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Episodic Memory Across the Lifespan

General Trajectories and Modifiers

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Introduction

Episodic memory (EM) refers to memory about events that are bound to specific times and places in the past (Tulving, 2002). It allows humans to re-experience multiple aspects of events that happened from minutes to years ago. The remembering of previously experienced episodes increases during childhood (Schneider and Pressley, 1997) and declines in old and very old age (Kausler, 1994). At first sight, then, it might appear that changes in adulthood are a reversal or mirror image of changes during childhood. However, development of EM is driven by a constellation of factors, including changes in neural brain mechanisms, accumulation of experience and learning, and genetic influences (Lindenberger, Li, and Bäckman, 2006; Werkle-Bergner et al., 2006). Importantly, the influences of these factors do not remain constant across the lifespan, such that the lower performance levels in children and older adults relative to younger adults may differ in etiology (Baltes, Lindenberger, and Staudinger, 2006).

In the present chapter, we provide an overview of the general trajectories of memory development across the lifespan, integrating both behavioral and neural evidence. We adopt the two-component framework (Shing et al., 2010) that conceptualizes change in EM across the lifespan as the interplay of two largely independent but interacting components, one associative and the other strategic (cf. Simons and Spiers, 2003). The associative component refers to mechanisms that bind different features of an event into a coherent representation, and is mediated by areas of the medial temporal lobe (MTL) at the neural level (Zimmer, Mecklinger, and Lindenberger, 2006; see also Chapter 18). The strategic component, on the other hand, refers to control processes that aid and regulate memory functions at encoding and retrieval (Chapter 7; for more discussion of the development of strategic memory processes, see Chapter 16). Neurally, the strategic component is supported by regions of the prefrontal cortex (PFC; Miller and Cohen, 2001) and the parietal lobes (Cabeza et al., 2008). A series
of behavioral experiments (Brehmer et al., 2007; Shing et al., 2008) provided initial evidence for a dissociation in the lifespan developmental trajectories of the two components, such that the associative component is relatively functional by middle childhood (ages 10–12), but exhibits age-related decline in older adults. In contrast, the strategic component is functioning below the levels of younger adults in both children and older adults, in line with the protracted maturation and early age-related decline in PFC regions. Normative age gradients in associative and strategic components coexist with individual differences in change (de Frias et al., 2007; Ghisletta et al., 2012) and plasticity (Fandakova, Shing, and Lindenberger, 2012). In the second part of the present chapter we address several factors that have been shown to affect individual differences in EM mechanisms at different life periods. We attempt to interpret these findings from the perspectives of the two-component framework of EM.

**EM Across the Lifespan: General Trajectories**

Evidence for memory improvement in children

EM undergoes substantial changes from infancy to adolescence. With some tasks, performance in source memory tasks is already above chance by preschool years (Lindsay, Johnson, and Kwon, 1991), but with more difficult tasks, such as distinguishing between internally generated stimuli or making distinctions after substantial delays, developmental improvements are observed into the school years (Kovacs and Newcombe, 2006). Preschoolers have difficulties in binding together an item and its context into a coherent representation (Sluzenski, Newcombe, and Ottinger, 2004). In contrast, there seems to be little change in binding abilities after the age of six (Sluzenski, Newcombe, and Kovacs, 2006). Recollection of specific details associated with past events also improves gradually during childhood, with little or no developmental change observed in familiarity-based processing (Ghetti and Angelini, 2008). Developmental differences are also observed when memory for specific details of past events (i.e., verbatim traces) is compared to the ability to extract the general semantic meaning of past events (i.e., gist traces). While increases in verbatim traces are observed during preschool and early elementary school years, gist traces continue to develop up to adolescence (Brainerd and Reyna, 2005).

With increasing age, the use of elaborative strategies to support the formation of new memories becomes an increasingly important part of children’s learning behavior, especially between late childhood and late adolescence (Schneider and Pressley, 1997). Age-related improvements in metamemory also contribute to the rise of memory accuracy during childhood (Ghetti, Castelli, and Lyons, 2010). Metamemory refers to a set of constructs, including beliefs, awareness, and knowledge about one’s memory, as well as about different memory strategies and their effectiveness in a particular task setting (Nelson and Narens, 1990). While children’s ability to utilize memory strategies to facilitate EM depends on a number of developmental factors such as metamemory and processing resources, temperament and motivation may also play important roles (Bjorklund et al., 1997; Miller and Seier, 1994).
Evidence for memory decline in older adults

At the other end of the lifespan, aging is associated with a pronounced decline in EM functioning (Ghisletta et al., 2012; Rönnlund et al., 2005; see also Chapters 17, 18, and 19). Different aspects of memory performance are disproportionally affected by aging, with memory for content showing smaller age-related decrease than memory for context (Chalfonte and Johnson, 1996; Spencer and Raz, 1995). Older adults exhibit difficulties creating and retrieving intra- and inter-item associations (Old and Naveh-Benjamin, 2008; see also Chapter 18). During retrieval, recollection of particular contextual details is more strongly affected by senescent changes than familiarity-based processing (Light et al., 2000).

Age-related differences in EM are magnified under conditions that require self-initiated strategic processing (Cohn, Emrich, and Moscovitch, 2008). Older adults are less likely than younger adults to spontaneously use effective strategies to mediate memory performance (Dunlosky and Hertzog, 1998). Metamemory abilities also decline during senescence (Dodson and Krueger, 2006). A number of studies have examined cognitive decline in relation to proximity to death in older individuals (i.e., terminal decline). Increased decline in EM has been identified as early as 8.4 years prior to death (Sliwinski et al., 2006), with a rate twice that observed as a function of chronological age (MacDonald, Hultsch, and Dixon, 2011). However, the paths associated with aging and terminal decline vary greatly across individuals (Ghisletta et al., 2012; Lindenberger and Ghisletta, 2009) as a function of various factors, including lifestyle (e.g., Schaie, 2012), vascular risk (e.g., Raz et al., 2005), and genetic influences (e.g., Deary et al., 2012).

Taken together, both children and older adults perform below the level of younger adults under conditions that require the detailed recollection of specific contextual details from previous experiences. Furthermore, strategic and metacognitive abilities of both age groups seem to be less efficient compared to younger adults. However, while associative binding is relatively functional by middle childhood, older adults show difficulties in forming and retrieving associations of episodic details.

Neural evidence from child development

**Functional differences at encoding.** Few studies have examined age differences in neural activation during EM tasks in childhood and adolescence. In general, developmental differences in memory functioning are paralleled by differences in the functional and structural integrity of the underlying brain circuitry during development. For example, Ofen and colleagues (2007) found that during the encoding of subsequently remembered scenes, PFC, but not MTL, activation increased with age in children and adults between 8 and 24 years. This finding is in contrast with a study by Ghetti and colleagues (2010), who, for both adolescents and younger adults, found higher activations in the hippocampus and the posterior parahippocampal gyrus during incidental encoding of items that were later recollected with specific detail compared to those that were later on forgotten. However, younger children (8 and 10–11 years old) did not show such discrimination, suggesting an increasing specialization of MTL regions to support recollection even in middle childhood.

**Functional differences in retrieval.** Only a few studies examined age differences in neural activation during EM retrieval. Paz-Alonso and colleagues (2008) investigated
retrieval activations for true and false memories among 8-year-olds, 12-year-olds, and adults. Anterior MTL was engaged in item-specific recollection for 12-year-olds and adults, but not in the younger children. Children of both age groups engaged ventrolateral and anterior PFC to a lesser degree than adults, suggesting continued maturation of semantic and monitoring processes during childhood. These findings were only partially supported by a recent neuroimaging study that examined retrieval of previously studied complex scenes in 8- to 21-year-olds (Ofen et al., 2012). In this study, activation in ventrolateral PFC increased with age for successfully retrieved scenes, confirming the late maturation of PFC regions. However, in contrast to Paz-Alonso and colleagues (2008), no age differences in MTL activations were found. Notably, age-related increases in parietal activations across middle childhood and adolescence were consistently reported in both studies. More research is needed to understand the neural mechanisms by which age differences in parietal regions contribute to age-related increases in the ability to recollect past episodes that are rich in contextual details.

Taken together, the majority of studies reported developmental trends in PFC regions that support control aspects of EM. On the structural level, gray matter volume in the frontal lobes initially increases up to middle childhood and subsequently declines during adolescence, with the most dorsal aspects of the frontal regions showing the latest maturation (Sowell et al., 2003). White matter volume increases linearly in both anterior–posterior and inferior–superior directions during childhood and adolescence (Colby, Van Horn, and Sowell, 2011), probably reflecting increments in the speed and efficiency of communication among brain regions as a consequence of axon myelination.

In contrast, the findings with regard to age differences in MTL activation are mixed. One possible reason for this discrepancy might be the heterogeneity of maturational changes across subregions of the hippocampus. For instance, Gogtay and collaborators (2006) did not find changes in total hippocampal volume between 4 and 25 years, but reported that the volume of the posterior hippocampus gradually increased with age, whereas the volume of the hippocampal head decreased with age. With regard to the functional significance of these structural changes, successful retrieval of item–color associations was shown to engage distinct regions along the hippocampal axis in children (8- to 11-year-olds) and adults (DeMaster and Ghetti, 2013). While children engaged the posterior hippocampus when correctly remembering the color with which line drawings were associated when previously studied, correct source memory was associated with anterior hippocampus activation in younger adults.

**Neural evidence from aging**

**Structural and functional changes in MTL.** At the other end of the lifespan, age-related changes in the functional and structural integrity of the cortical network supporting EM, particularly the PFC and MTL, are frequently observed in neuroimaging studies (for a review, see Chapter 17). Gray matter changes are especially pronounced in the hippocampus, and less so in the surrounding cortex (Raz et al., 2005). Findings regarding MTL functional alterations during episodic encoding and retrieval in old age are contradictory, with some studies reporting age-related decreases (Daselaar et al., 2006; Grady, McIntosh, and Craik, 2003), and others not (Dulas and Duarte, 2011; Persson et al., 2010). One potential factor that may explain differences across studies is that differences in brain activation are often confounded by differences in
memory performance, which compromise the interpretation of results at the neural level (Rugg and Morcom, 2005). In addition, age-related decreases in hippocampal activations are relatively minor before age 70 (Salami, Eriksson, and Nyberg, 2012), suggesting that differences across studies may be partly related to age range differences of the study samples.

Longitudinally, Persson and colleagues (2012) reported that for some older adults (55–79 years) examined in the context of the Betula study, memory performance decreased across a period of 10 years, whereas for others it remained stable or even increased. Importantly, a decrease in hippocampal activation and gray matter volume was observed only in the older adults who showed a pronounced decline in memory performance, but not in the older adults who remained stable in their performance. This study is important because it directly related longitudinal decline in memory performance in old age to functional and structural changes in relevant brain areas. At the same time, it underscores the need to delineate the physiological correlates that help to maintain memory performance in old age (Nyberg et al., 2012).

**Structural and functional changes in PFC.** Prefrontal brain regions are among the areas that undergo the strongest atrophy in old age (Raz et al., 2005). Changes in the microstructure of white matter integrity accompany these gray matter losses (Burzynska et al., 2010). Older adults often show lower PFC activation during both encoding (Dennis et al., 2008; Dulas and Duarte, 2011) and retrieval of past episodes (Duarte, Henson, and Graham, 2008; Fandakova, Lindenberger, and Shing, 2013). In contrast, some studies have reported additional PFC activation in older adults compared with younger adults (Cabeza et al., 2002), which has been interpreted as compensatory activity for age-related decline in posterior brain regions. Alternatively, additional PFC activations might reflect decrease of neural efficiency or less differentiated processing with advancing adult age (Baltes and Lindenberger, 1997).

It is worth noting that most analyses suggesting “over-recruitment” of taskrelevant brain regions with advancing adult age rely on cross-sectional evidence. Again, findings from the Betula study provide a notable exception. Nyberg and colleagues (2010) investigated longitudinal change in brain structure and function over a period of six years. While the cross-sectional analysis suggested increased activation of dorsal PFC in older adults, longitudinally activity in this region decreased, indicating that aging is associated with under- rather than over-recruitment of PFC regions. These findings are corroborated by recent evidence showing that older adults who deviate less from younger adults in the brain networks engaged during incidental picture encoding have higher recognition performance (Düzel et al., 2010), suggesting that the extent of preservation in functional networks in old age is an important determinant of individual differences in memory performance. Based on this pattern of findings, Nyberg et al. (2012) suggested that brain maintenance (or relative lack of brain pathology) constitutes the primary determinant of successful memory aging (see also Lindenberger, Burzynska, and Nagel, 2013).

**EM Across the Lifespan: Modifiers**

As reviewed above, a wide range of evidence points to substantial heterogeneity in EM performance during all age periods, and to reliable and substantial individual differences in developmental change. To understand these individual differences in level
and change, researchers need to delineate the mechanisms that influence the memory development of individuals. These mechanisms are likely to unfold as epigenetic interactions between genetic makeup and environmental factors, and appear as lifestyle choices such as physical exercise, cognitive stimulation, nutrition, social participation, and other dimensions of daily life that influence memory performance. In the following sections, we selectively review three key factors that have been identified to influence individual differences in level and change of memory performance: one in childhood (parental style), one in adulthood (vascular risk), and one that operates at both ends of the lifespan (physical fitness). We also provide an overview of key findings from the cognitive training literature, focusing on individual differences in training, as a demonstration of memory plasticity across the lifespan (for more discussion of memory training, see Chapter 21). These factors are discussed in the context of the two-component framework. We aim to demonstrate the framework’s utility for classifying them according to their effect on associative and strategic aspects of EM. Doing so may foster our understanding of their common and distinctive effects, and may help generate questions and models to be tested in future research.

**Parental style**

Animal models suggest that environmental enrichment in early life has long-lasting beneficial effects on brain development (Greenough, Black, and Wallace, 1987). However, the specific mechanisms by which experience shapes the brain in humans are not well understood. Enriched environment entails complexity in the sensory input and social stimulation from an individual’s surroundings. In early childhood, a major share of social stimulation is related to the parents and/or primary caregivers. Of particular relevance to human memory development are retrospective data suggesting a link between lack of early nurturance due to maltreatment and later impaired brain development, including hippocampal volumes. Animal models of stress provide hypotheses of how the hippocampus is particularly vulnerable to the effects of adverse early environment (see review in Tottenham and Sheridan, 2009).

There are relatively few human studies that prospectively track the effect of parental care on subsequent brain and cognitive development. In a longitudinal study of depressed and healthy children, maternal support (as measured with a parent–child interaction paradigm) in early childhood was positively associated with hippocampal volume measured at school age (Luby et al., 2012). The association between maternal support and hippocampal volumes was stronger in the sample of non-depressed children than in the sample of depressed children. In another longitudinal study by Rao and colleagues (2010), parental nurturance (as measured by warmth and availability of parental care) and environmental stimulation (as measured by the availability of cognitively stimulating toys and activities) were measured at ages 4 and 8 years. During adolescence, the participants underwent structural brain imaging. Parental nurturance at age 4 (but not at age 8) was associated with the volume of the left hippocampus in adolescence, but in the unexpected direction of better nurturance associated with smaller hippocampal volume. Environment stimulation, on the other hand, showed no effect on hippocampal volume. These findings point to the possibility of sensitive periods in which parental factors show heightened influence on subsequent brain development. Furthermore, the directionality of parental influence on subsequent hippocampal volume may be nonlinear, and needs to be interpreted taking into
account that the hippocampus volume undergoes an inverted U-shape trajectory across development (Gogtay et al., 2006).

Taken together, adverse early experiences shape brain development, including a negative stress-related effect on the hippocampus. However, the mechanisms underlying such environment–brain relations cannot be readily generalized to the effects of less extreme environments. While the existing evidence suggests normal range variation in parental factors is associated with subsequent brain development (particularly the hippocampus, related to the associative component of EM), many questions are left open. First, the two longitudinal studies reviewed above (Luby et al., 2012; Rao et al., 2010) did not measure hippocampal volume in early childhood, or in the parents. Therefore, one cannot be certain that the reported results do not merely reflect common sources of variance, notably genetic differences. This is a common critical issue in studies that examine effects of parenting practices, which calls for innovative use of methodologies, including animal models (e.g., Freund et al., 2013), to probe environmental influences on epigenetic effects. Second, studies that simultaneously measure parental factors, brain development, and memory development are scarce. The empirical links across neural, behavioral, and cognitive levels of analysis are yet to be demonstrated. Third, while parents and primary caregivers play an important role in the life of developing children, schooling context becomes increasingly salient in older children. Given behavioral evidence that suggests teachers’ memory-relevant language is related to the development of children’s memory skills (Coffman et al., 2008), there is a need to better understand the influence of schooling context on neural development underlying memory functioning.

Vascular health

Vascular changes are among the most important modifiers of normal aging. A growing body of evidence links vascular factors to age-associated changes in cognition during adulthood and old age. Indicators of vascular risk such as higher blood pressure, body mass index, cholesterol level, blood sugar level, and others have been associated with lower cognitive performance in old age, including an increased risk of being diagnosed with dementia. A number of behavioral measures are associated with lower vascular risk and maintenance of good health in old age, including exercise and refraining from smoking (for a review see Warsch and Wright, 2010).

Structural, functional, and behavioral evidence indicates that vascular health protects against accelerated forms of cognitive aging. For example, Raz et al. (2005) reported that hypertension is associated with shrinkage of the hippocampus in a sample of healthy older adults. In this study, age-related acceleration of shrinkage in the hippocampus was limited to hypertensive participants (treated with medication). Furthermore, Shing et al. (2011) reported smaller CA1 subfields of hippocampus in older adults with hypertensive status, whereas normotensive older adults showed CA1 volumes within the range of younger adults. This finding demonstrates regional differences in vulnerability to vascular disease within the hippocampus (Wu et al., 2008).

The extent to which vascular factors are a potential risk for cognitive decline may interact with common genetic variation. Bender and Raz (2012) found that normotensive carriers of the apolipoprotein (ApoE) ε4 allele with elevated systolic blood pressure showed lower verbal recognition than ε4 carriers with lower blood pressure.
Of note, blood pressure had a negative effect on the prefrontal volumes, but not hippocampal volumes, of ApoE ε4 carriers. Similar interactions between genetic and vascular risk in association with cognitive deficits have been reported previously. For instance, Raz et al. (2008) reported that elevated blood glucose predicted lower memory scores only in carriers of the 66Met allele of the BDNF gene, which is assumed to be associated with lower levels of the brain-derived neurotrophic factor in the central nervous system relative to Val homozygotes. These results suggest that the combination of a relatively mild elevation in physiological indicators of vascular risk with a genetic risk factor may lead to poorer functioning of brain and cognition.

Taken together, these studies indicate that vascular risk has a negative effect on memory functioning. Interacting with genetic burden, vascular risk seems to affect PFC and hippocampus volumes in particular, resulting in lower associative memory. Future studies should address the effect of interventions that try to reduce blood pressure, especially in individuals with genetic risk factors. In addition, future research is needed to delineate the degree to which vascular risk affects specific aspects of memory functioning. Based on the two-component framework, it would be expected that vascular risk would have a negative influence on both strategic and associative processes that strongly rely on PFC and hippocampal functioning.

Physical fitness
Physical fitness has profound effects on brain and cognitive function, during both child development and aging. In school-aged children, physical activity is positively associated with measures of learning and general cognitive ability (Sibley and Etnier, 2003). Recent studies have attempted to link the positive effects of physical fitness on memory to underlying brain mechanisms. For instance, Chaddock and colleagues (2010) investigated the effects of physical fitness on relational memory and hippocampal volume among higher- and lower-fit 9- and 10-year-olds. Both groups did not differ in item memory, but higher-fit children performed better than lower-fit children in an associative memory task. Higher-fit children also had greater hippocampal volumes than lower-fit children, and individual differences in hippocampal volumes were positively related to relational (but not item) memory performance across all children. The effect of physical fitness on relational memory was directly tested in a subsequent study (Monti, Hillman, and Cohen, 2012), in which a group of preadolescent children underwent a nine-month after-school aerobic exercise intervention and was compared to a waiting-list control group on measures of item and relational memory for faces and scenes. The groups did not differ in memory performance for item or relational information, but the intervention group allocated a greater proportion of time on viewing the correctly recognized faces. As the proportion of viewing time in relational memory paradigms has been related to the structure and functioning of the hippocampus (Hannula and Ranganath, 2009), these group differences may reflect more efficient hippocampal involvement in relational memory following increase in aerobic fitness (Monti, Hillman, and Cohen, 2012).

The beneficial effects of physical fitness have also been documented in old age. Older adults who underwent an aerobic training intervention for six months showed reliable increases in brain gray and white matter volume (Colcombe et al., 2006), and altered patterns of task-related functional activation (Colcombe et al., 2004). Paralleling the results from child development, Erickson and colleagues (2009) found
that the positive relationship between aerobic fitness and spatial memory performance was predicted by individual differences in hippocampal volume. The neural mechanisms driving the positive effects of aerobic fitness on brain status and cognitive performance in humans are not yet well understood. Evidence from animal studies suggests that these effects are, at least to some degree, associated with neurogenesis in the hippocampus, and the dentate gyrus in particular, due to changes in neurotransmitter and growth factor release (Kempermann, 2008; van Praag, 2009).

The extant evidence suggests that aerobic fitness has positive effects on the associative component of EM. However, studies in both children and older adults indicate that the positive effects of aerobic fitness training on brain functioning are not restricted to the hippocampus but may also affect brain networks engaged during tasks of attention and cognitive control (Chaddock et al., 2012; Colcombe et al., 2004). Thus, the beneficial effects of aerobic fitness may not be restricted to the associative, but also directly affect the strategic component of EM at both ends of the lifespan. Alternatively, the effects of aerobic fitness on the strategic component may not be direct, but may result from associative–strategic interactions such that a more functional associative component decreases the demand on memory control processes.

Given the importance of aerobic fitness for hippocampus and memory in old age, several studies have examined to what extent physical training may have a beneficial effect on ApoE ε4 allele carriers, who are at higher risk for developing Alzheimer’s disease in old age. Initial evidence from this research indicates that physical fitness may serve as a protective factor for ApoE ε4 carriers, such that aerobic fitness is positively associated with increased functional activation (Deeny et al., 2008) and better cognitive functioning in individuals with greater genetic risk for Alzheimer’s disease (Etnier et al., 2007). However, another study reported higher performance benefit from an aerobic training in ApoE ε4 non-carriers compared to ApoE ε4 carriers, indicating that cognitive benefits from physical exercise may be attenuated by genetic risk factors (Lautenschlager et al., 2008). These results suggest that the beneficial effects of physical fitness interventions may not be linear, but may depend on the functional status of the individual at the onset of the training. Finally, a recent study with older adults revealed that executive functioning, along with use of self-regulatory strategies and self-efficacy measures, at the beginning of a physical exercise program were predictive of adherence to the intervention (McAuley et al., 2011). The importance of these factors is not restricted to aerobic training programs, and should receive more attention in future developmental training studies.

Individual differences in memory training gains

EM performance in childhood and old age can be improved through instruction and practice (e.g., Noack et al., 2009; see also Chapter 21). Nevertheless, even after extensive practice, older adults do not reach the levels of performance of younger adults (Baltes and Kliegl, 1992). In contrast, when given the possibility to optimize a newly acquired strategy through extensive practice, children can advance to the level of younger adults (Brehmer et al., 2007, 2008; Shing et al., 2008). Importantly, there is a substantial degree of heterogeneity in training benefits in both children and older adults. Understanding the factors contributing to these age and individual differences is an important task for developmental research, as it will help identify programs and interventions that target specific mechanisms that may differ across individuals.
Instruction of an elaborative imagery strategy was shown to effectively increase memory performance in both children and older adults (Brehmer et al., 2007; Shing et al., 2008). In contrast, following extensive practice of the strategy, children surpassed older adults, presumably reflecting differences in associative binding mechanisms that may be relatively mature in school-aged children, but undergo age-related decline in older adults (Brehmer et al., 2007; Shing et al., 2008). In the samples of children and older adults originally reported by Brehmer and colleagues (2007), only children improved performance without further practice across an 11-month period of no testing, probably reflecting maturational changes in the brain networks underlying the strategic component (Brehmer et al., 2008). Furthermore, individual differences in initial performance gains (i.e., immediately following mnemonic strategy instruction) correlated negatively with baseline performance (Lövden et al., 2012). In line with conceptual considerations (Lövdén et al., 2010), this finding suggests that individuals who were already implementing efficient strategies at baseline had less to gain from strategic instruction. In contrast, the correlation between baseline performance and gains flipped its sign in both groups after extensive practice with the memory strategy, suggesting that the potential for plasticity of the associative component is higher among individuals with higher baseline performance.

In particular, the reanalysis of the Brehmer et al. (2007) results by Lövdén et al. (2012) suggests that individual differences in memory-relevant mechanisms and strategies modulate the benefits of training. For example, individuals with lower memory functioning may require a more directed strategy instruction, reflecting their greater need for environmental support (cf. Craik, 1983). In contrast, for individuals with relatively preserved strategic and associative functioning, providing the context for self-initiated strategy use followed by extensive practice may be sufficient (Fandakova, Shing, and Lindenberger, 2012; Jones et al., 2006). Accordingly, in recollection strategy training with older adults, self-initiation of controlled processing predicted individual differences in training efficacy (Bissig and Lustig, 2007). These findings have received further support from neuroimaging evidence indicating that older adults who show greater performance gains after instruction in an elaborative memory strategy show patterns of neural activation during word encoding that more closely resemble the activation patterns of younger adults (Jones et al., 2006; Nyberg et al., 2003).

Besides specific factors associated with memory functioning, differences in other aspects of cognition may also contribute to individual differences in the ability to benefit from training interventions. For example, performance on tests of perceptual speed and working memory predicts the degree to which individuals benefit from memory training programs (Kliegl, Smith, and Baltes, 1990; Verhaeghen and Marcoen, 1996), supporting the notion that individual differences are magnified by training (Baltes, 1987; Lövden et al., 2012). These findings are in line with the observation that successful aging is associated with higher performance across different cognitive tasks as well as with higher levels of education (Ghisletta et al., 2012; Habib, Nyberg, and Nilsson, 2007), presumably reflecting the ability to make flexible and efficient use of available brain resources, and to preserve structural and functional aspects of brain integrity into old age (Lindenberger, Burzynska, and Nagel, 2013; Nyberg et al., 2012).

Finally, recent studies suggest that heterogeneity in training benefits are related to genetic variation. For instance, the KIBRA gene has been associated with better EM for T-allele carriers in both younger and older adults (Schaper et al., 2008). In younger
adults, the positive memory effect for T-allele KIBRA carriers was additionally enhanced by presence of the CLSTN2 C-allele (Preuschhof et al., 2010), underscoring the need to examine interactions among multiple genes in order to understand their influence on complex cognitive functions (Lindenberger et al., 2008). As both these genes exert their influence primarily on MTL regions, future research should examine the degree to which variation in these genes is associated with benefits from memory intervention in childhood and old age. In working memory, variations in the DAT1 receptor gene were not related to performance prior to an adaptive training across four weeks, but DAT1 10-repeat carriers (characterized by less active dopaminergic pathways) demonstrated smaller training-related gains compared to DAT1 9/10-repeat carriers (Brehmer et al., 2009). These findings suggest that genetic effects on cognitive functioning may even be more pronounced in a training context rather than a single assessment.

Variation in dopamine modulation has also been related to individual differences in memory performance. For example, DAT1 and D2 receptor genes interactively influenced backward serial recall in younger and older adults (Li et al., 2013). In line with the resource modulation hypothesis (Lindenberger et al., 2008), the DAT1 and D2 genetic effects on recall were magnified in older adults, whose structural and neurochemical brain resources are compromised. Hence, genetic influence on training benefits may be crucially dependent on the available cognitive resources of the individual at the onset of the training program. Future research should determine whether differences in common genetic variation are related to distinct aspects of a training program depending on the brain networks that they are primarily targeting.

Overall, individual differences in the benefit from memory training have been more extensively investigated in aging research compared to child development. In general, training benefits seem to be positively associated with general cognitive resources. However, accumulating evidence suggests that genetic factors and factors specific to memory functioning need to be taken into consideration, as they may influence the degree to which different individuals benefit from an intervention.

**Conclusion**

In this chapter we outlined the main developmental trajectory for EM across the lifespan and noted that it is largely compatible with a two-component model of memory functioning, with strategic and associative memory components following distinct trajectories across the lifespan. We then outlined some of the potential modifiers of these trajectories that may contribute to heterogeneity of memory functioning in childhood and old age. To arrive at a more complete understanding of EM development from childhood to old age, we need to (1) isolate different components of EM and track their changes across the lifespan, (2) identify factors that may underlie individual differences in performance on these EM components, and (3) understand how the interactions between general and modifying factors change across the lifespan. The presented evidence demonstrates the utility of the two-component framework not only for examining mean differences among age groups, but also for generating predictions regarding mechanisms that drive individual differences in episodic memory.
References


