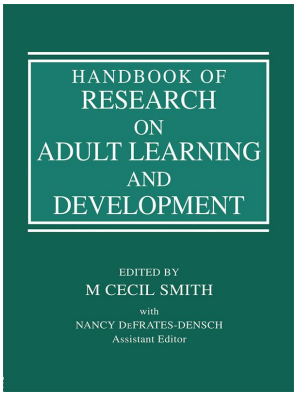

Handbook of Research on Adult Learning and Development

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What Do We Know About the Aging Brain?

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Chapter 24

What Do We Know About the Aging Brain?

Implications for Learning in Late Life

Benjamin T. Mast, Jennifer Zimmerman, and Sarah V. Rowe

The breadth of topics covered in this volume suggests that the intersection of adult development and learning is exceptionally complex and influenced by a wide variety of processes. This chapter focuses on how the brain changes in the context of adult development (with particular attention given to later life) and the ways in which these changes affect learning. To begin, we offer several general observations.

First, empirical investigation of direct links between age-related brain changes and the learning activities of older adults is largely uncharted territory. It has been noted that neuroscience and educational psychology have developed as separate fields with very little interaction (Blakemore & Frith, 2005). Much of the existing work that seeks to draw these fields together has focused largely on the first quarter of the developmental spectrum, with relatively little emphasis on later life. It is in this context that we approach the specific issue of how the changes observed in the aging brain affect learning. To accomplish this, we draw on evidence from the fields of cognitive aging, neurology/neuropathology, neuroscience, geriatric neuropsychology, and life-span developmental psychology. Empirical evidence is described where it is available and considerable attention is given to conceptual links in the absence of direct empirical evidence.

Second, the last decades of life are characterized by considerable heterogeneity; therefore when we consider the aging brain and learning, we must ask whether the discussion is focused on so-called "normal aging" or learning in the context of impaired brain functioning such as in Alzheimer's disease? This review focuses on both normal aging and dementia literatures because we believe that both offer important insights into the link between brain changes and late life learning, and because (as will be discussed) the boundary line between normal aging and certain forms of dementia such as Alzheimer's disease is not always clear.

Third, the learning activities of older adults rely on multiple cognitive skills which emerge from a complex interaction of multiple brain systems. Consider a 75-year-old woman who has decided to learn to use a computer so that she can communicate with her friends and family via email, research her genealogy, and write using basic word processing programs. She has several options to accomplish her goal of learning to use a computer. She could enroll in a formal computer literacy class offered at a local college. In this setting, the class will likely be structured for her (syllabus, lectures, textbook, and exercises designed by the instructor), but she will likely be required to focus her attention on the lectures while taking notes (drawing on working memory, inhibition of irrelevant stimuli, processing speed), as well as study and memorize terms and concepts for exams (sustained attention, inhibition of irrelevant stimuli, semantic and episodic memory). Alternatively, she may choose to learn to use a computer on her own outside of a classroom setting. This self-directed learning process will require her to develop a plan for learning the skills needed, finding and acquiring resources, carrying out the plan

and modifying as needed, and evaluating whether the learning was effective (Lamdin & Fugate, 1997). This form of learning is clearly less structured than formal classroom learning and places greater demands on the organizational, planning, and problem solving skills of the older learner. On the other hand, it may be advantageous in that there is less dependence upon processing speed and memory resources. Learning clearly relies upon a wide variety of cognitive skills and brain structures that interact with one another to produce successful learning outcomes, but some forms of learning may rely more heavily on specific cognitive skills than others. Furthermore, some of these skills are more affected by age and disease than others. Therefore, the various types of learning activities (e.g., formal learning vs. self-directed learning) may be differentially affected by age-related changes in cognition.

This chapter focuses on cognitive changes that are most notable among older adults and that appear to be most relevant to these two broad learning activities (formal learning and self-directed learning). The probability that learning will be disrupted by age and the diseases of aging is great because learning relies on a multitude of cognitive abilities and declines in any one component skill could have an impact on learning. On the other hand, to the extent that aging does not affect all cognitive abilities equally, spared cognitive abilities may enhance the opportunity for compensation (Baltes & Baltes, 1990; Marsiske, Lang, Baltes, & Baltes, 1995).

In the sections that follow, we first review common structural brain changes in the later decades of life as well as the most common cognitive changes observed in normal aging. As part of this discussion, we review results from selected cognitive intervention studies, using these results as evidence of learning potential among older adults (Baltes & Baltes, 1990; Willis, 1985). The second section includes a discussion of brain changes and their cognitive correlates in Alzheimer's disease (AD) and preclinical AD syndromes. We also review the results from a series of studies that used cognitive training paradigms from the normal cognitive aging literature to examine learning potential in dementia. Finally, using the Selective Optimization with Compensation model (SOC; Baltes & Baltes, 1990) as a broad framework, we describe how the brain and cognitive changes observed in the normal aging and dementia literature might affect the learning activities of older adults and how older adults might compensate for these changes. SOC provides a framework for understanding how cognitive changes lead elders to shift learning goals, methods, and contexts to maintain learning activities despite significant brain changes. Using this framework, we conclude the chapter by discussing how elders with dementia might achieve successful learning outcomes and thereby enhance functioning.

Normal Cognitive Aging

The last several decades have seen tremendous growth in research addressing the pattern of cognitive change observed in later life, particularly in the absence of dementia or other major neurological syndromes. Rigorous empirical efforts such as the Seattle Longitudinal Study (SLS; Schaie, 1994, 2005) have demonstrated that the long held assumption that aging is necessarily accompanied by declines in most, if not all, cognitive abilities is an oversimplification of the actual pattern of change in cognitive and intellectual abilities over time. Among SLS participants without dementia or other neurological conditions, results indicate that many basic intellectual abilities continue to improve or remain stable over the life span (Figure 24.1) until at least age 60, and that when abilities begin to show decline these are somewhat modest until the 80s (Schaie, 1994, 2005). Verbal memory had a slightly positive trajectory through age 60 after which

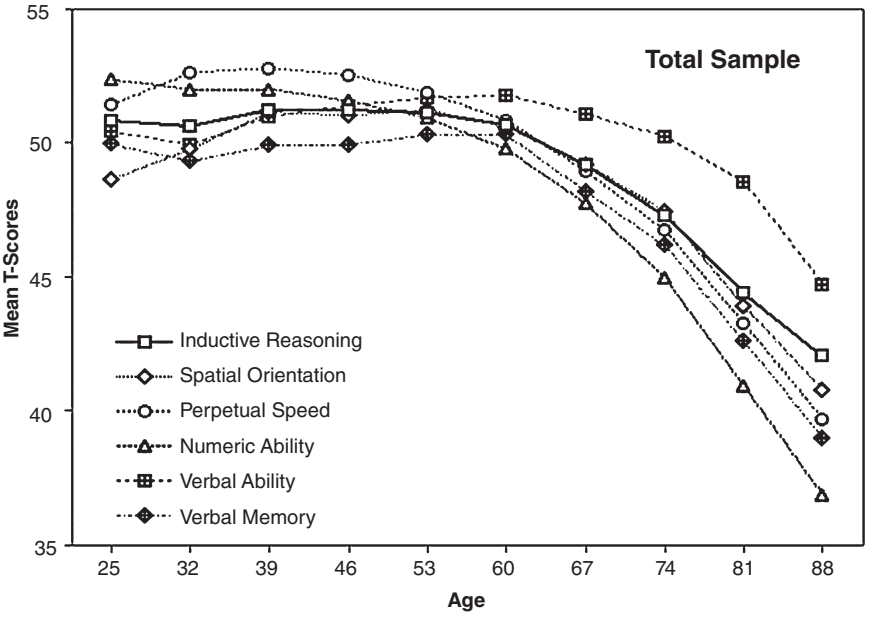

Figure 24.1 Estimated 7-year longitudinal changes in cognitive abilities (measured as latent variables) over the adult lifespan in the Seattle Longitudinal Study. *Source:* Schare (2005), *Developmental influences on adult intelligence: The Seattle Longitudinal Study.* New York: Oxford University Press. Reprinted with permision.

declines began and continued into the 80s. Verbal ability was relatively stable until the 80s. Numeric ability began declining earlier in the life span, but the greatest declines did not occur until the late 60s and 70s. Thus, the general pattern across abilities measured was one of improvement or relative stability in primary mental abilities until later in life (generally 70s and 80s), where declines have been consistently observed across abilities. One notable exception was processing speed, which does show fairly regular declines from middle adulthood until late life. This exception is critical because many of the abilities considered in cognitive aging research rely heavily on processing speed, such that once this variance is controlled the magnitude of age related changes in other abilities is significantly reduced (Salthouse, 1993, 1994; Schaie, 1994). Thus, although studies like the SLS demonstrate later life cognitive declines, some abilities demonstrate relatively greater decline, and some of these changes may be at least partly responsible for deficits observed in other cognitive abilities.

The inhibitory control deficit proposed by Hasher and Zacks "attributes age-related differences in memory and other cognitive functions to a decline in attentional inhibitory control over the contents of working memory" (Zacks, Hasher, & Li, 2000, p. 296). This hypothesis suggests that older adults have difficulty inhibiting stimuli that are not relevant to the task (e.g., thoughts or external stimuli such as noise), and lead to "mental clutter." Two implications of increased mental clutter include (1) less efficient encoding of information into long-term memory, and (2) slower and more error-prone retrieval of information from long-term memory (Zacks, Hasher, & Li, 2000). Thus, the inhibitory deficit model has implications for attention, working memory, and long-term memory encoding and retrieval.

The basic cognitive functions emphasized in the processing speed and inhibitory deficits hypotheses are highly relevant to the efficiency and methods of learning in later life.

As will be discussed later in this chapter, declines in processing speed and the inability to focus attention while inhibiting irrelevant information have both conceptual and empirical links to learning potential among older adults. However, we first turn our attention to the research concerning the underlying brain changes that occur in normal aging and the extent to which these are related to observed cognitive changes in later life.

Brain Changes in Normal Aging

Advances in neuroimaging technologies have allowed investigators to examine the underlying brain changes accompanying the aging process, and consider how they are linked to cognitive changes like those described above. Just as there is "no uniform pattern of age-related changes in adulthood across all intellectual abilities" (Schaie 1994, p. 306), there is similarly no uniform pattern of change across all brain regions. The normally aging brain does experience atrophy (or shrinkage) in general (i.e., whole brain volume) but the changes of aging are better characterized by differential volume loss in terms of gray vs. white matter volumes and within specific brain regions.

Raz (2005) notes that, despite variability across studies, the pattern of change observed in cross-sectional studies generally suggest that gray matter volumes show a negative gradient with increasing age, beginning early in life, and a leveling or plateau in later life. White matter changes, on the other hand, follow an inverted U function with increases in white matter volume into the early 20s, leveling through midlife until the 60s when declines begin and appear to accelerate with advancing age (see Raz, 2005, for review). Some studies reviewed by Raz suggest greater declines in frontal brain regions when compared to other brain regions. This is largely confirmed in his analysis of cross-sectional studies of normal age changes. Cross sectional results were presented as bivariate correlations between chronological age and regional brain volumes. Among cortical regions, prefrontal cortex (PFC) volumes show the strongest negative correlation with chronological age when compared with other brain structures. For example, frontal gray and white matter volumes show stronger correlations with age than do temporal, parietal or occipital cortex volumes. Not surprisingly, PFC changes have received considerable attention in normal aging reviews (West, 1996; Hedden & Gabrieli, 2004). However, although PFC volumes show the strongest cross-sectional correlation with age, it should also be noted that certain limbic structures, particularly the hippocampus, also show significant negative correlations with age in structural imaging studies of normal aging (Raz, 2005). Moreover, within the striatum, caudate and putamen volumes also show substantial negative correlations with age on a similar magnitude as the hippocampus (see Raz, 2005).

The reviews by Raz (2005; Raz & Rodrigue, 2006) also provided comprehensive summaries of median effect sizes based upon longitudinal studies of the normally aging brain (although there are fewer longitudinal studies). Areas of the brain that show relatively greater shrinkage over time include (in order): entorhinal cortex, hippocampus, caudate nucleus, and prefrontal cortex. These are important because these are regions of the brain that may be most critical for optimal learning in later life. Incidentally, these are also regions that are most affected by abnormal aging syndromes such as Alzheimer's disease (entrorhinal cortex and hippocampus) and cerebrovascular disease (prefrontal cortex and striatum) in late life, but the changes observed here are generally of lower magnitude than in pathological conditions (Campbell & Coffey, 2001; Jack, Shiung, Gunter, O'Brien, Weigand, Knopman, et al., 2004).

Small white matter lesions (or white matter hyperintensities; WMH) are a key

component of the white matter volumetric changes described above. WMH occur in greater frequency and severity in the aging brain, and frequently have vascular etiologies and are linked to cerebrovascular risk factors (Campbell & Coffey, 2001; O'Brien, 2006; O'Brien, Erkinjuntti, Reisberg, Roman, Sawada, Pantoni, et al., 2003; Roman, 2003). In fact, Raz notes that in empirical studies of normal aging, the extent of white matter changes is likely affected by inclusion or exclusion of individuals with cardiovascular disease and related risk factors because they contribute to white matter lesions (Raz, 2005; Raz & Rodrique, 2006). This raises the key issue concerning the nature and definition of normal aging because many cardiovascular risk factors, such as hypertension, increase in prevalence with age. Many authors have noted that WMH are ubiquitous in older brains, but historically their significance was not clear (Campbell & Coffey, 2001; Gunning-Dixon & Raz, 2000). WMH are more common in the brains of individuals with dementia and depression (Campbell et al., 2001), but also occur in individuals without dementia, depression, or clinically significant cognitive impairment (Gunning-Dixon et al., 2000).

Changes in Specific Cognitive Abilities Relevant to Learning

Thus far we have provided a brief overview of the structural brain changes observed in normal aging and selected cognitive changes that are prominent in later life. Yet, the most relevant issue for our purposes concerns how strongly these age-related changes affect learning in later life, which is dependent upon how strongly these brain changes are linked to the observed cognitive changes discussed in the broader aging literature (e.g., processing speed, memory, and inhibition). Ideally, it would be helpful to have direct empirical links between volumetric brain measurements and actual learning performance, or at minimum, longitudinal studies linking changes in brain volumes with performance changes on cognitive tests. However, recent reviews suggest that the evidence linking structural brain changes with cognitive change is not strong or consistent across studies (Raz & Rodrique, 2006). Although there are positive results linking regional brain volumes and cognitive functioning in normal aging, Raz and Rodrigue (2006) argue that because most studies are cross-sectional, and utilize diverse measurement approaches across studies, that "the true magnitude and even direction of association between regional volumes and cognitive functions in normal aging cannot be assessed at this stage" (p. 737).

The evidence is somewhat stronger for the link between WMH and cognition in late life. In a recent review, O'Brien (2006) suggested that although there are a variety of cerebrovascular changes that affect cognition, subcortical white matter changes may be the most common form of cerebrovascular/white matter pathology and is typically linked to changes in attention, processing speed, and executive functions thought to be mediated by the frontostraital circuitry. An earlier meta-analysis by Gunning-Dixon and Raz (2000) revealed a similar conclusion. Their analysis of twenty-three structural neuroimaging studies considered the relationship between WMH and cognitive functioning in elders without dementia or other cognitive impairment. Their findings suggest a significant relationship between WMH and global cognitive functioning, executive functioning, episodic memory (immediate and delayed recall) and processing speed. WMH's were not associated with measures of crystallized or fluid intelligence. Moreover, there was some evidence to suggest that WMH have relatively greater effects on executive functioning and processing speed. Lastly, each of these relationships was significant even after controlling for age (Gunning-Dixon & Raz, 2000).

As we consider the implications for learning in later life, several possibilities emerge from this study. First, measurable brain changes have a greater impact on cognitive changes independent of chronological age, suggesting that when considering learning in later life, individual difference variables such as white matter integrity may be more important than the age of the individual learner. Second, these individual differences in WMH were not significantly linked to general intelligence, but were linked to other cognitive functions including executive functioning, processing speed and recall of episodic memories. Relatively preserved intellectual functioning may enable older adults to continue learning new information, but changes in more vulnerable abilities may alter the setting and methods for continued learning. Individuals with changes in these abilities may learn more slowly, need more externally structured learning opportunities, and may choose to avoid formal learning settings which rely upon delayed recall of learned information (e.g., via exams).

Despite a limited yet developing empirical base bearing on the question at hand, there has been considerable conceptual synthesis of relevant literatures that can be used to make reasonable inferences about the links between normal aging brain changes and learning. In particular, West (1996) has integrated neuroimaging and cognitive aging literatures and concluded, consistent with later reviews by Raz (2005; Raz & Rodrique, 2006), that the prefrontal cortex (PFC) is the brain region that is most susceptible to aging in that it is the earliest and most severely affected in the context of normal aging. His proposed prefrontal cortex function theory of cognitive aging also incorporates more circumscribed cognitive aging literatures that focus on specific functions (e.g., sustained attention, inhibition, working memory) that are highly relevant to learning. Although the model was developed as a broad cognitive aging model, here we emphasize its potential application to learning in late life.

This model proposes the PFC has both primary and secondary cognitive functions. The primary function of the PFC "is to support the temporal organization of behavior through the formation and execution of temporal gestalts or complex behavioral sequences that are both novel to the organism and complex in nature" (West, 1996, p. 280). In other words, PFC is crucial for organizing and carrying out complex behavioral plans (e.g., new learning). The older learner described at the beginning of this chapter could accomplish her goal of learning to use a computer via a formal or self-directed learning (SDL) approach. Although both approaches require organizational and executive skills, the SDL approach may place greater demands upon the primary, organizational function of the PFC because it is less structured. For this learning project to be successful, the older learner would need to develop and carry out a plan of learning, whereas if she had opted for a formal learning experience, this organization may have been provided to a greater degree by an external source (e.g., the instructor).

West (1996) also delineates secondary functions of the PFC that support the primary organizational function: provisional memory, prospective memory, and interference control. Provisional memory is the process that recalls and holds relevant information in working memory while the behavioral plan is formed or, as West explains, "provisional memory serves to maintain task relevant information in an active state while the behavioral structure is constructed" (p. 280). Prospective memory builds upon provisional memory by working to initiate and execute the behavioral plan, or in some cases remembering to construct and execute this plan. Interference control is an inhibitory mechanism of the PFC that is similar to the concept highlighted by Zacks, Hasher, and Li (2000), which seeks to filter out irrelevant stimuli which could be external (competing noise or visual stimuli) or internal (thoughts or memories that are not task relevant).

Thus in our learning example, the older learner must be able to keep relevant information in working memory while developing and executing her learning plan (provisional memory), remembering to act upon the plan at appropriate times (prospective memory), and filtering out irrelevant information that reduces her ability to successfully engage in the learning plan (interference control).

Thus far we have observed (1) some limited empirical connection between structural brain changes and cognitive change in late life, and (2) a conceptual model that seeks to describe links between normal age related brain changes and cognition. In the section below, we examine the cognitive training literature as more direct evidence of the learning potential of older adults, and the extent to which the brain changes described above limit that learning potential. If the conceptual analysis above is correct, then (1) older adults should demonstrate lower learning potential than younger adults because of these brain changes (especially PFC), and (2) those older adults with evidence of age or disease induced cognitive deficits should demonstrate lower learning potential than those who do not.

Cognitive Training Research: Learning Potential and Limits in Normal Aging

In attempting to answer questions related to learning potential later in life, we discuss empirical literature related to cognitive interventions, also referred to as cognitive training studies. Here we take the perspective of Willis (1985) who argues that the short-term changes observed over a cognitive intervention trial have the "most direct and immediate implications for an educational/instructional psychology of the adult learner (p. 819)." In this view, cognitive training studies are well-controlled learning experiments that provide a unique perspective on the potential and limits of learning in later life, and the extent to which learning potential is affected by characteristics of the learner. Below we review selected cognitive training studies to highlight issues related to learning rather than seeking a comprehensive review of the cognitive training literature. Interested readers should consult Kramer & Willis (2002) and Salthouse (2006).

Cognitive intervention studies generally provide positive results concerning learning potential (often referred to as cognitive plasticity) among non-demented elders including those who have demonstrated modest declines in intellectual functioning (i.e., declines not sufficient to warrant a diagnosis of dementia). Elders in the SLS who demonstrated declines in specific abilities (e.g., inductive reasoning) over 14 years were selected to participate in one of two cognitive interventions. After receiving a pre-test assessment, they received cognitive training in the ability on which they had demonstrated decline. As described in Schaie (1994), "Approximately two thirds of our experimental subjects showed significant improvement, and about 40% of those who had declined significantly over the prior 14 years were returned to their pre-decline level" and "training was also somewhat more effective for those individuals who declined prior to the intervention" (p. 311). He goes on to conclude that the results from the SLS "suggest that observed ability declines in many community-dwelling older people are probably due to disuse and are consequently reversible, at least in part, for many persons" (p. 311).

On the other hand, older adults have been shown to improve their performance on cognitive tests after practice alone (i.e., repeated assessment without intervention; Rabbitt, Diggle, Smith, Holland, & McInnes, 2001), and therefore studies that compare specific interventions to control conditions which involve repeated assessment—but no intervention—are needed. In this regard, the most systematic evidence comes from the ACTIVE study, which is a large cognitive intervention study involving 2,832 relatively

healthy and cognitively intact adults aged 65 to 94 (Ball et al., 2002). Individuals were excluded if they demonstrated cognitive impairment (Mini-Mental State Exam (MMSE) <23), had experienced functional loss, had a self-reported diagnosis of AD, or had conditions that could lead to functional decline (e.g., stroke). Participants were randomly assigned to a control group (n = 704) or to intervention groups receiving memory training (n = 711), reasoning training (n = 705), or training in speed of processing (n = 712) over a 10 week training period. Immediately after training, roughly 87 percent of speed trained elders, 74 percent of reasoning trained elders, and 26 percent of memory trained elders showed reliable improvement (at least one standard error of measurement over baseline), and these improvements were largely retained over one- and two-year follow-up assessments. Those who received booster speed and reasoning training sessions 11 months after the initial training demonstrated even greater benefit but memory booster sessions did not have a significant effect. Similar results were obtained at five-year follow up. Training gains were maintained in all three abilities and booster sessions enhanced processing speed and reasoning, but not memory (Willis, Tennstadt, Marsiske, Ball, Elias, Koepke et al., 2006).

These results are consistent with the conclusions of prior reviews that although older adults experience cognitive changes in the context of normal aging, they clearly benefit from cognitive intervention programs (Kramer & Willis, 2002; Schaie, 1994). Interestingly, the ACTIVE study also supports the finding from the SLS that the magnitude of training gains roughly approximate the amount of decline experienced by nondemented elders over a 7 to 14 year period (Ball et al., 2002; Schaie, 1994).

In this context then, the question may be less concerned with the extent to which cognitive declines associated with normal aging contribute to learning problems in later life, but rather to what extent new learning might remediate the cognitive changes that many older adults experience. On the other hand, it would seem premature to suggest that the training literature indicates that all age-related cognitive changes can be reversed via training (Salthouse, 2006). Indeed, although findings from the SLS and ACTIVE studies indicate that the magnitude of training gain is similar to the magnitude of age related loss over the preceding periods, there is evidence from memory training studies that suggests younger adults derive greater benefit from training than do older adults (see Kliegl, Smith, & Baltes, 1989; Jones, Nyberg, Sandblom, Stigsdotter, Ingvar, Magnus et al., 2006; Singer, Lindenberger, & Baltes, 2003). This finding has led to the conclusion that although older adults benefit from training, they may demonstrate less cognitive plasticity or learning potential. Recent results from the Berlin Aging Study (BASE) concerning the plasticity of episodic memory among those aged 75 to 101 appear to support both of these points (Singer et al., 2003).

Ninety-six survivors from the ongoing BASE study were taught a mnemonic technique for improving episodic memory. Because this was part of an ongoing longitudinal study of cognitive aging, it was possible to track changes in cognitive abilities (memory, processing speed, verbal fluency, verbal knowledge) that had occurred in the six years prior to memory training. Change in memory performance in the six-year pre-intervention period predicted both pre- and post-intervention performance on tests of memory, but did not predict learning gains associated with the intervention. Interestingly, the best predictor of learning gain was processing speed, including change in processing speed in the six years prior to intervention. Older adults who had demonstrated greater declines in processing speed were less likely to benefit from memory training (i.e., show less new learning). The authors conclude that although memory plasticity was generally preserved in this old-old sample, it was significantly reduced, particularly among those

who had demonstrated declines in processing speed in the years preceding the intervention. These findings provide some evidence that the extent to which older adults maintain learning potential depends upon individual differences in their cognitive status, specifically whether they have been demonstrating greater cognitive decline.

Jones and colleagues (2006) build upon this theme in their discussion of the limits of cognitive and neural plasticity among older adults. They also discuss age-related reductions in new learning (plasticity) and seek to explain individual differences in plasticity or new learning using neuroimaging techniques. They review findings from their research group that examined neural correlates of learning gain using mnemonic training (Nyberg, Sandblom, Jones, Neely, Petersson, & Ing, 2003). Reductions in learning potential were observed in that younger adults demonstrated greater learning gain from the training procedure than did older adults. To explain this change using PET imaging, the older adults were divided into two groups: those who benefited from the training and those who did not. These two groups were compared to each other and to the younger participants to determine differences in regional brain activation. When using the mnemonic technique, younger adults demonstrated greater activation in the dorsolateral prefrontal cortex (DLPFC) than the older adults in general, which they interpreted as indicating that older adults had less overall processing resources. Both younger adults and older adults who benefited from training demonstrated increased activity in the left occipital/parietal region, whereas the older adults who did not benefit failed to demonstrate this pattern of activation. Importantly, follow-up questioning of participants regarding the mnemonic strategy indicated that older adults who failed to benefit from training understood the technique but had difficulty implementing it during the task, a result generally consistent with the predictions of the West PFC model of cognitive aging.

Further re-analysis of the Nyberg et al. (2003) data suggested that there were differences between the groups in medial temporal lobe (MTL) activation during use of the mnemonic strategy when compared to pre-training levels of activation. Young adults demonstrated greater bilateral MTL activation, and older adults, who benefited from training, demonstrated increased activity only in the left MTL. Older adults who did not benefit did not show increased MTL activation. Jones and colleagues concluded that there are age-related reductions in cognitive and neural plasticity (the basis for new learning) and that these "may reflect both a general processing (frontal) deficit, and a more task-specific utilization deficit (pariental, MTL)" (p. 870).

These findings suggest that there are limits in terms of new learning in late life, and that these limits may be associated with underlying brain changes. They also provide some support for the notion that PFC dysfunction and its related cognitive deficits may reduce learning potential in late life by limiting both processing resources (such as speed) or by interfering with one's ability to utilize obtained skills (executing behavioral plans). These findings also point to the importance of other brain structures that decline with age in predicting new learning. The finding that MTL activation was associated with training outcomes (learning potential) is particularly intriguing in that it is not only one of the structures most affected by aging (Raz, 2005), but it is also the region that is the earliest affected in Alzheimer's disease, which we turn our attention to in the next section.

Alzheimer's Disease and Dementia Syndromes

Alzheimer's disease (AD) is the most common cause of dementia in late life, and age is the strongest risk factor for AD. As such, any consideration of age-related changes in

the brain and their impact on learning must consider AD. Recent estimates suggest that approximately 4.5 million individuals in the United States have AD, and that by 2050 this number will grow to over 13 million with the greatest growth occurring among those over the age of 85 years (Hebert, Scherr, Bienias, Bennett, & Evans, 2003). Although the cause of AD is still unknown, the characteristic neuropathological changes (neuritic plaques and neurofibrillary tangles) have been well-described. These changes initially accumulate in the medial temporal lobe structures in the earliest stages of disease, but eventually spread to other cortical regions including parietal and frontal cortex. Because AD can only be confirmed by neuropathological evidence, the clinical diagnosis of AD must be considered either "possible" or "probable" prior to death (McKhann, Drachman, Folstein, Katzman, Price, & Stadlan, 1984).

Clinically, AD is characterized by progressive declines in cognitive functioning with particularly prominent changes in learning and episodic memory early in the disease process, and additional impairments in executive functioning, language, apraxia, and visual-spatial functioning as the disease progresses (Grober & Kawas, 1997; Stout, Bondi, Jernigan, Archibald, Delis, & Salmon, 1999; Welsh, Butters, Hughes, Mohs, & Heyman, 1991, 1992).

In this context of a progressive dementia, it is not particularly surprising that as more cognitive abilities are compromised by AD, learning will become considerably less efficient. Over the past two decades, considerable neuropsychological research has emphasized the inability to retain information over time as the core deficit of AD throughout the duration of the disease (i.e., episodic memory; Bondi, Salmon, Galasko, Thomas, & Thal, 1999; Welsh et al., 1991). The inability to retain newly learned information poses clear problems to late life learning, such that AD may largely prevent new explicit learning. Moreover, Grober and Kawas (1997) have demonstrated that, in addition to the commonly cited retention failures, individuals with AD also exhibit deficits in new learning, and that this learning deficit may precede the onset of other cognitive problems.

Grober and Kawas (1997) followed 537 non-demented elders from the Baltimore Longitudinal Study of Aging (BLSA) for up to seven years. Twenty participants developed clinically diagnosed AD and were matched with control participants (those who did not develop dementia) based on age and gender. Total learning and retention on the Free and Cued Selective Reminding (FCSR) test were taken both at baseline (when no participants had clinical AD) and at three-year follow-up (when the AD participants were in a mild AD stage). At the baseline assessment, those with preclinical AD demonstrated poorer learning over multiple trials but had similar retention performance over a 30 minute delay when compared with controls (see Figure 24.2). These findings suggest that learning is impaired in AD even before the onset of other cognitive problems including retention which is typically thought to be the earliest symptom of AD (e.g., Bondi et al., 1999). Furthermore, at the three-year follow-up, AD patients continued to show declines in both learning ability and retention of information learned, while controls had stable performance in both learning and retention.

Not surprisingly, these studies have been taken as evidence that AD leads to profound disruption in the learning process. Margaret Baltes and her colleagues (1992, 1995, 1996) took this perspective one step further to suggest that reduced learning potential is the core deficit in dementia, particularly AD. A series of studies by M. Baltes and her colleagues provide some of the most compelling evidence concerning the link between AD and reduced learning potential.

Baltes, Kühl, and Sowarka (1992) studied 81 community dwelling elders in Berlin, Germany, of whom 25 were determined to be at risk for dementia based upon a standardized

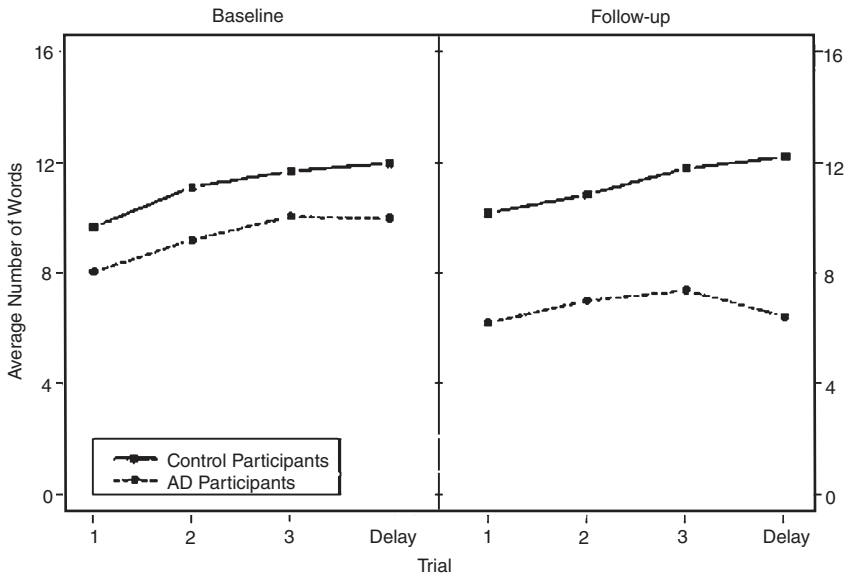

Figure 24.2 Learning and recall over trials baseline and at 3 year follow-up in participants with and without Alzheimer's disease (AD). *Source:* Grober and Kawas (1997). Learning and retention in preclinical and early Alzheimers disease. *Psychology and Aging, 12*, 183–199. Reprinted with permission of the American Psychological Association.

psychiatric interview conducted by a psychiatrist specializing in dementia. Twenty-one of these were determined to have early Alzheimer's type dementia syndromes. All participated in a standardized cognitive training protocol aimed at improving figural relations (fluid intelligence). They hypothesized that elders with early dementia would demonstrate less benefit from the cognitive training (i.e., less pre-post training change) than normal controls. Pre-test figural relations scores did not differentiate controls from those with early dementia and did not predict individual differences in training gain. Individuals with dementia (or at-risk) did not demonstrate statistically significant training gains, whereas controls did. In short, "only the trained subjects identified as healthy significantly improved their scores from pretest to posttest" (Baltes et al., 1992, p. 166). These findings were interpreted as indicating that elders with early AD do not have sufficient learning potential (or reserve capacity) to benefit from cognitive training procedures.

In a follow-up study, M. Baltes, Kühl, Gutzmann, and Sowarka (1995) sought to replicate these findings and extend them to consider (1) whether similar results could be obtained using other fluid intelligence tests (inductive reasoning) and (2) whether the relative difference in training gains would better differentiate between demented and control elders than standard psychometric tests used in AD assessments (Word List and Digit Span memory tests from the Nurenberg Age Inventory). Of 108 participants trained in either figural relations or inductive reasoning, 59 had scores in the mild impairment range or worse on the Structured Interview for the Diagnosis of Alzheimer or Multi-Infarct Dementia (SIDAM). In multiple regression analyses, individual differences in training gains and free recall on the Word List assessment measure were the strongest predictors of dementia status. Figural relations training gains explained the greatest portion of variance in SIDAM scores, followed by free recall of the Word List, and inductive reasoning training gains, respectively. These findings confirm the findings of Baltes

and colleagues (1992) that learning potential (cognitive reserve) was a strong, unique predictor of dementia, even after controlling for performance on other measures of memory. Moreover, this effect generalized to different training protocols addressing different fluid abilities (inductive reasoning and figural relations).

M. Baltes and Raykov (1996) followed this study with a longitudinal extension to determine whether the training gains would predict dementia status two years after the initial post-test. Structural equation model results indicated that training gains in figural relations predicted dementia status at the two-year follow-up after controlling for the impact of pre-test dementia status and pre-test figural relations performance. It was determined that the differential training gains (figural relations post-test) was as strong a predictor of two year dementia status (two year SIDAM score) as was pre-test dementia status (i.e., pre-training SIDAM score). They concluded:

> The findings indicate that learning gain, namely posttest scores, predict later mental status even when Time 1 mental status and pretest are controlled for. In fact, we found no difference between the predictive power of posttest scores and initial mental status. (p. 555)

Recently, Fernández-Ballesteros and colleagues (2005) created a clinical assessment battery for dementia that builds upon the work of Baltes and colleagues. The Battery of Learning Potential for Assessing Dementia (BEPAD) involves pretesting on four cognitive domains (visual-spatial, verbal recall, executive functioning, and verbal fluency), followed by standardized training and a post-test in each domain. In one study, the learning potential (training gain) scores were better able to differentiate individuals with AD, Mild Cognitive Impairment (MCI), and controls than did pre-test scores. Overall, 89% of participants were correctly classified using learning potential scores.

These studies indicate that those who are in the early stages of dementia or who are at high risk for dementia show significantly less learning (training gains) even after being taught specific techniques for improving their performance. As a result, Baltes and colleagues suggest that reduced learning potential is a better marker for AD than standard psychometric tests given as part of a one-time static neuropsychological assessment. Baltes and Raykov (1996) suggest the reason for this is that the underlying brain pathology of AD affects cognitive reserve or learning potential.

Neuroimaging and clinico-pathology studies provide evidence concerning why this might occur by identifying the earliest and most notable changes in the brains of individuals who have probable AD. In the sections below, we review selected studies that address links between the AD brain changes and their corresponding cognitive change with the dual goal of (1) identifying the locus of reduced learning efficiency in elders with AD and (2) understanding the extent to which AD related brain changes might affect the learning of older adults who are not yet experiencing clinical AD. As will be seen, the links between underlying AD and reduced learning potential may be highly relevant to understanding learning among a portion of non-demented elders.

For example, one analysis from the Nun Study included both neuropathological and common imaging indicators of AD (Mortimer, Gosche, Riley, Markesbery, & Snowdon, 2004). Using postmortem MRI and autopsy results, the Nun Study investigators found that delayed recall of a word list was associated with temporal lobe volume, and the number of neurofibrillary tangles (NFT) in CA-1 and subiculum. Individuals with a greater number of NFTs and smaller temporal lobe volumes recalled fewer words after a short delay. Hippocampal volume was a better predictor of learning and recall than autopsy

indicators of AD pathology. But, perhaps most importantly for our current purposes, hippocampal volume was associated with poorer recall of learned information in *both* demented and non-demented research participants. It has long been hypothesized that AD changes are present in the brain long before cognitive impairment appears or the clinical expression of AD occurs, and as a result, there are many individuals who have AD pathology but have not yet demonstrated cognitive changes sufficient to draw clinical attention (Collie & Maruff, 2000).

The findings of Mortimer and colleagues (2004) suggest that these underlying changes may have cognitive and functional consequences, even when the individual does not meet criteria for a dementia syndrome and provide a compelling rationale for considering the impact of early AD brain changes on learning in individuals who do not have dementia or significant cognitive impairment. The Baltes intervention studies suggest that AD reduces learning potential. Therefore, a key question is whether the underlying brain changes observed by Mortimer et al. also lead to subtle changes in cognition that affect learning potential in non-demented elders? In the next section, we consider this question among those elders who fall in the gray area between the normally aging elders considered in cognitive aging studies and the clinical cases considered in research on neuropsychological aspects of AD.

MCI and Preclinical AD Changes in Cognitively Intact Elders

Cognitive changes that fall short of dementia are common in late life (Panza, D'Intiono, Colacicco, Capurso, Del, Caselli, et al., 2005; Unverzagt, Gao, Baivewu, Ogureuvi, Gureje, Perkins, et al., 2001) and have been linked with AD pathology (Markesbery, Schmitt, Kryscio, Davis, Smith, & Wekstein, 2006). These changes appear to be very important for understanding links between the aging brain and learning in late life. Of particular interest are the earliest detectable brain and cognitive changes in AD, and in some cases, changes that precede the development of AD. Emphasis on Mild Cognitive Impairment (MCI) syndrome as a possible transition between normal aging and AD has grown in recent years with the hope that identifying a clinical state the reliably precedes the development of AD will enhance treatment and prevention outcomes (Petersen, Doody, Kurz, Mohs, Morris, Rabins et al., 2001).

MCI can likely take a number of forms, but the amnestic MCI type has received the most research to date. This syndrome occurs in non-demented individuals and is characterized by an isolated impairment in memory accompanied by subjective memory complaints in the context of otherwise normal cognitive functioning (i.e., no other cognitive decline) and no impairment in activities of daily living (Petersen et al., 2001). Some investigators have concluded that amnestic MCI represents very early AD based upon high conversion rates to clinical AD in comparison to individuals who were cognitively normal at baseline (Morris, Storandt, Miller, McKeel, Price, Rubin, et al., 2001). For example, Morris and colleagues (2001) observed that 100% of participants with MCI (CDR score of 0.5) progressed toward greater dementia severity over the study period (up to 9.5 years). At five years, only 6.8% of cognitively normal elders had developed dementia (CDR = 1). Moreover, of the 25 autopsied MCI cases, 24 demonstrated AD neuropathology.

Recent studies confirm the hypothesis that the amnestic form of MCI is characterized by AD pathology on autopsy and likely represents early AD. Bennett and colleagues (2005) followed 180 Catholic clergy with annual clinical evaluations until death and brain autopsy. Thirty-seven had MCI, 60 had no cognitive impairment, and the remain-

ing 83 had dementia at the last clinical evaluation (the average lag between the last clinical evaluation and autopsy was 6–7 months across groups). More than half of the MCI participants met pathological criteria for probable or definite AD using CERAD criteria. The MCI group fell between the normal control and AD groups in terms of severity of AD pathology and in the frequency of cerebral infarction (Bennett, Schneider, Bienias, Evans, & Wilson, 2005).

Markesbury and colleagues (2006) compared 10 amnestic MCI patients with 10 early AD and 10 controls on the neuropathological features of AD: neuritic plaques and neurofibrillary tangles. Participants had been followed longitudinally through the transition from normal cognition to MCI and early AD, and had an average period of roughly nine months between their last clinical evaluation and death across all three groups. Patients with amnestic MCI demonstrated greater AD pathology than normal controls including greater neuritic plaques in the frontal, temporal and parietal cortices, posterior cingulate gyrus, and amygdala, and greater neurofibrillary tangles (NFT) than controls in the parietal lobe, amygdala, entorhinal cortex, CA1, and subiculum. Amnestic MCI patients were similar to early AD patients in terms of the number of neuritic plaques in the neocortical regions, CA1, and entorhinal cortex, but had fewer neuritic plaques in the amygdala and subiculum. Early AD patients had greater neurofibrillary tangles than MCI patients in the frontal, temporal, amygdala and subiculum, but were similar in terms of tangle counts in other regions such as CA1, entorhinal cortex, the parietal lobe and posterior cingulate gyrus. Moreover, delayed recall of a word list was correlated with several neuropathologic indicators of AD. Greater NFT counts in the entorhinal cortex and CA1 region were associated with poorer recall of the word list after a short delay. These findings suggest that amnestic MCI patients demonstrate early AD pathology and that this pathology may interfere with cognitive functions that are key to optimal learning (i.e., poorer retention).

Longitudinal studies of amnestic MCI suggest that these individuals will progress toward AD at a much higher rate than non-MCI elders (Morris et al., 2001) and, by implication, will experience increasing difficulty with the learning process as memory impairment worsens and other cognitive functions are compromised. As has been suggested by the clinico-pathology correlation studies above, these core deficits appear to be linked predominantly to AD pathology, which is thought to be linked to reduced learning potential in those persons with early AD (Baltes et al., 1996; Baltes, Kuhl, Gutzmann, & Sowarka, 1995; Baltes, Kuhl, & Sowarka, 1992; Raykov, Baltes, Neher, & Sowarka, 2002). Below we consider more direct evidence that patients with MCI demonstrate reductions in learning potential (Darby, Maruff, Collie, & McStephen, 2002; Schrijnemaekers, de Jager, Hogervorst, & Budge, 2006).

Schrijnemaekers et al. (2006) examined the verbal learning and recall performance of elders with AD, to those with MCI, and to normal controls. On repeated testing over a two year test interval, those with AD demonstrated mild decline, and normal controls demonstrated improvement on the Hopkins Verbal Learning Test (HVLT). Elders with MCI were less likely than normal elders to demonstrate improvement on the HVLT, consistent with the hypothesis that MCI (in addition to dementia) is associated with reduced learning potential. In this study, this reduction was partially attributable to lack of strategy use at follow-up testing. The cognitively intact controls appeared to utilize a semantic clustering learning strategy (likely learned on the previous testing) and used this to their advantage in learning and recalling more words over time, whereas elders with MCI were less likely to recall and incorporate this learning strategy at the two-year follow-up, which led to recall of fewer words.

This result is consistent with earlier results from Darby et al. (2002) who found that MCI patients were less likely than matched normal elders to demonstrate practice effects over a three hour test battery on tests of reaction time and working memory. In their protocol, both MCI and normal cognition elders repeated very short, simple cognitive tasks over four occasions within three hours. On initial testing, the MCI patients were not significantly different from normal controls, but these two groups did differ in terms of their changes in performance over the next three testing occasions. The controls clearly demonstrated learning via improved reaction time on a simple reaction time task, and greater speed and accuracy on a simple working memory task (one-back test). MCI patients, on the other hand, demonstrated relatively flat performance over trials suggesting an absence of new learning.

Taken together, the literatures above suggest that amnestic MCI and AD pathology are linked to problems in learning via two core cognitive deficits. The first is reduction in learning capacity, as demonstrated by poorer performance on list learning memory tasks and by failure to benefit from repeated exposure of items to be learned (or a practice effect). Second, failure to employ strategies to optimize learning over trials or repeated exposures may contribute to poorer learning outcomes, which is consistent with predictions made by West's PFC theory. These conclusions are particularly intriguing in light of the training results discussed above in the context of normal aging indicating that learning potential may be reduced in normal aging, and that this reduction may be linked to dysfunction within the MTL and PFC (Nyberg et al., 2003; Singer et al., 2003), which are the same regions which appear to experience greatest volume losses in normally aging longitudinal samples (Raz, 2005; Raz & Rodrigue, 2006; West, 1996).

Recognizing that AD and its pathology may lie on a continuum both in terms of the underlying severity of brain changes and corresponding cognitive changes, in the section below we turn our attention to the question of whether early, preclinical AD pathology interferes with cognitive functioning and learning in those who have not yet demonstrated cognitive impairment. The notion of preclinical AD is based upon the hypothesis that AD changes are present in the brain long before cognitive impairment or the clinical expression of the underlying AD pathology appears. Individuals with preclinical AD are believed to have underlying AD pathology but have not yet demonstrated changes sufficient to draw clinical attention. In the context of the literature concerning the link between AD pathology and reduced learning potential, a key issue is whether the preclinical AD involves subtle changes in cognition that affect learning potential.

Longitudinal clinical studies indicate that individuals at risk for AD demonstrate isolated weakness in multiple areas of cognitive functioning, particularly delayed recall (explicit memory), executive functioning and processing speed. For example, Bäckman and colleagues (2005) performed a meta-analysis on 47 longitudinal studies which examined preclinical AD in clinically non-demented elders. Those who were free of clinical dementia at baseline but went on to receive a diagnosis of AD at follow-up were considered cases of preclinical AD (n = 1207) and were compared with those who did not receive a subsequent diagnosis of dementia at follow-up (normal controls, n = 9097). Those with preclinical AD demonstrated significantly poorer (effect sizes > 1.0) global cognitive functioning, processing speed, executive functioning, and episodic memory (particularly delayed recall). Verbal ability, visual-spatial functioning, and attention were also affected in preclinical AD, but to a lesser extent (Backman, Jones, Berger, Laukka, & Small, 2005).

Clinicopathological studies also appear to support the notion of preclinical AD and appear to be highly relevant to learning in late life. A recent study (Bennett, Schneider,

Arvanitakis, Kelly, Aggarwal, Shah, et al., 2006) examined 134 older adults without cognitive impairment. They observed that, although participants in this study were cognitive normal (i.e., no impairment), those who had pathology consistent with AD demonstrated subtle yet significantly poorer episodic memory than elders without significant AD pathology. An earlier study by Schmitt and colleagues (2000) yielded similar findings in that non-demented elders who had AD pathology on autopsy demonstrated poorer immediate paragraph recall and delayed recall of a word list at their last clinical evaluation when compared to those who did not have AD pathology on autopsy (Schmitt et al., 2000). Both studies suggest that preclinical AD may interfere with memory functions that contribute to optimal learning.

Galvin, Powlishta, Wilkins, McKeel, Xiong, Grant, et al. (2005) studied 41 community dwelling elders without clinical evidence of dementia prior to death (i.e., normal cognitive functioning over repeated longitudinal neuropsychological assessments) and found that 34% had pathological evidence of AD on autopsy. These elders generally did not demonstrate cognitive differences from those without AD pathology, but they did demonstrate a subtle yet detectable difference over repeated assessments. Because the cognitive testing was repeated on an annual basis, most participants received the same tests multiple times (mean number of assessments per participant = 6), and the elders without AD pathology demonstrated the typical practice effect on these tests (i.e., their performance improved over time). However, the elders with AD pathology demonstrated significantly less improvement over time on a test of learning paired associated and demonstrated no improvement on a test of object naming. At one level of explanation, this is yet another cognitive difference between two groups of non-demented elders—those who have autopsy evidence of AD (preclinical AD) and those who do not demonstrate AD. At another level, this finding suggests the possibility that the absence (or reduction) of new learning over time is associated with underlying AD pathology that has not been clinically expressed (preclinical AD; Galvin et al., 2005). This lends further support to Baltes' hypothesis that learning potential is a sensitive marker for dementia and underlying dementia risk (Baltes et al., 1992) and extends this hypothesis to elders who are not yet expressing clinical dementia.

In summary, AD clearly interferes with new learning, and indeed may be primarily a disorder characterized by a deficit in learning. In addition, other clinical states that typically precede AD, such as MCI, also appear to affect learning. Furthermore, a portion of older adults who do not demonstrate cognitive impairment, may nonetheless have underlying AD pathology which is associated with poorer, yet unimpaired performance in episodic memory, executive functioning, and processing speed.[1] There is emerging evidence that these changes interfere with learning over time (Galvin et al., 2005). These findings are consistent with the cognitive intervention studies described above in which both cognitive (processing speed) and neuroimaging indicators (medial temporal and prefrontal cortex activation) were significantly associated with reduced ability to benefit from cognitive training (memory).

Across normal aging and AD syndromes there are brain changes, particularly in the MTL and PFC regions, that contribute to cognitive changes associated with reduced learning potential. This conclusion is consistent with prior reviews (e.g., Hedden et al., 2004) that suggest that PFC changes (including WMH) may be more influential in normal age related cognitive change, and that the medial temporal lobe (MTL) structures are more affected and influential in early AD, although there is considerable overlap between these changes (de Leeuw, Barkhof, & Scheltens, 2004, 2006) and the boundary lines in this context are unclear.

The PFC changes described above may interfere with the efficiency of learning activities by reducing the ability to organize behavior, selectively focus attention, filter irrelevant stimuli, and process information quickly. On the other hand, the underlying AD changes in the MTL also interfere with long-term storage of information that is to be learned, and this deficit is superimposed upon the organizational changes associated with PFC dysfunction. Thus elders with dementia may have both the normal age related reductions in PFC functioning combined with MTL deficits, and therefore may fail to benefit from the learning activities described in cognitive intervention studies.

Thus far, we have highlighted cognitive changes and their links to learning. In the section below, we address social and emotional changes associated with PFC dysfunction that may interfere with learning in late life: depression, apathy, and theory of mind. We include these here because these problems have been linked to PFC dysfunction and because the research literature indicates that learning activities fulfill not only cognitive but also social and emotional needs as individuals grow older (Willis, 1985).

Depression and Apathy

Although depression and apathy are not a part of the normal aging process, they are associated with common brain changes observed among older adults. Significant white matter lesions, particularly within the frontostriatal circuitry are relatively common among older adults (Alexopoulos, 2002; Alexopoulos et al., 1997a; Alexopoulos et al., 1997b; Campbell et al., 2001). The frequent vascular etiology for lesions within the frontal-subcortical circuitry has led to the vascular depression hypothesis which suggests that vascular diseases increase risk for late life depression via small vessel disease in these regions of the brain (Alexopoulos et al., 1997a; Lyness et al., 1998). Empirical studies support the link between depression and vascular lesions within the frontal-subcortical regions (Kales, Maixner, & Mellow, 2005; Krishnan, Hays, & Blazer, 1997; Krishnan, Taylor, McQuoid, Macfall, Payne, Prvenzale, et al., 2004; Steffens, Helms, Krishnan, & Burke, 1999; Steffens, Krishnan, Crump, & Burke, 2002) and between depression and cerebrovascular risk factors (Lyness, King, Conwell, Cox, & Caine, 2000; Mast, MacNeill, & Lichtenberg, 2004; Mast, Neufeld, MacNeill, & Lichtenberg, 2004). Other consequences of frontal lobe disruption, such as executive dysfunction, have been emphasized in geriatric depression as well (Alexopoulos, 2003; Alexopoulos, Kiosses, Klimstra, Kalayam, & Bruce, 2002; Alexopoulos, Raue, & Arean, 2003; Mast, Yochim, MacNeill, & Lichtenberg, 2004). The presentation of vascular depression and depression with executive dysfunction syndromes have emphasized apathy, loss of interest in activities, loss of initiative and social withdrawal (Alexopoulos et al., 2002; Krishnan et al., 2004; Mast, 2004).

The relevance of these depression syndromes to learning in late life is highlighted not only by their association with cognitive inefficiency in key domains such as memory, executive dysfunction and processing speed (Alexopoulos, 2003; Lichtenberg, Ross, Millis, & Manning, 1995; Massman, Delis, Butters, Dupont, & Gillin, 1992; Nebes et al., 2000), but also in their effects on initiative and social activity. The presence of such syndromes would reduce the probability that those affected would engage in new learning project, and if initiated, they would experience greater difficulty due to cognitive dysfunction associated with depression. Thus, depression is one individual difference variable that could affect motivational and emotional aspects of learning in late life. Below, we discuss an emerging construct that could interfere with social aspects of learning.

Theory of Mind

Theory of Mind (ToM) is a relatively new social cognitive construct which describes "the ability to attribute independent mental states to other people to explain and predict their behavior" (Keightley, Winocur, Burianova, Hongwanishkul, & Grady, 2006, p. 559). ToM abilities enable individuals to infer or understand what others are thinking based on nonverbal cues or indirect information (Frith & Frith, 1999) ToM appears to be distinct from other social cognitive constructs, such as personality and emotion processing, and has been used along with other measures of social cognition to predict social functioning (Keightley et al., 2006; Phillips, MacLean, Allen, et al., 2002; Washburn, Sands, Walton, et al., 2003). Thus, ToM appears to be a very important and unique element in social behavior across the life span.

Intact ToM abilities may aid learning across the life span in both formal and self-directed learning settings. Formal learning settings, while characterized by the direct verbal exchange of information, still contain many elements that require the correct interpretation of social nuances: assignments that must be carried out by working with others in groups, the nonliteral interpretation of an instructor's dry wit or tongue in cheek presentation of material, and the appropriate timing of a student comment or question. In self-directed settings, ToM abilities may become even more important, as the expertise and assistance of others (librarians, bookstore clerks, friends, and spouses) become primary resources. In the self-directed learning situation, then, the learner's progress may depend directly on his or her ability to carry out basic social exchanges.

The majority of research on ToM has focused on the relatively late emergence of ToM in childhood (approximately age 3), and its developmental relationship to language, inhibition, and executive function. More recently, researchers have begun exploring ToM in older adults, seeking to understand whether ToM abilities are maintained throughout the life span, increase through the acquisition of wisdom, or decrease through age-related cognitive decline. Studies of age-related differences in ToM in later life have had mixed results with some suggesting age-related improvement in performance on some ToM tasks, other suggesting no changes, and still others demonstrating age-related declines (German & Hehman, 2006; Happe, Winner, & Brownell, 1998; Keightley et al., 2006; Maylor, Moulson, Muncer, & Taylor, 2002; Phillips et al., 2002; Sullivan & Ruffman, 2004). More studies, both cross-sectional and longitudinal, are needed that utilize a breadth of ToM measures in order to fully understand its developmental course at the end of the life span in healthy adults.

It has been suggested that ToM abilities may rely on distinct neuroanatomical circuitry (Baird et al., 2006; Rowe, Bullock, Polkey, & Morris, 2001; Stuss, Gallup, Jr., & Alexander, 2001). Evidence from fMRI studies demonstrate increases in blood flow during ToM tasks in several regions of interest including the medial prefrontal cortex, both temporal poles, anterior superior temporal sulcus, and from the bilateral temporo-parietal junction to the posterial superior temporal sulcus (Gallagher, Happe, Brunswick, Fletcher, Frith, & Frith, 2000; Saxe & Kanwisher, 2003; Saxe, Carey, & Kanwisher, 2004). Some diseases associated with aging that disrupt these brain regions have demonstrated differential effects on ToM abilities. On simple ToM tasks that do not require working memory, patients with Alzheimer's disease (AD) tend to score similarly to healthy controls. However, ToM tasks that rely heavily on working memory do reveal significant differences between elders with AD and healthy controls, although these differences may be attributed to increased demands on working memory and not deficits in ToM in AD patients, as the deficits are seen in both control conditions and ToM conditions (Zaitchik, Koff,

Brownell, Winner, & Albert, 2004, 2006). ToM, like other cognitive domains involved with learning, has been linked to the prefrontal cortex. ToM deficits have also been observed in non-demented Parkinson's patients (Mengelberg & Siegert, 2003; Saltzman, Strauss, Hunter, & Archibald, 2000) and in patients with frontotemporal dementia (FTD; Gregory et al., 2002). Diseases associated with age, then, may produce deficits in ToM abilities in older adults that can be expected to compound increasing problems with learning. These deficits may, in turn, lead to increases in social isolation due to the decreased ability to understand and respond appropriately to others.

ToM has been studied in relation to domains that have demonstrated key influences on learning, such as fluid intelligence, working memory, and executive functioning. Some studies have demonstrated that ToM is significantly associated with fluid intelligence (Maylor et al., 2002; Sullivan & Ruffman, 2004), while others have not (Phillips et al., 2002). Many ToM tasks, particularly second-order false belief tasks, have demonstrated dependence on working memory, a finding which has major implications for late-life learning. Researchers have sought to mitigate the confound of working memory in ToM studies by allowing older adult subjects to refer back to scenarios as often as needed before answering (Maylor et al., 2002) and by employing tasks that require less storage of verbal information (Washburn et al., 2003; Zaitchik, Koff, Brownell, Winner, & Albert, 2004). Similar modifications in formal learning settings may increase their appeal for the older learner. Maylor et al. (2002) have also provided evidence that ToM abilities are independent of executive functioning as associations between ToM and executive functioning disappeared when age was partialled out. Therefore, when considered in conjunction with cognitive constructs associated with successful learning toward the end of the life span, it appears that ToM may uniquely contribute to the older adult's learning process, particularly social aspects of common learning activities. PFC changes may impact learning in late life via both the cognitive abilities delineated by West and others, but also via non-cognitive changes reflecting mood, motivation and social functioning.

Observations about General Learning Trends in Later Life

We have identified a range of neuropsychological changes that likely affect learning in late life and in this final section we consider the more direct ways in which these changes influence the learning activities of older adults. A comprehensive survey of the educational gerontology and lifelong learning literatures is beyond the scope of this chapter. However, several general themes have been observed that we wish to highlight here in the context of considering the impact of neuropsychological changes associated with aging and age-related disease. These general observations described below can be understood in the context of the Selective Optimization with Compensation (SOC) model (Baltes & Baltes, 1990) which will be explained in greater detail below. In short, changes in memory, processing speed and executive functioning may force elders to select a smaller number of learning projects, optimize the use of existing strengths and expertise, and incorporate compensatory techniques and environmental support in order to continue learning effectively. It is our view that the basic principles of the SOC model contribute to the following three observations about learning in later life.

First, participation in formal adult education appears to decline with increasing age. Data from the most recent Adult Education and Lifelong Learning Survey of the 2001 National Household Education Surveys Program indicate that 22% of respondents over the age of 65 participated in formal adult education classes, as compared with 53%–55% of individuals between the ages of 16 and 55 (Kim, Hagedorn, Williamson, & Chapman,

2004). Among older survey respondents, participation in formal learning took the form of work-related classes or personal interest courses, rather than formal university or college degree programs (Kim et al., 2004). Yet, despite this apparent decline, the broader gerontological literature suggests that most older adults continue with some type of learning activities (Tough & Ontario Institute for Studies in Education, 1999). Although learning potential may decline, this is not sufficient to prevent new learning in most cases. Empirical studies of learning activities of older adults and the results of cognitive intervention studies (e.g., Ball, Berch, Helmers, Jobe, Levech, Marissle, et al., 2002; Willis et al., 2006) clearly dispute this claim. As we will discuss below, there is some evidence that even in the case of advancing AD, which involves a clear deficit in learning potential (Baltes et al., 1992, 1995, 1996; Raykov et al., 2002), some new (albeit limited) learning remains possible. The key to successful learning in the context of cognitive decline is to shift methods of learning appropriately to compensate for lost skills and optimize remaining functions (Baltes & Baltes, 1990).

Second, the learning activities of many elders differ from those of younger adults. Several authors have reported a shift away from formal learning settings (e.g., college and university courses) toward informal self-directed learning (SDL; Lamdin et al., 1997; Tough, 1979; Willis, 1985). SDL has been described as "independent learning projects," that are "self-initiated and self-designed" and "constitute a direct response to the learner's own interests, needs, and life style choices" (Lamdin et al., 1997). Tough reports that 90% of adults engage in some form of learning (at least one major learning project per year) and that up to 80% of learning efforts are SDL projects (Lamdin et al., 1997; Tough, 1979, 1999). These projects typically involve several stages, including generating the idea for a learning project, developing a plan for learning, conducting a search for resources, and carrying out the actual learning process (Lamdin et al., 1997). The core of SDL is that the planning, process, and evaluation of learning are taken on by the learner, whereas in most formal learning opportunities the learning objectives, methods, and evaluation are already structured and planned by an instructor.

Both normal age-related and pathological changes in the brain will likely alter the goals, methods and setting of new learning. Thus, although learning potential may be reduced in later life, many older adults compensate by changing the types of learning they engage in, the learning goals targeted and the methods used to achieve their specific learning goals. In a report to a U.S. Department of Education conference on lifelong learning, one scholar noted:

> In summarizing his research on adult learners, Penland (1978, p. 6) noted that adults 'often feel a strong need to establish the pace and control the character of their learning experiences.' Most adults express a preference for independent learning over formalized courses for a number of reasons: pacing, learning style, flexibility and the ability to change, control of structure, lack of classes, immediacy, time limitations, dislike of a classroom setting, expense, and transportation. (Tough, 1979, p. 39, cited by Van Fleet, 1995)

Although not frequently emphasized in the existing literature, it is likely that the cognitive changes discussed thus far (e.g., declining processing speed, memory retention, executive functioning) play a key role in the learning preferences described above. We discuss this hypothesis further in our discussion of Selective Optimization with Compensation below.

Finally, the context of learning in later life is often increasingly social and often serves

both social and emotional needs, whereas in earlier developmental periods the goals of learning tend to be more focused on educational attainment and occupational needs. For example, Lamdin and Fugate (1997) report results from the Elderlearning Survey (ES) indicating that 68% of older respondents prefer learning in groups, as compared to 22% who prefer learning on an individual basis. This mirrors a general shift observed in interpersonal aspects of aging as described in Carstensen's socioemotional selectivity theory (SST) in which social relationships functions shift away from informational purposes toward emotion regulatory functions (Carstensen, Isaacowitz, & Charles, 1999). SST observes that when individuals are younger, a broad social network serves achievement oriented goals and functions (e.g., educational and occupational attainment), but as individuals grow older and perceive they have less time left to live, they pare down their social network and focus on those relationships that are emotionally fulfilling and enhance meaning (Carstensen, 2006). Just as relationship functions shift with age, it appears that the needs served by learning in later life may follow a similar shift and may serve more than just informational functions.

In the sections below, we consider in greater detail how the Selective Optimization with Compensation (SOC) framework described by Baltes and Baltes (1990) can provide a conceptual model for understanding how the neuropsychological changes described in this chapter are related to these three observations about late life learning. Specifically, SOC provides a framework for understanding how, despite significant changes in functioning, older adults continue learning, and how new learning might be promoted in the context of dementia.

SOC and Shift in Ways and Means of Learning in Normal Aging

The SOC model was developed by Baltes and Baltes (1990) in their life-span development research related to intellectual and cognitive changes, and it seeks to answer the following question: "Can we envision a prototypical strategy of effective aging that allows for self-efficacy and growth in the context of increasing biological vulnerability and reduced reserve capacity?" (1990, p. 21). This question is remarkably similar to the central question of this chapter: Can we envision a strategy which allows for effective learning in the context of increasing biological vulnerability (both age and disease related change) and reduced learning potential? As noted by Marsiske and colleagues (1995), this model is sufficiently broad and flexible that it can be applied to a wide variety of life-span developmental issues, and here we apply it to the issue of how older adults can continue with successful learning activities in the face of cognitive decline and reduced learning potential.

The three processes (selection, optimization, and compensation) interact with one another to make it possible for elders experiencing reductions in learning potential and greater cognitive vulnerability to continue learning. The first process (selection) refers to a shift toward a reduction in the number of domains a person maintains as he or she grows older and greater specialization that occurs in line with one's priorities (Baltes & Baltes, 1990; Marsiske et al., 1995). According to SOC, as individuals age they may no longer seek to focus their attention on multiple domains of functioning, but may instead seek to shift their focus to selected domains due to limited time or declining cognitive resources. However, it should be noted that "although selection connotes a reduction in the number of high-efficacy domains, it can also involve new or transformed domains and goals of life" (Baltes & Baltes, 1990, p. 22). The shift therefore in relation to learning may involve a focus on a smaller number of projects in valued domains or taking on a

new goal or domain (e.g., computer learning for enhanced communication). The prediction of SOC for learning might be that in the face of declining cognitive resources, individuals will reduce the number activities they engage in, and focus in on only those deemed to be most important or valuable.

Optimization suggests that older adults are more likely to select those domains of functioning which make use of and enhance their remaining skills and cognitive strengths and thereby serve to improve quality of life and functioning (Baltes & Baltes, 1990; Marsiske et al., 1995). Thus the older learner may choose to focus attention on specific learning projects which have high value in terms of maintaining interest and efficacy rather than focusing attention more broadly as might have been done at earlier developmental periods. Optimization is a process that necessarily focuses on enhancing function within selected domains, and therefore the older learner may also take advantage of and build upon existing areas of specialization or strength (Marksiske et al., 1995).

Finally, the process of compensation is activated "when specific behavioral capacities are lost or reduced below a standard required for adequate functioning" (Marsiske et al., 1995, p. 22) and, as a result, the "individual and their contexts are challenged to reassess their earlier means-ends, and/or to construct alternative strategies" (p. 44).

An elder learner who places a priority on learning, but is nevertheless faced with declining cognitive resources, may choose a smaller number of focused learning projects (selection), or build upon remaining cognitive strengths (optimization). If cognitive functioning has been compromised such that old methods of learning are no longer adequate, the elder learner may seek to find alternate strategies for continued learning in the focused domains selected (compensation).

Changes in memory, processing speed, and executive functioning may force elders to modify their learning strategies, settings, and goals via the processes of selection, optimization, and compensation. If we return to the computer learning example with which we began this chapter, the elder learner may choose to drop other activities or tasks to focus her resources on the computer learning project (selection) that she views as more important and possibly emotionally fulfilling (optimization) because it allows her to communicate more regularly in key interpersonal relationships. She may also choose to learn via a self-directed learning method rather than a formal class because she prefers to learn at her own pace (compensation for possible declines in processing speed) and has excellent reading ability and can make use of various instructional books (optimization). Instead of taking university computer classes, she may find that other learning opportunities may be more appealing particularly because of the cognitive demands of highly structured courses which move very quickly, rely on dual-task processing (e.g., taking notes while processing the lecture), and draw heavily on delayed recall of information. By selecting and compensating in this fashion, elders may be able to continue learning despite cognitive change, and optimize their current functioning and quality of life.

The observed shift from formal learning to self-directed learning observed in later life appears to represents an example of SOC in response to the cognitive changes that occur secondary to age and disease related changes in the brain. (This is not meant to imply that cognitive change is the sole, or even primary, reason for this shift—which likely reflects a number of changes including financial resources and interests among other factors.) Self-directed learning may allow individuals to optimize intellectual abilities and learning opportunities while compensating for declines in basic cognitive abilities, such as speed or episodic memory, by allowing them to work at their own pace, engage in as much practice as needed, and reducing demands on memory required for many classroom performance indicators. In essence, the SDL approach may reduce demands on

cognitive abilities that reliably decline while allowing individuals to select the learning experiences that have the greatest personal value and potential to optimize functioning while building on existing skills or expertise. The positive implication of this framework is that, although cognitive declines are likely to be observed with increasing age, most elders who seek to engage in new learning will have a reasonable probability of being successful because they find ways to compensate for losses, optimize using spared abilities, and select only the most valued domains.

On the other hand, SDL by definition has less external structure which has both positive and negative features for older adults. On the positive side, the SDL approach allows older learners to establish the pace, goals, and methods of learning (Lamdin et al., 1997; Tough, 1979, 1999; Van Fleet, 1995). On the negative side, SDL requires considerable initiative, organization, planning, and problem solving in response to learning activities (i.e., revised plan if the original plan is not successful or needs modification). These prerequisite skills are among those thought to be most affected by aging of the prefrontal cortex (West, 1996). Disruption of both the primary (organization of behavior and cognition) and secondary functions (interference control, provisional/working memory, prospective memory) of the PFC would clearly affect learning in late life quite broadly, but may particularly affect SDL. In addition, to the extent that lesions within the frontal-subcortical circuitry lead to depression, apathy, and lack of initiative (Cummings, 1993; Tekin & Cummings, 2002), a subset of elders will likely have difficulty judging their ability to complete such projects and lack the initiative to develop and carry out a plan for accomplishing the learning. It should be noted however, that it would be expected that these behavioral issues would likely be expected in those with more severe PFC pathology and would be most applicable to only a subset of elders. Yet, as further connections between elderlearning and neurosciences literatures develop further, it will be important to consider these behavioral/motivational factors in addition to the traditional domains of cognitive aging research. For the discussion below, however, we will restrict our focus on the cognitive changes associated with PFC dysfunction.

PFC changes are common in later life (Gunning-Dixon & Raz, 2000; Raz & Rodrique, 2006; Raz, 2005; West, 1996), which conceptually would be expected to be particularly disruptive to SDL. Yet, data indicate many older adults tend to prefer this form of learning (Lamdin et al., 1997; Tough, 1999). Moreover, evidence from cognitive intervention studies suggest that older adults derive similar benefit from self-directed cognitive interventions as they do from interventions that are more structured and led by an instructor (see Thompson & Foth, 2005, for a review). Here again we draw on the SOC framework to understand how older adults might optimize SDL learning in the context of declining PFC functions (organization, interference control, prospective memory). In this context, the SOC processes of selection and optimization will likely function in a very similar fashion as in the computer example given above. Those elders who continue with SDL despite declining PFC resources will likely have narrowed the number of learning projects (or other activities) to focus their cognitive resources more narrowly (selection) on a project which enhances their functioning or quality of life (optimization). Compensation for any apparent weaknesses in cognitive ability will again be important, but in the context of declining PFC, external compensatory techniques to provide structure, guidance and reminders to accomplish specific tasks might become increasingly necessary. The use of external aids or cognitive enhancement strategies (list making, external cues to remember steps to be taken, organizational notebooks) can help to maintain learning in valued domains. Thus the three processes work together to help maintain SDL activities in the context of PFC changes. The conclusion again is that the brain changes

observed in normal aging do not prevent new learning for most older adults, but likely will affect the compensatory methods used for optimizing new learning. For some this may take the form of learning in increasingly social settings or with other learners who can help provide structure for SDL. Learning with other individuals may act as an external compensatory strategy in that they can assist in structuring the learning project, provide reminders to carry out the various steps, help obtain needed resources, and provide feedback, assist in problem solving and help utilize strategies for completing the task (see Saczynski, Margrett, & Willis, 2004). In short, learning in social settings may allow for other individuals to serve the functions that were mediated by PFC in the past.

The preference for learning in social settings over individual learning may have a compensatory function as described above, but it is more frequently cited as an example of selection and optimization (Willis, 1985). That is, social learning opportunities serve important social and emotional functions for older learners, such that these individuals select these options over other, less social learning activities (Tough, 1999), and, as a result, the social aspects of these learning activities optimize both quality of life and functioning.

Whether any of the neuropsychological changes observed in late life might affect the success of social learning activities remains an open question. Depression and ToM may be particularly important in this context. If ToM declines with age (the evidence is not clear) and frontal lobe dysfunction occurs, social functioning might become impaired, and older adults may have difficulty getting their social and emotional needs met in this context. As neuropsychological research extends into social contexts the extent to which ToM declines and interferes with social functioning will likely become clearer. Consistent evidence is not yet available to make firm conclusions, but to the extent that cognitive and affect goals and skills become integrated in late life (Labouvie-Vief, 2003; Labouvie-Vief & Diehl, 2000), the ToM construct may prove particularly useful in investigating successful learning activities.

SOC and Learning in AD

Thus far we have focused our discussion of SOC on situations where cognitive decline is not sufficient to warrant a diagnosis of dementia. Yet, despite the lack of clarity as to the boundary line between normal aging and dementia syndromes like AD, a portion of older adults clearly experience brain changes that are sufficient to cause dementia syndromes, and these changes appear to be sufficiently severe to interfere with formal learning (Baltes et al., 1992; Baltes et al., 1995). The combination of significant impairments in episodic memory, the ability to benefit from feedback, and implement plans clearly impact the extent to which elders can learn new information (O'Brien et al., 2003; Royall, 2000; Swanberg, Tractenberg, Mohs, Thal, & Cummings, 2004; Welsh et al., 1991, 1992). Moreover, as discussed above, the cognitive intervention studies conducted by M. Baltes and colleagues clearly demonstrate that both dementia and pre-dementia states interfere with new learning (Baltes et al., 1992, 1995, 1996; Raykov et al., 2002). It is in this context that we turn our discussion to emerging work describing how learning may be possible even in severely impaired individuals. SOC is used as a broad framework to describe how the techniques described below offer ways that those who have AD might be able to compensate for significant cognitive decline while optimizing other cognitive functions (implicit learning and memory) that are relatively spared in AD.

Spaced Retrieval in AD. As reviewed above, dementia (especially AD) interferes with patients' ability to learn new information in traditional training paradigms, and, as a

result, it was thought that little could be done to improve learning outcomes in individuals with dementia. Throughout the past decade, however, this perspective has been challenged by the finding that certain types of memory are relatively spared in persons with AD (Bayles & Kim, 2003; Bondi & Kaszniak, 1991; Mahendra, 2001; Squire & Zola, 1996).

Individuals with dementia experience greater damage to declarative (explicit) memory systems than to non-declarative (implicit) memory systems (Bayles & Kim, 2003). Evidence that learning occurs through preserved implicit memory processes stems from the finding that although people with dementia have the ability to remember target responses successfully, they do not explicitly remember the actual learning process or training sessions (Bayles & Kim, 2003; Camp, Foss, O'Hanlon, & Stevens, 1996; Mahendra, 2001).

Spaced retrieval is one behavioral intervention that capitalizes on spared implicit memory systems and can lead to new learning in individuals with moderate to advanced dementia. Spaced retrieval is essentially a shaping technique that utilizes principles of cognitive rehabilitation (e.g., spacing effect and operant learning) to facilitate learning (Cherry, Simmons, & Camp, 1999). The subject is asked to make a particular response (such as verbally answering a question or physically performing an action) repeatedly over increasing intervals of time (e.g., 15, 30, 60, 90 sec., etc.). If the subject provides an incorrect response, the trainer provides the correct response and then asks the subject to repeat it. The between trial delay then returns to the last time interval that the subject was able to respond correctly. If the subject provides the correct response, the next time delay is increased.

Early spaced retrieval research focused on younger adults but eventually shifted toward individuals with dementia (Camp, Bird, & Cherry, 2000). In Camp's initial studies, he was able to teach one woman and two men diagnosed with Alzheimer's disease to remember face-name associations (Camp & Stevens, 1990). The woman was able to demonstrate a one week retention interval (the ability to remember the to-be-learned information at the beginning of the next training session one week later) after seven weeks of training and the men were able to do so after three weeks of training. Camp also taught six individuals diagnosed with AD to recall the location of common objects (e.g., location of glasses). Three of the participants were able to demonstrate retention of learned material over at least one week during the eight weeks of training and all three demonstrated retention of the target information at a follow-up session (five weeks later). Cherry, Simmons, and Camp (1999) were also able to teach (or re-teach) the names of everyday objects (e.g., carrot, chair) to four older adults with probable AD. In each of these cases, new learning was demonstrated using SR in the context of relatively advanced dementia.

In one report SR was successfully implemented by a patient's caregiver—suggesting that caregivers can incorporate SR techniques to enhance learning in AD (McKitrick & Camp, 1993). The participant was a 77-year-old woman diagnosed with AD, and the training targeted forgotten names of common objects. During 10 weekly one hour visits, the caregiver observed the experimenter train the patient on two initial items (canoe and globe) using the SR method. The caregiver then began training two new objects (toothbrush and mushroom). Lastly, the patient was trained to learn an unfamiliar item (floppy disk). The experimenter began the training of the unfamiliar item and then the caregiver took over training. All items were learned by the participant, including the unfamiliar item. The items were also recalled with greater consistency after the training than before.

Spaced retrieval has also been used with more complex tasks. Because instructions for future actions (i.e., prospective memory) are often forgotten by individuals with AD, investigations of behavioral interventions that could enhance prospective memory abilities in AD patients are needed. McKitrick, Camp, and Black (1992) conducted such an investigation with four participants diagnosed with AD who demonstrated an inability to perform both a verbal and nonverbal prospective memory screening task (McKitrick et al., 1992). The training goal of this study was to learn and demonstrate the ability to accurately choose a pre-selected color coupon to be redeemed for money. At the beginning of each weekly training session, the participants were given the chance to select the correct color coupon (i.e., the color they had been taught to select) out of nine possible colors (shown in a 3 × 3 matrix). If the participant was able to choose the correct color coupon, a new color was chosen to be the target of the next training session. If the participant was unable to select the correct color coupon, SR training for that color coupon continued. Three participants were able to successfully execute the task after one week of SR training and the other participant required five sessions. In addition, all participants were able to successfully adjust their performance when the target coupon color was changed.

Spaced retrieval has also been used to enhance other cognitive interventions and compensatory techniques. Memory wallets and memory books can help individuals with cognitive impairment compensate for their deficits. However individuals with advanced dementia may not be able to utilize these compensatory methods because they forget when to use it, where they left it, or even that it exists (Bourgeois, Camp, Rose, White, Malone, Carr et al., 2003). Bourgeois et al. (2003) investigated the effectiveness of SR and a cueing hierarchy approach to teaching goals (a clinician-identified problem behavior, such as forgetting what activities are available each day) to individuals with dementia through the use of external memory aids (e.g., memory book). Twenty-five participants with dementia worked on individualized goals to decrease identified problem behaviors. Researchers found that the majority of participants were successful in achieving their goals by learning to use the external aids via SR or cueing, but that SR had slightly better outcomes. Camp, Foss, O'Hanlon, and Stevens (1996) also used SR training to teach participants with AD how to successfully use a daily calendar as an external memory aid. Participants were successful in learning to remember to use the calendar and to complete the secondary task (i.e., help with dishes, send a note to a family member) written on the calendar that day.

Brush and Camp (1998) investigated the effectiveness of training speech pathologists to use SR techniques to help patients with dementia use compensatory communication techniques in order to address language problems in dementia. Nine participants who were enrolled in speech language therapy at the Menorah Park Center for the Aging participated in this study. Seven had a diagnosis of dementia and two had a stroke within the year prior to the study. Training took place approximately three times a week during the patient's regularly scheduled speech-language therapy sessions. Using SR, the clients were first taught the name of their speech-language therapists. Once the subject could remember and correctly state the therapists' name in two consecutive sessions, the training for that target stopped. Spaced retrieval was then used to train the subject to remember a piece of meaningful information chosen by the participant, such as date of birth. Again, when the subject successfully remembered the target piece of information at the beginning of two consecutive training sessions, training for that target ended. For each of the training goals, the subject continued to be tested at the beginning of the remaining therapy sessions to determine retention. Finally, SR was used to train a compensatory

technique that would facilitate communicative functioning, such as teaching a client to make eye contact when speaking or to properly use a voice amplifier. Training continued until the subject could use the compensatory technique correctly at the beginning of two consecutive therapy sessions. Five out of the seven participants with dementia and both clients with stroke completed the study and progressed toward meeting speech-language therapy goals (Brush & Camp, 1998).

Although there is considerable research to support using the SR method, most of the studies investigating SR do not meet standards typical of clinical trials, in that most "have used small ($n < 10$) sample sizes, have not used control groups, have not randomly assigned patients to conditions, and have not used assessors blind to condition" (Davis, Massman, & Doody, 2001, p. 2). In addition, although SR has been shown to work on teaching specific to-be-learned pieces of information or behaviors, it has not resulted in improvements in overall cognitive functioning or general memory abilities (Camp et al., 1996; Davis et al., 2001).

Davis et al. (2001) conducted a randomized, placebo-controlled study with 37 patients diagnosed with probable AD. The placebo group received five weeks of mock intervention, while the intervention group received five weeks of cognitive intervention consisting of face-name training, SR, and general cognitive stimulation. Intervention patients received individual weekly one-hour clinic visits for five weeks and were instructed to also participate in 30-minute home attention exercises for six days per week. The patients in the intervention group showed significant improvement on recall of personal information and face-name associations but not on standard measures of cognitive performance (e.g., verbal memory, motor speed, verbal fluency), mood or caregiver-rated quality of life).

Camp, Foss, O'Hanlon, and Stevens (1996) investigated the efficacy of SR training on calendar use and also found that, although participants were successfully trained to effectively use daily calendars, this success did not carry over to improvements in general cognitive functioning. In fact, significant cognitive declines were apparent over the course of the study. Therefore, consistent with the broader cognitive training literature (e.g., Ball et al., 2002; Schaie, 1994; Willis et al., 2006), the training effects of SR are highly specific to the trained ability and do not appear to generalize (or transfer) to other functions. On the other hand, the method has demonstrated that individuals with advanced dementia can learn target information and retain this information for a considerable period of time after the initial learning (Cherry et al., 1999).

In this regard SR appears to be a very promising behavioral intervention for persons with dementia. The SR method has been shown to be effective across a number of simple associations (face-name associations, object naming, object location, object selection, and remembering personal information) as well as more complex tasks (using external memory aids such as calendars and memory books, completing prospective memory tasks, and using compensatory speech-language techniques). As noted by Bourgeois et al. (2003), SR has also been shown to work in a number of dementing conditions including Alzheimer's disease, vascular and mixed dementia, post-anoxia dementia, dementia associated with Parkinson's disease, dementia associated with Korsakoff's syndrome, and dementia associated with HIV. Finally, the SR method has been successfully implemented not only by researchers, but by individuals who use spaced retrieval techniques in the real world, such as speech-language pathologists and caregivers.

Perhaps most importantly for our purposes, SR offers some insights into effective procedures to enhance learning in AD and other dementias. Camp and colleagues (2000) have focused much of their efforts on procedural changes to the SR technique that have resulted in a reduced cognitive effort required of the subject and resulted in more social

and enjoyable training sessions (Hochhalter, Overmier, Gasper, Bakke, & Holub, 2005). Camp et al. (2000) found that an expanding interval schedule used in early SR research resulted in high error rates, and therefore switched to a more gradual increase in time between recall levels (5, 10, 20, 30, 40, 60, 90, 120 seconds, etc.). Thus, a more gradual recall interval expansion rate is utilized and the length of the between trial delays is based on the subjects, performance on previous trials (Camp et al., 1990, 2000; Hochhalter et al., 2005). In addition, one piece of information is taught at a time versus multiple pieces concurrently (Camp et al., 1990; Hochhalter et al., 2005). Focusing on one target at a time reduces the chance that targets will interfere with one another in the learning process.

The SR techniques used by Camp and colleagues emphasize errorless learning (1990; Hochhalter et al., 2005). According to Mahendra (2001), "errorless learning (EL) is a technique whereby patients are prevented, as far as possible, from making mistakes while they are learning a new skill or acquiring new information" (p. 296). Because common forms of dementia, such as Alzheimer's disease, disrupt episodic and working memory systems, individuals with dementia "cannot use the past as a basis for correcting themselves" (Bourgeois et al., 2003, p. 364). Therefore, "only if they are not allowed to make errors will they learn new information accurately; otherwise, inaccurate learning will take place" (Bourgeois et al., 2003, p. 364). By capitalizing on errorless learning and implicit memory systems, learning occurs "effortlessly" (Camp et al., 1990) with a high frequency of success, resulting in greater self-esteem and mastery (Camp et al., 1990; Brush & Camp, 1998).

The SR technique is also advantageous in that it can be nested within social and leisure activities and, thereby, enhance motivation for training and reduce performance anxiety (Camp et al., 1990, 1996; Mahendra, 2001). The social setting may increase engagement and may fulfill social and emotional needs in later life, which may be a key feature of effective learning in dementia. In the next section, we turn our attention to complementary techniques that have been used to further enhance engagement and thereby enhance learning in AD.

Montessori Techniques in AD

Montessori methods have been utilized to increase the ability of AD patients to engage in and learn appropriately stimulating activities and thereby reduce the frequency of problem behaviors and increase overall quality of life (Orsulic-Jeras, Schneider, & Camp, 2000). We discuss the Montessori method here because it also has the potential to enhance learning in individuals with AD by increasing engagement in selected learning activities, optimizing use of relatively spared cognitive skills, while compensating for declining abilities, particularly processing speed and executive functioning.

The Montessori approach emphasizes "the importance of self-paced learning and developmentally appropriate activities" (Camp & Skrajner, 2004, p. 427) and "modifying learning environments and activities to accommodate adult learners" (Vance, Camp, Kabacoff, & Greenwalt, 2006, p. 11). Many of the Montessori principles and techniques can be utilized to create interventions for promoting learning among individuals with dementia. First, materials are taken from the everyday environment, are designed to give extensive cuing and guidance in terms of how an activity should be completed, and are self-correcting. This allows for immediate feedback to control against error and lets learners know whether or not the task has been completed successfully (Judge, Camp, & Orsulic-Jeras, 2000; Orsulic-Jeras, Schneider, Camp, Nicholson, & Helbig, 2001;

Schneider, Diggs, Orsulic, & Camp, 1999; Vance et al., 2006). Second, larger tasks are often times broken down into smaller steps and are initially taught at the simplest or most concrete level, followed by more complex or more abstract levels if appropriate (Camp & Skrajner, 2004; Judge, Camp, & Orsulic-Jeras, 2000; Orsulic-Jeras et al., 2000; Vance et al., 2006). Third, learning occurs in a sequence that progresses from instructor modeling toward recall and demonstration of the activity by the learner (Orsulic-Jeras et al., 2001). These principles provide the structure and assistance needed to compensate for deteriorating executive functioning in dementia. A more complete list of Montessori principles as they relate to dementia can be found in Orsulic-Jeras et al. (2001).

Like spaced retrieval, Montessori-based activities rely on practice and guided repetition and capitalize on spared implicit memory systems in persons with dementia (Camp & Skrajner, 2004; Orsulic-Jeras, Schneider, & Camp, 2000), thereby improving performance on an activity even if the learner cannot explicitly remember participating in the activity (Orsulic-Jeras, Schneider, Camp, Nicholson, & Helbig, 2001). Modeling is an important aspect of the Montessori method. The person teaching the activity will demonstrate the activity prior to asking the learner to try that same activity (Schneider, Diggs, Orsulic, & Camp, 1999).

The Montessori method stresses the importance of individualizing activities and considering each participant's "past occupations, past interests and present cognitive and physical abilities" when creating activities (Orsulic-Jeras et al., 2001, p. 108). By tailoring activities to an individual's needs, interests, and cognitive ability, the activities become more engaging and can increase the probability of success. This may optimize function in that new learning builds upon spared abilities or expertise to enhance functioning and quality of life (Baltes & Baltes, 1990).

Montessori-based activities can be conducted with both individuals and groups, and can be used with individuals across various stages of dementia (Orsulic-Jeras et al., 2000). Individual Montessori activities may focus on intergenerational programming in which an older adult is paired with a child, or other one-on-one individual activities, in which an older adult is paired with a staff member. Examples of activities that have been used in prior research include "picture and word sorts" and "matching cufflinks and earrings" (Schneider et al., 1999). The interaction with a child gives the individual with dementia the opportunity to serve as a mentor and be successful in an activity (Orsulic-Jeras et al., 2001) and provides for the social and emotional functions of learning in late life as discussed in earlier sections of this chapter.

Small group Montessori activities include Memory Bingo and Question Asking Reading (QAR). These group activities capitalize on a number of Montessori principles to increase engagement including frequent feedback, provision of structure to the activity participation, and considerable practice/repetition (Orsulic-Jeras, Schneider, & Camp, 2000). QAR is "designed to incorporate external cues in order to strengthen participants' focus on the task and improve comprehension and concentration" (Orsulic-Jeras et al., 2000, p. 83). Each participant is given a copy of the story to follow along with, gets a turn to read a paragraph, and is given a color coded statement to read out loud to encourage discussion (Orsulic-Jeras et al., 2001). Although the content of the story may change, the procedure remains the same for each QAR session, allowing for participants to learn the procedure through implicit memory systems and thereby increase the probability of successful engagement and learning.

Several empirical studies have investigated the effect of Montessori activities on learning and engagement in individuals with dementia. For example, Judge, Camp, and Orsulic-Jeras (2000) conducted a study in which nineteen participants with dementia

aged 60- to 101-years-old were assigned to either a treatment (9 subjects) or control group (10 subjects) and were matched according to their MMSE scores. Participants in the treatment group participated in both individual and group Montessori-based activities twice a day throughout the study as an alternative to the regularly scheduled activities at the center. Subjects were rated on their level of engagement in the activities over 10-minute segments throughout the day at three different times throughout the study: at baseline, before Montessori-based activities were implemented, and at two follow-up points (four and eight months after baseline). Results indicated that participants in the Montessori condition demonstrated significantly greater constructive engagement when compared to those in the control condition.

Thus far, the Montessori-based activities utilized in these research studies have been implemented by trained investigators. However, to have practical application on a broad scale the activities need to be implemented by others, including caregivers and employees of long-term care nursing facilities. Orsulic-Jeras et al. (2001) investigated whether the activities staff could incorporate Montessori-based activities into their established programming schedules. The activities staff first participated in three half-hour preliminary training sessions (*Understanding Dementia*, *The Montessori Method*, and *Presenting Montessori-Based Activities*) and had access to a training manual developed by Camp and colleagues. The staff was then trained on the Myers Menorah Park/Montessori Assessment System (MMP/MAS), an assessment tool used to examine a subject's "remaining cognitive, motor, sensory, and social skills" (p. 116) to help guide the development of appropriate activities for that individual. The results showed that the activities staff members were able to successfully implement the Montessori-based activities and reported positive changes in both the residents and the staff members themselves after implementing these activities. In a similar study, Schneider, Diggs, Orsulic, and Camp (1999) taught nursing assistants at the Myers Research Institute of the Menorah Park Center for Aging to implement Montessori based activities to residents with dementia. The results of the seven-month study showed that patients were more actively engaged during individual and small group Montessori activities then when participating in their regular activities. Also, the residents showed less negative affect and greater positive affect while participating in Montessori activities compared to their regular environment.

Camp and Skrajner (2004) conducted a study investigating whether dementia patients themselves could learn to serve as group leaders and implement Montessori-based activities for their fellow residents. Four female residents of a senior living facility, diagnosed with either Alzheimer's disease or another form of dementia, were trained to lead Memory Bingo. Training sessions were conducted once or twice a week until the trainee felt comfortable with the game. Data collection began once the leaders indicated that they felt capable of leading the Montessori group activity. Assessments of both the leaders and players (who were residents with more severe dementia) were conducted. The assessments of the leaders revealed a high frequency of at least partial adherence to protocol, which enabled all games to be completed. Players demonstrated greater constructive engagement after the implementation of Montessori program activities.

These findings suggest that Montessori techniques have positive psychosocial effects and that persons with dementia can continue learning successfully when they are engaged in stimulating activities that are matched to their cognitive strengths (optimization) and are accompanied by external compensatory support (e.g., task break down) provided by this approach (Judge et al., 2000; Orsulic-Jeras et al., 2001; Schneider et al., 1999). Taken together, SR and Montessori techniques may enhance learning in dementia by compensating for declines in memory (SR), executive functioning and processing

speed (Montessori), and optimizing the use of both implicit learning and memory and environmental structure.

Conclusion

Normal brain aging is accompanied by declines in prefrontal cortex (PFC) volumes, greater white matter lesions, particularly within the PFC. In this context, normally aging elders experience declines in processing speed, executive functioning, and working memory. These brain-behavior changes may lead to modification of the methods and goals of late life learning via the processes of selection, optimization and compensation (SOC), but these changes do not preclude new learning in most older adults. However, more severe forms of cognitive change, such as dementia, do appear to significantly interfere with new learning. Dementia syndromes, such as Alzheimer's disease, lead to more dramatic changes in the brain, particularly within the medial temporal and prefrontal cortex. Nonetheless, even in moderate stages of dementia, some forms of new learning may be possible when the learning techniques can optimize the use of spared cognitive abilities and compensate for affected abilities. The broad SOC framework may serve as a fruitful guide for future research into late life learning, particularly in the context of dementia and other cognitive syndromes. It can also assist both caregivers and educators in appropriate planning for individualized interventions.

Notes

1. The challenge of fully utilizing the concept of preclinical AD lies in the fact that AD pathology cannot be detected prior to death. Until very recently, it was not possible to use neuroimaging methods to image AD characteristic changes in the living brain. Preliminary evidence suggests that a new compound (Pittsburgh compound) can be injected into the body and appears to accumulate in amyloid plaques in the brain imaged with positron emission tomography (PET) methods(Fagan, Mintun, Mach, Lee, Dence, Shah, et al., 2006). This technique has potential for detecting preclinical AD, but will require further replication and development before it is used clinically.

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