to the other end of the human lifespan, it is not known at this point whether (a) declines in the strategic component precede declines in the associative component, or vice versa; and whether (b) declines in the associative component are as normative as declines in the strategic component, or foreshadow, to some degree at least, the later onset of dementia (cf. Bäckman et al., 2005; Buckner, 2004).

Furthermore, just as cognitive control does not refer to a unitary process, memory-related strategic processes (at encoding and retrieval) likely follow somewhat different developmental trajectories. Depending on the specific control demands of the task at hand, performance may become fully functional at earlier (i.e., late adolescence) or later (i.e., young adulthood) time points in the course of ontogeny. Compared to older adults, the manifestation of deficiency in the strategic component may also take different forms in childhood. In the Shing et al. (2008) study described above, a pronounced lifespan dissociation in the error patterns was revealed when the hit and false alarm rates were inspected separately. Compared to children and younger adults, older adults consistently showed greater difficulties in rejecting rearranged pairs that were made of studied words taken from different word pairs at encoding. Furthermore, a comparison of effect sizes suggests that the age differences in false-alarm rates for rearranged pairs were especially pronounced in the GM condition.

Children, on the other hand, mainly showed lower hit rates than younger and older adults in both the GG and GM conditions before strategy instruction. However, in contrast to older adults' tendency for false alarms, this age difference was eliminated after strategy instruction and practice, again reflecting children's latent potential in associative binding after initial strategic deficiencies have been overcome. Taken together, the lifespan comparisons suggest that the nature of memory difficulties children have is different from those of older adults. As rejecting the rearranged pairs puts particularly high demands on controlled processing during retrieval to overcome response tendencies triggered by familiarity signals, older adults' high false-alarm rate may have resulted from a reduction in distinctiveness of memory traces stemmed from deficiency in the associative component in combination with less efficient strategic control at retrieval. This is why the inclusion of older adults helps to elucidate the particular characteristics of episodic memory in children, and vice versa. In addition, the goal for future research is to move the level of explanation from components to mechanisms, including a better understanding of specific associative and strategic processes and their lifespan developmental trajectories.

Finally, in this review, we focused on young and old adulthood and said little about middle adulthood, primarily because most large-scale longitudinal investigations suggest that episodic memory decrements before age 60 are relatively small (e.g., Rönnlund et al., 2005; Schaie, 1996). However, this does not rule out that relevant neural changes take place before behavioral changes become evident. For instance, even though senescence may take a greater and earlier toll on the strategic than on the associative component, this physiological loss may not manifest itself in behavior for quite some time because prefrontal regions may offer more room for compensatory reorganization than MTL structures. The inclusion of middle age in neuroimaging studies can potentially provide an estimate of whether the functional brain changes that are evident in older adults can already be tracked in younger ages. It is worth noting that physiological changes in relevant brain structures, as observed by Raz et al. (2005), often seem to be gradual and discernible in early and middle adulthood, whereas behavioral changes are more curvilinear, or threshold-like in shape, and tend to commence at later ages (e.g., de Frias et al., 2007; Rönnlund et al., 2005). Longitudinal investigations that combine neural and behavioral levels of analysis will help to determine whether there is a shift in brain activity between childhood and old age despite behavioral stability, whether differences in brain activity accelerate with increasing age and coincide with memory decline in old age, or both.

To add further complexity to the issue, the interactions within the MTL–PFC network differ between memory formation and memory retrieval, and these memory modes are rarely fully separated, as encoding also elicits retrieval, and vice versa. With new lines of evidence emerging from developmental, aging, and brain imaging studies, future research that combines insights from these domains of inquiry may contribute to attaining a more comprehensive picture of episodic memory functioning. In the last section, we identify three main lines of inquiry that may move research closer towards this goal.

5.1. Encoding differences in children and older adults

Based on the two-component framework, we suggest that extensive periods of memory development during middle childhood can be characterized as primarily reflecting changes in frontal areas and their interactions with MTL areas. Along these lines, initial attempts have been made to delineate how the late maturation of the PFC (but not MTL) alters the brain activation patterns representing effective encoding (Ofen et al., 2007). An important issue to pursue is to derive a better understanding of the exact functions of increasing PFC activation, and to examine the extent to which PFC engagement can be facilitated by explicit instructions and training. It has been shown that children possess greater potential than older adults for improving episodic memory performance through strategy training and subsequent practice (Brehmer et al., 2007; Shing et al., 2008). Based on the two-component model, and in line with the findings reported by Ofen et al. (2007), we predict that improvements in episodic memory performance induced by strategy training are accompanied by enhanced activation in PFC regions in general, and stronger connectivity between PFC and MTL in particular. The effects of training may also be more durable in children than in older adults because task-induced PFC activation and PFC maturation support each other. Behavioral evidence on lifespan age differences in maintenance of skilled memory performance over a period of 11 months support this prediction (Brehmer et al., 2008), but relevant imaging evidence has not yet been obtained.

The aging field has gathered relatively extensive information about how memory encoding changes in old age. With respect to medio-temporal structures, Wilson et al. (2006) postulated that age-related memory impairments may emerge from the failure to encode new contexts in ways that are sufficiently distinct from already-stored memories (see also Li et al., 2005). Specifically, in the aged hippocampus, the dentate gyrus may be deficient in resolving and separating new input patterns, coupled with the auto-associative fibers of layer CA3 being overly entrenched in pattern completion. Together, these specific alterations may result in weakened processing of new information and over-strengthening of previously stored patterns. Therefore, encoding processes in older adults may rely too heavily on the integration of new information with the abundant number of stored patterns. These conjectures remain to be validated in human research.

Interestingly, the age-related reduction in activity observed in posterior areas of the brain is often coupled with increased frontal activity (Davis et al., 2008). For example, using event-related fMRI to investigate age differences in subsequent memory for pictures, Gutchess et al. (2005) observed reduced activity in bilateral MTL regions but greater subsequent memory effects in bilateral PFC regions for older than for younger adults (see also Morcom et al., 2003). Grady et al. (2003) examined the functional connectivity of the hippocampus during encoding of words and objects in younger and older adults. In younger adults, connectivity among hippocampus, ventral PFC, and visual cortices during encoding supported subsequent recognition, while in older adults, connectivity among hippocampus, dorsolateral PFC, and parietal regions supported memory. Increased activation may indicate a failure to appropriately engage specialized regions, reflecting dedifferentiation in processing (Lindenberger and Baltes, 1997; Logan et al., 2002). Alternatively, as suggested by Cabeza (2002), additional PFC activation in older adults, assumed to indicate additional strategic effort, may reflect attempts to compensate for deficient MTL activation. In other words, older adults show a shift in cognitive resources, from more perceptually based processes to those involved in executive and organizational functions (see also Dennis et al., 2008). Taken together, additional recruitment of PFC in older adults may be a compensatory reaction to normal aging that, if successful, may attenuate the adverse consequences of normal aging, or a primary sign of senescence, reflecting a decreasing ability to activate the brain in a selective and task-appropriate manner.

Note that these options are not mutually exclusive but may co-exist and influence each other, creating a need for within-subject experimental and longitudinal designs to disentangle their causal contributions to the dedifferentiation phenomenon. Interestingly, a compensation account suggests that older adults rely more on frontal regions for cognitive processing, but aging itself leads to a reduction in frontal resources. Emerging evidence from the working memory literature may help to resolve this paradox. Several studies (Mattay et al., 2006; Reuter-Lorenz and Cappell, 2008) suggest that older adults may recruit more neural resources to achieve computational output equivalent to that of older adults, and this compensatory activation may be effective at lower levels of task demand. However, as demand increases, a resource ceiling is reached in older adults, leading to frontal underactivation and impaired performance. Therefore, an important next step is to delineate the relative merits of the two accounts by examining functional activation and connectivity within individuals, taking a closer look at the contribution of inter- and intra-individual differences in performance in relation to varying task difficulty (e.g., Mattay et al., 2006), task instruction, and training (e.g., Erickson et al., 2007).

Interestingly, the findings of dedifferentiation in aging stand in contrast to child neuroimaging studies that suggest child development is accompanied by changes towards more differentiated patterns of brain activation, including enhanced activation in critical regions, attenuation in others, as well as shifts in lateralization (e.g., Durston et al., 2006; Müller et al., 2009). These changes can be described as resulting in more focal and less diffuse brain activation as children grow into adulthood, such that the activation patterns seen during one type of cognitive task show less overlap with the pattern of neural activation when performing a different task. These findings fit quite well with an interactive specialization framework (Johnson, 2001; Schaie, 1965; Werner, 1957). In this sense, during development, processing pathways go through an experience-dependent process of fine-tuning such that specific regions become increasingly specialized. With development, pathways that function partially at early stages shape subsequent changes, leading to refinements in interactions within functional neural circuitries.

Taken together, an interesting line of future research is to compare and contrast the structure and function of neural reorganization across the lifespan. In this light, mixed-block, event-related functional magnetic resonance imaging (fMRI) designs (Donaldson et al., 2001) may be used to isolate block-related, sustained neural activities from trial-related, transient ones across age groups as these processes may follow different patterns for different age groups. For children, starting from middle childhood, development in episodic memory may be characterized by (1) increases in transient recruitment of task-relevant PFC neural resources (e.g., ventrolateral PFC for semantic elaboration) that signify increases in engagement of effective encoding at the trial level; (2) reductions in transient nonselective recruitment of frontal neural resources that signify specialization of neural processes and/or lower cognitive demand; and (3) increases in sustained PFC neural activity that signify an increased ability to sustain attention over time. On the other hand, the aging memory system may undergo changes characterized by (1) a decreased transient engagement of the hippocampus; (2) an increased transient recruitment of frontal neural resources that are tangential to task requirements but support semantic processes; and (3) a decline in sustained activity in PFC signifying age-related deficits in sustained attention (Dennis et al., 2007).

5.2. Retrieval differences in children and older adults

Processes engaged during remembering can be conceptualized in two classes. The first class is associated with top-down strategic processes engaged during the search for the appropriate information, whereas the second is associated with the representation of the reconstructed memory content. Frontal regions may interact with posterior regions, such as the parietal cortex and MTL, thereby providing top-down control for the reconstruction of retrieval content (e.g., Rugg and Wilding, 2000; Wheeler and Buckner, 2003). As discussed above, these interactions map nicely onto the strategic–associative distinction.

The efficiency and nature of retrieval operations change markedly with age. During childhood, the development of efficient retrieval processes continuously increases from 4 to 12 years of age (Schneider and Pressley, 1997). The spontaneous use of simple retrieval strategies (e.g., use of external associative cues) can already be observed in the kindergarten years. The use of more complex retrieval strategies, including reorganization of stored information and monitoring of retrieval, emerge only in middle childhood to adolescent years (Schneider and Pressley, 1997). This evidence, coupled with studies indicating protracted development of recollection in children, suggests that the neural mechanisms underlying developmental progression of retrieval may more strongly relate to the control operations of PFC and/or a more efficient connection between PFC and MTL regions. These conjectures remain to by tested empirically.

At the other end of the lifespan, older adults have been shown to experience greater difficulties in monitoring the retrieval of newly learned information than younger adults (e.g., Souchay and Isingrini, 2004). In the study by Shing et al. (2008), older adults persisted in committing higher rates of false-alarm errors than any of the other three age groups, including children. Most interestingly, older adults also associated these false alarms with the subjective feeling of high confidence, and their disproportionate tendency to commit these errors with high confidence was resilient against instruction and extensive practice of encoding strategies (Shing et al., 2009). Dodson et al. (2007) suggest that older adults' high-confidence errors may arise from their susceptibility to erroneously associate features from separate events, so that associations based on miscombined features become subjectively indistinguishable from associations based on correctly combined features. It remains to be elucidated to what extent the erroneous association is formed at encoding and/or retrieval.

The above evidence seems to suggest that the manifestation of age differences in retrieval may be more variable among older adults than among children. Given that the two-component framework posits declines in both the strategic and associative components with age, it is conceivable that the sources of variation in aging are more numerous and diverse. Lesion and functional neuroimaging studies have associated recollection with the hippocampus (e.g., Yonelinas, 2002), which shows substantial and accelerated atrophy in adulthood and old age. Linking behavioral and neural evidence, functional neuroimaging studies have associated older adults' recollection deficits with reduction in hippocampal and ventral parietal cortex activity (see Cabeza et al., 2008). At the same time, Daselaar et al. (2006) observed an enhanced rhinal activation in older adults that was coupled with an increase in connectivity within a rhinal-frontal network, suggesting a top-down bias. In this context, it is worth noting that volume losses in entorhinal cortex differ reliably across individuals (Raz et al., 2005) and predict individual differences in fluid intelligence (Raz et al., 2008). Taken together, hippocampal deficit in aging, coupled with a frontally modulated greater reliance on familiarity signals from rhinal cortex, may account for aging-related differences in retrieval processes. In sum, it appears that the neural organization of memory retrieval differs widely between children and older adults. The framework of strategic and associative components may serve as a theoretical orientation that guides future research to disentangle the differences in retrieval mechanisms across the lifespan.

5.3. The missing link between encoding and retrieval: consolidation across the lifespan

At this point, it is imperative to point out that although in the current framework we focus on encoding and retrieval processes for newly acquired information, lifespan differences in remote memory and the closely linked issue of consolidation also deserve consideration, as the formation of new memory episodes inevitably implies interactions between incoming information and the stored past experiences of an individual.

In this light, the mechanisms that drive memory formation and consolidation may differ profoundly between children and older adults. Relative to children, older adults have acquired extensive semantic and rich autobiographical knowledge in the course of their lives. Therefore, it is conceivable that older adults are more likely to process new information in connection to existing representations. On the other hand, children's encoding of new information may rely heavily on the creation of representations that carry a strong novelty value and may be less readily to be blended with retrieved past experiences.

According to the standard view, system level consolidation of novel information is heavily dependent on hippocampal mechanisms. By contrast, access to remotely acquired (and presumably consolidated) memory traces engages neocortical regions, including ventro-medial prefrontal regions (e.g., Dudai, 2004; Takashima et al., 2006). Based on the two-component framework, it is tempting to speculate that age-related changes in MTL-PFC connectivity across life period may affect memory consolidation. Accordingly, a testable hypothesis would be that the shift in fMRI activation patterns from decreasing hippocampal activation to enhanced activation in ventro-medial PFC during consolidation (e.g., Takashima et al., 2006) may occur earlier with increasing age. Particularly in older adults, recently acquired memory traces may become independent of the hippocampus too quickly, as they are more easily integrated in an existing knowledge network or schema (see Fernandez and Tendolkar, 2009). A suggestion that seems reconcilable with the observed pattern of enhanced prefrontal and reduced hippocampal activations in older adults at retrieval (e.g., Daselaar et al., 2006). However, the fast integration and consolidation may hinder the detailed relational processing of memory contents, which may be a reason for reduced vividness of memory in older adults.

Studies of autobiographical memory, as a form of remote memory, can also be informative of memory consolidation. Remembering autobiographical memories is a complex reconstructive process consisting of episodic and semantic contents (Rubin, 2006), and this interplay may differ for both ends of the lifespan. The role of the hippocampus in retrieval of autobiographical memory is an issue that remains to be resolved. The involvement of the strategic component, on the other hand, is clearly evident in searching, monitoring, and self-referential processes of autobiographical memory. For older adults, the recall of remote autobiographical memory likely involves more semantic or script-like components than episodic details (see Cabeza and St. Jacques, 2007). It remains an open question to what degree this shift reflects a direct consequence of aging-related deterioration in hippocampus, an effect of remoteness of old memory, or a combination of both factors. For children, on the other hand, the strengthening of PFC-MTL connectivity may allow for increasingly effective and efficient consolidation across development. Therefore, not only more memories are stored, the traces may include richer features that are personally relevant. By building upon these higher quality episodic instances, autobiographical memory may develop through the gradual formation of schema and awareness about one's self (Bauer, 2006).

Taken together, the integration of system level consolidation and the expansion into the domain of autobiographical memory are necessary and potentially fruitful avenues towards the refinement of the two-component framework.

6. Closing remarks

In this article, we have proposed a two-component framework of episodic memory that is guided by the theoretical propositions of lifespan developmental theory (cf. Baltes et al., 2006a; Lindenberger et al., 2006). According to the framework, children's difficulties in episodic memory primarily originate from low levels of strategic involvement, and reflect the protracted development of the PFC. Deficits in episodic memory performance among older adults, on the other hand, originate from impairments in both strategic and associative components, reflecting senescent changes in the PFC and the MTL. Initial behavioral and neural evidence, as reviewed above, support the conjectures of this framework.

The complex nature of cognitive development and aging projects a need for theoretical construction and methodological

innovation crossing neural and behavioral levels of analysis. Lifespan theory posits that the study of ontogeny requires the consideration of commonalities, individual differences, and intraindividual plasticity in development. As an outlook, it would be highly desirable to implement neuroimaging designs in the context of longitudinal studies that enable comparisons of brain activity in different age groups during learning and remembering, so that both individual differences and the developmental functions of the neural reorganization of behavior can be discerned. We hope that the distinction between associative and strategic components will provide a useful conceptual framework in this endeavor.

Acknowledgements

This research was carried out in the context of the Research Group, "Binding: Functional Architecture, Neuronal Correlates, and Ontogeny," funded by the German Research Foundation (DFG FOR 448). The authors thank Lars Bäckman, Chris Hertzog, Moshe-Naveh-Benjamin, and Hubert Zimmer for productive discussions and valuable comments.

References

Achim, A.M., Lepage, M., 2005. Dorsolateral prefrontal cortex involvement in memory post-retrieval monitoring revealed in both item and associative recognition tests. Neuroimage 24, 1113–1121.
Ackil, J.K., Zaragoza, M.S., 1995. Developmental differences in eyewitness suggestibility and memory for souce. J. Exp. Child Psychol. 60, 57–83.
Aggleton, J.P., Brown, M.W., 2006. Interleaving brain systems for episodic and recognition memory. Trends Cogn. Sci. 10, 455–463.
Badre, D., Wagner, A.D., 2007. Left prefrontal cortex and the cognitive control of memory. Neuropsychologia 45, 2883–2901.
Baltes, P.B., Lindenberger, U., Staudinger, U.M., 2006a. Lifespan theory in developmental psychology. In: Damon, W., Lerner, R.M. (Eds.), Handbook of Child Psychology: Vol. 1. Theoretical Models of Human Development. Sixth ed. Wiley, New York, pp. 569–664.
Baltes, P.B., Reuter-Lorenz, P.A., Rösler, F. (Eds.), 2006b. Lifespan Development and the Brain: The Perspective of Biocultural Co-Constructivism. Cambridge University Press, Cambridge, UK.
Bäckman, L., Jones, S., Berger, A.-K., Jonsson Laukka, E., Small, B.J., 2005. Cognitive impairment in preclinical Alzheimer's disease: a meta-analysis. Neuropsychology 19, 520–531.
Bauer, P.J., 2006. Constructing a past in infancy: a neuro-developmental account. Trends Cogn. Sci. 10, 175–181.
Brainerd, C.J., Reyna, V.F., 1990. Gist in the grist: fuzzy-trace theory and the new intuitionism. Dev. Rev. 12, 164–186.
Brainerd, C.J., Reyna, V.F., 2004. Fuzzy-trace theory and memory development. Dev. Rev. 24, 396–439.
Brainerd, C.J., Reyna, V.F., Mojardin, A.H., 1999. Conjoint recognition. Psychol. Rev. 106, 160–179.
Brehmer, Y., Li, S.-C., Müller, V., von Oertzen, T., Lindenberger, U., 2007. Memory plasticity across the lifespan: uncovering children's latent potential. Dev. Psychol. 43, 465–478.
Brehmer, Y., Li, S.-C., Straube, B., Stoll, G., von Oertzen, T., Müller, V., Lindenberger, U., 2008. Comparing memory skill maintenance across the lifespan: preservation in adults, increase in children. Psychol. Aging 23, 227–238.
Buckner, R.L., 2004. Memory and executive function in aging and AD: multiple factors that cause decline and reserve factors that compensate. Neuron 44, 195–208.
Buckner, R.L., Wheeler, M.E., 2001. The cognitive neuroscience of remembering. Nat. Rev. Neurosci. 2, 624–634.
Cabeza, R., 2002. Hemispheric asymmetry reduction in older adults: the HAROLD model. Psychol. Aging 17, 85–100.
Cabeza, R., Ciaramelli, E., Olson, I.R., Moscovitch, M., 2008. The parietal cortex and episodic memory: an attentional account. Nat. Rev. Neurosci. 9, 613–625.
Cabeza, R., Nyberg, L., 2000. Imaging cognition II: an empirical review of 275 PET and fMRI studies. J. Cognitive Neurosci. 12, 1–47.
Cabeza, R., Nyberg, L., Park, D.C. (Eds.), 2004. Cognitive Neuroscience of Aging: Linking Cognitive and Cerebral Aging. Oxford University Press, New York.
Cabeza, R., St. Jacques, P., 2007. Functional neuroimaging of autobiographical memory. Trends Cogn. Sci. 11, 219–227.
Chalfonte, B.L., Johnson, M.K., 1996. Feature memory and binding in young and older adults. Mem. Cognition 24, 403–416.
Cohen, N.J., Eichenbaum, H., 1993. Memory, Amnesia, and the Hippocampal System. MIT Press, Cambridge, MA.
Cowan, N., Naveh-Benjamin, M., Kilb, A., Saults, J.S., 2006. Life-span development of visual working memory: when is feature binding difficult. Dev. Psychol. 42, 1089–1102.
Craik, F.I.M., 1986. A functional account of age differences in memory. In: Klix, F., Hagendorf, H. (Eds.), Human Memory and Capabilities: Mechanisms and Performances. Elsevier, Amsterdam, pp. 409–422.
Craik, F.I.M., Bialystok, E., 2006. Cognition through the lifespan: mechanisms of change. Trends Cogn. Sci. 10, 131–139.
Craik, F.I.M., Lockhart, R.S., 1972. Levels of processing: a framework for memory research. J. Verb. Learn. Verb. Behav. 11, 671–684.
Craik, F.I.M., McDowd, J.M., 1987. Age differences in recall and recognition. J. Exp. Psychol. Learn. 13, 474–479.
Crone, E.A., Donohue, S.E., Honomichl, R., Wendelken, C., Bunge, S.A., 2006. Brain regions mediating flexible rule use during development. J. Neurosci. 26, 11239–11247.
Cycowicz, Y.M., Friedman, D., Duff, M., 2003. Pictures and their colors: what do children remember. J. Cognitive Neurosci. 150, 759–768.
Cycowicz, Y.M., Friedman, D., Snodgrass, J.G., Duff, M., 2001. Recognition and source memory for pictures in children and adults. Neuropsychologia 39, 255–267.
Daselaar, S.M., Fleck, M.S., Dobbins, I.G., Madden, D.J., Cabeza, R., 2006. Effects of healthy aging on hippocampal and rhinal memory functions: an event-related fMRI study. Cereb. Cortex 16, 1771–1782.
Daselaar, S.M., Veltman, D.J., Rombouts, S.A.R.B., Raaijmakers, J.G.W., Jonker, C., 2003. Deep processing activates the medial temporal lobe in young but not in old adults. Neurobiol. Aging 24, 1005–1011.
Davachi, L., 2006. Item, context and relational episodic encoding in humans. Curr. Opin. Neurobiol. 16, 693–700.
Davachi, L., Wagner, A.D., 2002. Hippocampal contributions to episodic encoding: insights from relational and item-based learning. J. Neurophysiol. 88, 982–990.
Davidson, M.S., Amso, D., Anderson, L.C., Diamond, A., 2006. Development of cognitive control and executive functions from 4 to 13 years: evidence from manipulations of memory, inhibition, and task switching. Neuropsychologia 44, 2037–2078.
Davis, S.W., Dennis, N.A., Daselaar, S.M., Fleck, M.S., Cabeza, R., 2008. Que PASA? The posterior–anterior shift in aging. Cereb. Cortex 18, 1201–1209.
de Frias, C.M., Lovden, M., Lindenberger, U., Nilsson, L.G., 2007. Revisiting the dedifferentiation hypothesis with longitudinal multi-cohort data. Intelligence 35, 381–392.
Dennis, N.A., Daselaar, S.M., Cabeza, R., 2007. Effects of aging on transient and sustained successful memory encoding activity. Neurobiol. Aging 28, 1749–1758.
Dennis, N.A., Hayes, S.M., Prince, S.E., Madden, D.J., Huettel, S.A., Cabeza, R., 2008. Effects of aging on the neural correlates of successful item and source memory encoding. J. Exp. Psychol. Learn. 34, 791–808.
Dodson, C.S., Bawa, S., Krueger, L.E., 2007. Aging, metamemory, and high-confidence errors: a misrecollection account. Psychol. Aging 22, 122–133.
Donaldson, D.I., Peterson, S.E., Ollinger, J.M., Buckner, R.L., 2001. Dissociating state and item components of recognition memory during fMRI. Neuroimage 13, 129–142.
Dudai, Y., 2004. The neurobiology of consolidations, or, how stable is the engram? Annu. Rev. Psychol. 55, 51–86.
Dunlosky, J., Hertzog, C., 1998. Aging and deficits in associative memory: what is the role of strategy production? Psychol. Aging 13, 597–607.
Dunlosky, J., Hertzog, C., Powell-Moman, A., 2005. The contribution of mediator-based deficiencies to age differences in associative learning. Devel. Psychol. 41, 389–400.
Durston, S., Davidson, M.C., Tottenham, N., Galvan, A., Spicer, J., Fossella, J.A., Casey, B.J., 2006. A shift from diffuse to focal cortical activity with development. Dev. Sci. 9, 1–20.
Eichenbaum, H., 2002. The Cognitive Neuroscience of Memory: An Introduction. Oxford University Press, New York.
Erickson, K.I., Colcombe, S.J., Wadhwa, R., Bherer, L., Peterson, M.S., Scalf, P.E., Kim, J.S., Alvarado, M., Kramer, A.F., 2007. Training-induced plasticity in older adults: effects of training on hemispheric asymmetry. Neurobiol. Aging 28, 272–283.
Fernandez, G., Tendolkar, I., 2009. Declarative memory consolidation. In: Rösler, F., Ranganath, C., Röder, B., Kluwe, R.H. (Eds.), Neuroimaging of Human Memory: Linking Cognitive Processes to Neural Systems. Oxford University Press, New York, pp. 109–125.
Fletcher, P.C., Henson, R.N., 2001. Frontal lobes and human memory: insights from functional neuroimaging. Brain 124, 849–881.
Fletcher, P.C., Shallice, T., Dolan, R.J., 1998. The functional role of prefrontal cortex in episodic memory: I. Encoding. Brain 121, 1239–1248.
Foley, M.A., Johnson, M.K., 1985. Confusions between memories for performed and imagined actions: a developmental comparison. Child Dev. 56, 1145–1155.
Gathercole, S.E., 1998. The development of memory. J. Child Psychol. Psyc. 39, 3–27.
Ghetti, S., Angelini, L., 2008. The development of recollection and familiarity in childhood and adolescence: evidence from the dual-process signal detection model. Child Dev. 79, 339–358.
Gogtay, N., Nugent, T.F.I., Herman, D.H., Ordonez, A., Greenstein, D., Hayashi, K.M., Clasen, L., Toga, A.W., Giedd, J.N., Rapoport, J.L., Thompson, P.L., 2006. Dynamic mapping of normal human hippocampal development. Hippocampus 16, 664–672.
Grady, C.L., McIntosh, A.R., Craik, F.I.M., 2003. Age-related differences in the functional connectivity of the hippocampus during memory encoding. Hippocampus 13, 572–586.
Gutchess, A.H., Welsh, R.C., Hedden, T., Bangert, A., Minear, M., Liu, L.L., Park, D.C., 2005. Aging and the neural correlates of successful picture encoding: frontal activations compensate for decreased medial-temporal activity. J. Cognitive Neurosci. 17, 84–96.

Healy, M.R., Light, L.L., Chung, C., 2005. Dual-process models of associative recognition in young and older adults: evidence from receiver operating characteristics. J. Exp. Psychol. Learn. 31, 768–788.
Jacoby, L.L., Hay, J.F., 1998. Age-related deficits in memory: theory and application. In: Conway, M.A., Gathercole, S.E., Cornoldi, C. (Eds.), Theories of Memory. Psychology Press, Hove, UK, pp. 111–134.
Johnson, M.H., 2001. Functional brain development in humans. Nat. Rev. Neurosci. 2, 475–483.
Johnson, M.K., Hashtroudi, S., Lindsay, D.S., 1993. Source monitoring. Psychol. Bull. 114, 3–28.
Kausler, D.H., 1994. Learning and Memory in Normal Aging. Academic Press, New York.
Kramer, J.H., Rosen, H.J., Du, A.T., Schuff, N., Hollnagel, C., Weiner, M.W., Miller, B.L., Delis, D.C., 2005. Dissociations in hippocampal and frontal contributions to episodic memory performance. Neuropsychology 19, 799–805.
Li, S.-C., Naveh-Benjamin, M., Lindenberger, U., 2005. Aging neuromodulation impairs associative binding: a neurocomputational account. Psychol. Sci. 16, 445–450.
Lindenberger, U., 2001. Lifespan theories of cognitive development. In: Smelser, N.J., Baltes, P.B. (Eds.), International Encyclopedia of the Social and Behavioral Sciences. Elsevier Science, Oxford, UK, pp. 8848–8854.
Lindenberger, U., Baltes, P.B., 1997. Intellectual functioning in old and very old age: cross-sectional results from the Berlin Aging Study. Psychol. Aging 12, 410–432.
Lindenberger, U., Li, S.-C., Bäckman, L., 2006. Delineating brain–behavior mappings across the lifespan: substantive and methodological advances in developmental neuroscience. Neurosci. Biobehav. R 30, 713–717.
Lindsay, D.S., Johnson, M.K., Kwon, P., 1991. Developmental changes in memory source monitoring. J. Exper. Child Psychol. 52, 297–318.
Logan, J.M., Sanders, A.L., Snyder, A.Z., Morris, J.C., Buckner, R.L., 2002. Under-recruitment and nonselective recruitment: dissociable neural mechanisms associated with aging. Neuron 33, 827–840.
Luck, S.J., Vogel, E.K., 1997. The capacity of visual working memory for features and conjunctions. Nature 390, 279–281.
Mattay, V.S., Fera, F., Tessitore, A., Hariri, A.R., Berman, K.F., Das, S., Berman, K.F., Das, S., Meyer-Lindenberg, A., Goldberg, T.E., Callicott, J.H., Weinberger, D.R., 2006. Neurophysiological correlates of age-related changes in working memory capacity. Neurosci. Lett. 392, 32–37.
McIntosh, A.R., Nyberg, L., Bookstein, F.L., Tulving, E., 1997. Differential functional connectivity of prefrontal and medial temporal cortices during episodic memory retrieval. Human Brain Mapp. 5, 323–327.
Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Ann. Rev. Neurosci. 24, 167–202.
Mitchell, K.J., Johnson, M.K., Raye, C.L., D'Esposito, M., 2000. fMRI evidence of age-related hippocampal dysfunction in feature binding in working memory. Cognitive Brain Res. 10, 197–206.
Morcom, A.M., Good, C.D., Frackowiak, R.S.J., Rugg, M.D., 2003. Age effects on the neural correlates of successful memory encoding. Brain 126, 213–229.
Moscovitch, M., 1992. Memory and working-with-memory: a component process model based on modules and central systems. J. Cognitive Neurosci. 4, 257–267.
Moscovitch, M., Nadel, L., Winocur, G., Gilboa, A., Rosenbaum, R.S., 2006. The cognitive neuroscience of remote episodic, semantic and spatial memory. Curr. Opin. Neurobiol. 16, 179–190.
Müller, V., Gruber, W., Klimesch, W., Lindenberger, U., 2009. Lifespan differences in cortical dynamics of auditory perception. Dev. Sci. 12, 839–853.
Murre, J.M.J., Wolters, G., Raffone, A., 2006. Binding in working memory and long-term memory: towards an integrated model. In: Zimmer, H.D., Mecklinger, A., Lindenberger, U. (Eds.), Handbook of Binding and Memory: Perspectives from Cognitive Neuroscience. Oxford University Press, Oxford, UK, pp. 221–250.
Naveh-Benjamin, M., 2000. Adult age differences in memory performance: tests of an associative deficit hypothesis. J. Exp. Psychol. Learn. 26, 1170–1187.
Naveh-Benjamin, M., Brav, T.K., Levy, O., 2007. The associative memory deficit of older adults: the role of strategy utilization. Psychol. Aging 22, 202–208.
Nelson, C.A., 2001. The ontogeny of human memory: a cognitive neuroscience perspective. In: Johnson, M.H., Munakata, Y., Gilmore, R.O. (Eds.), Brain Development and Cognition: A Reader. Blackwell Publishing, Oxford, UK, pp. 151–178.
Norman, K.A., Schacter, D.L., 1997. False recognition in younger and older adults: exploring the characteristics of illusory memories. Mem. Cognition 25, 838–848.
O'Reilly, R.C., Norman, K.A., 2002. Hippocampal and neocortical contributions to memory: advances in the complementary learning systems framework. Trends Cogn. Sci. 6, 505–510.
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. Nat. Neurosci. 10, 1198–1205.
Old, S.R., Naveh-Benjamin, M., 2008. Differential effects of age on item and associative measures of memory: a meta-analysis. Psychol. Aging 23, 104–118.
Otten, L.J., Rugg, M.D., 2001. Task-dependency of the neural correlates of episodic encoding as measured by fMRI. Cereb. Cortex 11, 1150–1160.
Paller, K.A., Wagner, A.D., 2002. Observing the transformation of experience into memory. Trends Cogn. Sci. 6, 93–102.
Park, D.C., Gutchess, A.H., 2005. Long-term memory and aging: a cognitive neuroscience perspective. In: Cabeza, R., Nyberg, L., Park, D.C. (Eds.), Cognitive Neuroscience of Aging: Linking Cognitive and Cerebral Aging. Oxford University Press, New York, pp. 218–245.
Paz-Alonso, P.M., Ghetti, S., Donohue, S.E., Goodman, G.S., Bunge, S.A., 2008. Neurodevelopmental correlates of true and false recognition. Cereb. Cortex 18, 2208–2216.
Perlmutter, M., Lange, G., 1978. A developmental analysis of recall-recognition distinctions. In: Ornstein, P.A. (Ed.), Memory Development in Children. Erlbaum, Hillsdale, NJ, pp. 243–258.
Perner, J., Ruffman, T., 1995. Episodic memory and autonoetic consciousness: developmental evidence and a theory of childhood amnesia. J. Exper. Child Psychol. 59, 516–548.
Poole, D.A., Lindsay, D.S., 1995. Interviewing preschoolers: effects of nonsuggestive techniques, parental couching and leading question on reports of nonexperienced events. J. Exper. Child Psychol. 60, 129–154.
Prull, M.W., Gabrieli, J.D.E., Bunge, S.A., 2000. Age-related changes in memory: a cognitive neuroscience perspective. In: Craik, F.I.M., Salthouse, T.A. (Eds.), The Handbook of Aging and Cognition. Erlbaum, Mahwah, NJ, pp. 91–153.
Rajah, M.N., D'Esposito, M., 2005. Region-specific changes in prefrontal function with age: a review of PET and fMRI studies on working and episodic memory. Brain 128, 1964–1983.
Raz, N., Lindenberger, U., Ghisletta, P., Rodrigue, K.M., Kennedy, K.M., Acker, J.D., 2008. Neuroanatomical correlates of fluid intelligence in healthy adults and persons with vascular risk factors. Cereb. Cortex 18, 718–726.
Raz, N., Lindenberger, U., Rodrigue, K.M., Kennedy, K.M., Head, D., Williamson, A., Dahle, C., Gerstorf, D., Acker, J.D., 2005. Regional brain changes in aging healthy adults: general trends, individual differences, and modifiers. Cereb. Cortex 15, 1676–1689.
Raz, N., Rodrigue, K.M., Head, D., Kennedy, K.M., Acker, J.D., 2004. Differential aging of the medial temporal lobe: a study of a five-year change. Neurology 62, 433–438.
Reuter-Lorenz, P.A., Cappell, K.A., 2008. Neurocognitive aging and the compensation hypothesis. Curr. Dir. Psychol. Sci. 17, 177–182.
Rönnlund, M., Nyberg, L., Bäckman, L., Nilsson, L.-G., 2005. Stability, growth, and decline in adult life span development of declarative memory: cross-sectional and longitufinal data from a population-based study. Psychol. Aging 20, 3–18.
Rubin, D.C., 2006. The basic-systems model of episodic memory. Perspect. Psychol. Sci. 1, 277–311.
Rugg, M.D., Wilding, E.L., 2000. Retrieval processing and episodic memory. Trends Cogn. Sci. 4, 108–115.
Schaie, K.W., 1965. A general model for the study of developmental problems. Psychol. Bull. 64, 92–107.
Schaie, K.W., 1996. Intellectual Development in Adulthood: The Seattle Longitudinal Study. Cambridge University Press, New York.
Schneider, W., Pressley, M., 1997. Memory Development Between Two and Twenty, Second ed. Erlbaum, Mahwah, NJ.
Schneider, W., Shiffrin, R.M., 1977. Controlled and automatic human information processing: I. Detection, search, and attention. Psychol. Rev. 84, 1–66.
Shing, Y.L., Werkle-Bergner, M., Li, S.-C., Lindenberger, U., 2008. Associative and strategic components of episodic memory: a lifespan dissociation. J. Exp. Psychol. Gen. 137, 495–513.
Shing, Y.L., Werkle-Bergner, M., Li, S.-C., Lindenberger, U., 2009. Committing memory errors with high confidence: older adults do but children don't. Memory 17, 169–179.
Simons, J.S., Spiers, H.J., 2003. Prefrontal and medial temporal lobe interactions in long-term memory. Nat. Rev. Neurosci. 4, 637–648.
Singer, T., Verhaeghen, P., Ghisletta, P., Lindenberger, U., Baltes, P.B., 2003. The fate of cognition in very old age: six-year longitudinal findings in the Berlin Aging Study (BASE). Psychol. Aging 18, 318–331.
Sluzenski, J., Newcombe, N., Kovacs, S.L., 2006. Binding, relational memory, and recall of naturalistic events: a developmental perspective. J. Exp. Psychol. Learn 32, 89–100.
Souchay, C., Isingrini, M., 2004. Age related differences in metacognitive control: role of executive functioning. Brain Cognition 56, 89–99.
Sowell, E.R., Peterson, B.S., Thompson, P.M., Welcome, S.E., Henkenius, A.L., Toga, A.W., 2003. Mapping cortical change across the human life span. Nat. Neurosci. 6, 309–314.
Spencer, W.D., Raz, N., 1995. Differential effects of aging on memory for content and context: a meta-analysis. Psychol. Aging 10, 527–539.
Squire, L.R., 2004. Memory systems of the brain: a brief history and current perspective. Neurobiol. Learn. Mem. 82, 171–177.
Takashima, A., Petersson, K.M., Rutters, F., Temdolkar, I., Jensen, O., Zwarts, M.J., McNaughton, B.L., Fernandez, G., 2006. Declarative memory consolidation in humans: a prospective functional magnetic resonance imaging study. Proc. Natl. Acad. Sci. 103, 756–761.
Treisman, A., 1996. The binding problem. Curr. Opin. Neurobiol. 6, 171–178.
Tulving, E., 1972. Episodic and semantic memory. In: Tulving, E., Donaldson, W. (Eds.), Organization of Memory. Academic Press, New York, pp. 381–403.
Underwood, B.J., 1969. Attributes of memory. Psychol. Rev. 76, 559–573.
Warrington, E.K., Weiskrantz, L., 1982. Amnesia: a disconnection syndrome? Neuropsychologia 20, 233–248.
Werkle-Bergner, M., Müller, V., Li, S.-C., Lindenberger, U., 2006. Cortical EEG correlates of successful memory encoding: implications for lifespan comparisons. Neurosci. Biobehav. R 30, 839–854.
Werner, H., 1957. The concept of development from a comparative and organismic point of view. In: Harris, D.B. (Ed.), The Concept of Development: An Issue in the Study of Human Behavior. University of Minnesota Press, Minneapolis, MN, pp. 125–148.

West, R.L., 1996. An application of prefrontal cortex function theory to cognitive aging. Psychol. Bull. 120, 272–292.
Wheeler, M.R., Buckner, R.L., 2003. Functional dissociation among components of remembering: control, perceived oldness, and content. J. Neurosci. 23, 3869–3880.
Wheeler, M.A., Stuss, D.T., Tulving, E., 1997. Toward a theory of episodic memory: the frontal lobes and autonoetic consciousness. Psychol. Bull. 121, 331–354.
Wilson, I.A., Gallagher, M., Eichenbaum, H., Tanila, H., 2006. Neurocognitive aging: prior memories hinder new hippocampal encoding. Trends Neurosci. 29, 662–670.
Yonelinas, A.P., 2002. The nature of recollection and familiarity: a review of 30 years of research. J. Mem. Lang. 46, 441–517.
Zimmer, H.D., Mecklinger, A., Lindenberger, U. (Eds.), 2006. Handbook of Binding and Memory: Perspectives from Cognitive Neuroscience. Oxford University Press, Oxford, UK.