The Wiley-Blackwell Handbook of Individual Differences

Edited by Tomas Chamorro-Premuzic,
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Individual Differences in Cognitive Aging

Elliot M. Tucker-Drob and Timothy A. Salthouse

As populations of healthy adults grow older, average levels of performance in many different areas of cognitive functioning gradually decrease. Recently, however, researchers have begun moving beyond conceptualizing cognitive aging merely as a population-level phenomenon. Instead, there is a growing appreciation for person-to-person *individual differences* in the cognitive aging process. The two quotes that follow exemplify this shift.

> Researchers are recognizing increasingly that the study of mean change with age does not give a full account of cognitive change across the life span. Although the average performance on most tasks may decline with age, studies have suggested that many older individuals may change very little, whereas others deteriorate dramatically. (Christensen et al., 1999, p. 365)

> In some people cognition declines precipitously, but in many others cognition declines only slightly or not at all, or improves slightly. Determining the factors that contribute to this variability is likely to require detailed knowledge about individual differences in patterns of change in different cognitive abilities in old age. (Wilson et al., 2002, p. 179)

There are seven questions that we believe to be foundational to this burgeoning area of inquiry. These are:

1 To what extent do individual differences exist in aging-related cognitive changes?
2 How many explanations are needed for cognitive aging?
3 What are the moderators of cognitive aging?
4 What can improve cognitive performance in adulthood?
5 How does cognitive aging relate to real-world functioning?

The Wiley-Blackwell Handbook of Individual Differences, First Edition.
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6 What are the neurobiological substrates of individual differences in cognitive aging?
7 What are the genetic risk factors for cognitive aging?

In this chapter we summarize the progress that has been made toward answering each of these questions and discuss prospects for future research. First, we describe the pattern of cognitive aging at the population level.

When Does Cognitive Aging Begin and for What Cognitive Abilities?

Before addressing questions about individual differences in cognitive aging, it is useful to describe the basic population-level phenomenon in question. Lay intuition might suggest that aging-related cognitive decline only occurs for memory, that decline only occurs later in life, and that decline only occurs for the small segment of the population that experiences neurological disease. However, there is now strong evidence that declines occur for a number of different abilities apart from memory (e.g. reasoning, speed of processing, and spatial visualization); that these declines begin in early adulthood; and that declines occur for healthy, disease-free adults (Salthouse, 2004a).

Cross-sectional evidence

The most abundant sources of information about age-related effects on cognitive functioning come from cross-sectional studies, in which people of many different ages are tested during the same general period of time and compared to one another in their test performance. Among the first reports of cross-sectional age trends for cognitive abilities was an article published by Jones and Conrad in 1933. This study was based on a community sample of close to 1,200 rural New England residents between 10 and 60 years of age. Jones and Conrad observed that on nearly all of the sub-tests of the Army Alpha Intelligence Test, including Numerical Completion, Common Sense, and Analogies, mean levels of performance increased until approximately 18 years of age, at which point they declined continuously throughout adulthood. Two exceptions were the Opposites sub-test (i.e. antonym vocabulary) and the General Information sub-test, mean levels of which increased steeply in childhood and then leveled off in adulthood. Nearly identical cross-sectional trends in similar cognitive tests have been reported over the 75 years since Jones's and Conrad's original observations (e.g. Cattell, 1987; Li et al., 2004, Tucker-Drob, 2009; Wechsler, 1958). For tests that require effortful processing at the time of assessment (i.e. tests of *processing abilities*), mean levels of performance are highest during late adolescence and young adulthood, and monotonically decline with advancing adult age. For tests that require the production of previously acquired information (i.e. declarative knowledge) and/or highly automatized forms of processing (i.e. procedural knowledge), mean levels of performance peak in middle adulthood, after which point they remain relatively stable. These trends are illustrated in Figure 9.1, which is based on data

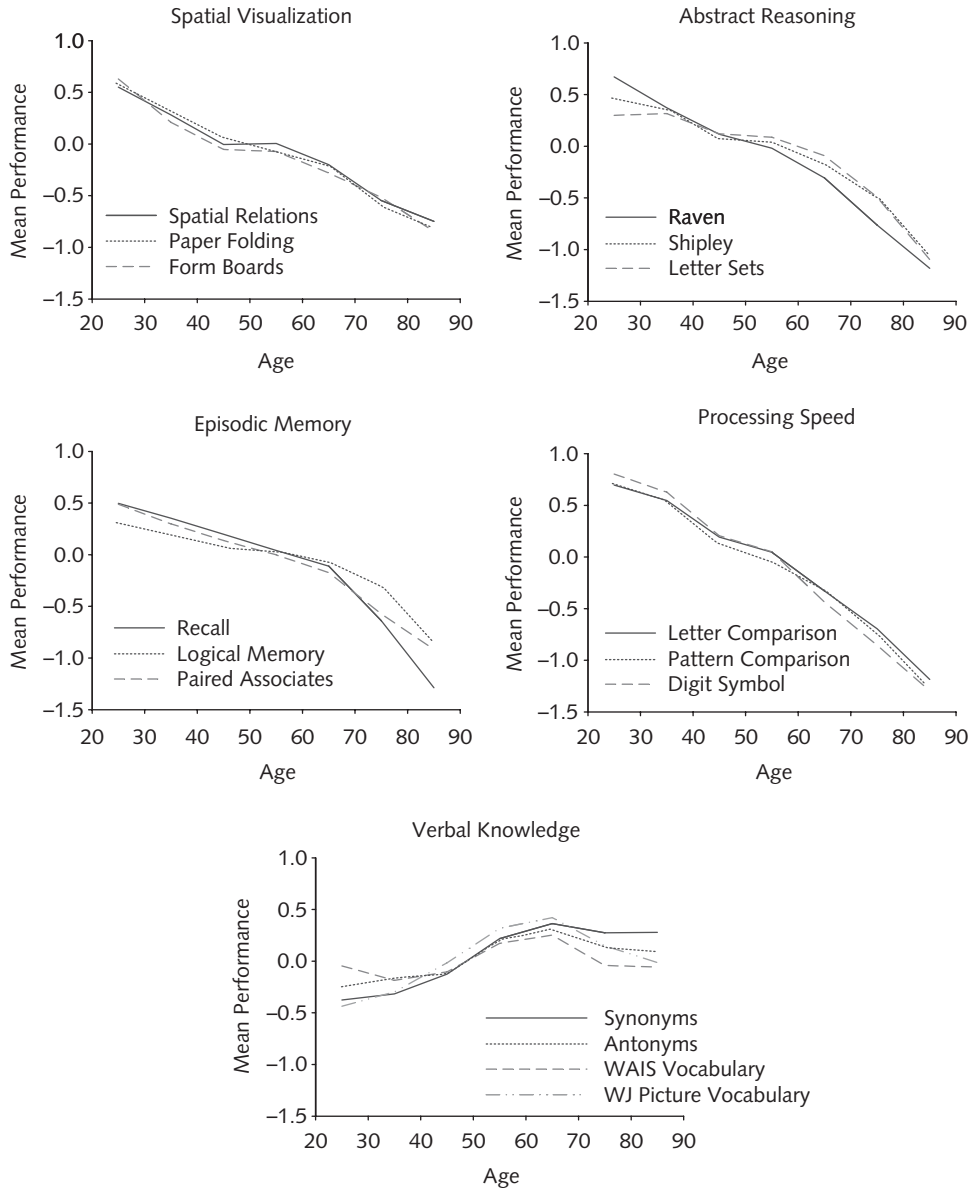

Figure 9.1 Cross-sectional age trends from the Virginia Cognitive Aging Project at the University of Virginia. VCAP; $N = 2{,}541$. All variables have been standardized to have a mean of 0 and a standard deviation of 1 in the entire sample

from the Virginia Cognitive Aging Project at the University of Virginia (VCAP; $N = 3{,}560$; Salthouse, 2004b; Salthouse, Pink, & Tucker-Drob, 2008; Tucker-Drob, in press a; Tucker-Drob & Salthouse, 2008, 2009). These data were collected over 16 tests representative of five different cognitive abilities, four of which (spatial visualization, abstract reasoning, episodic memory, and processing speed) require

effortful processing and begin declining in early adulthood, and one of which (verbal knowledge) reflects stores of previously acquired information and increases until approximately 65 years of age. It can be inferred that these patterns are not attributable to age trends in the prevalence of dementia, as the correlations between abilities and age are very similar, both before and after the exclusion of individuals with scores below 27 out of 30 on the Mini Mental State Examination (a popular dementia screening instrument; Folstein, Folstein, & McHugh, 1975). For composite scores representing each ability, these are:

- spatial visualization ($r_{\text{full sample}} = -.474$, $r_{\text{MMSE}\geq 27} = -.477$);
- abstract reasoning ($r_{\text{full sample}} = -.482$, $r_{\text{MMSE}\geq 27} = -.477$);
- episodic memory ($r_{\text{full sample}} = -.433$, $r_{\text{MMSE}\geq 27} = -.427$);
- processing speed ($r_{\text{full sample}} = -.629$, $r_{\text{MMSE}\geq 27} = -.627$); and
- verbal knowledge ($r_{\text{full sample}} = .245$, $r_{\text{MMSE}\geq 27} = .311$).[1]

Longitudinal evidence

Whereas cross-sectional data clearly demonstrate declines in multiple domains of effortful processing beginning in early adulthood, results of a number of longitudinal studies appear to indicate that declines do not begin to transpire until middle to late adulthood. Because logistic issues make it very difficult for longitudinal studies to span an entire lifetime, longitudinal evidence typically comes from what are termed "accelerated" or "sequential" designs, in which participants of different ages are followed over a few years (although see McArdle, Grimm, Hamagami, Bowles, & Meredith, 2009 for a notable exception). Figure 9.2 illustrates some typical findings. Data come from the Seattle Longitudinal Study (reproduced from Salthouse, 2005). It can be seen that, for longitudinal changes in inductive reasoning, for which

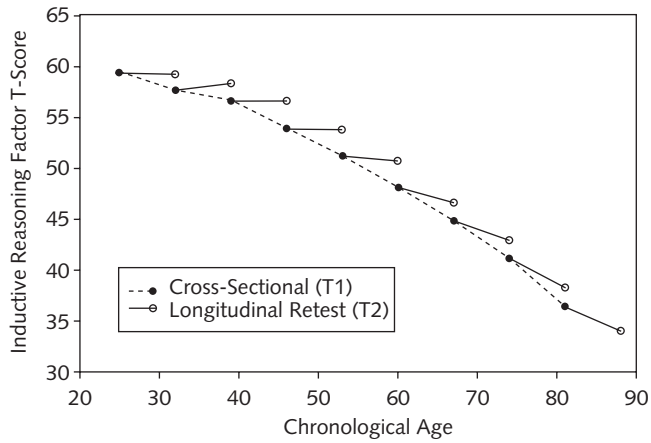

Figure 9.2 Cross-sectional and longitudinal age trends in inductive reasoning from the Seattle Longitudinal Study. Reproduced from Salthouse (2005, p. 553). The factor has been standardized to have a mean of 50 and standard deviation of 10 in the entire sample

cross-sectional studies indicate declines beginning in early adulthood, mean levels of performance actually increase until approximately 50 years of age, only after which point they begin to decline. How can the discrepancy between cross-sectional deficits and longitudinal gains be reconciled?

A number of factors, or validity threats, have the potential to contribute to the differences typically observed between cross-sectional and longitudinal studies (Salthouse, 2010b). One potential validity threat is the existence of cohort differences in cognitive functioning. If, all else being equal, individuals born in later generations begin adulthood with higher overall levels of performance (see e.g. Flynn, 1987) than those born in earlier generations, then these younger participants will outperform older participants (i.e. the participants born earlier) at any given time point, not because of aging-related changes, but because of historical differences (e.g. in nutrition or education). A second potential validity threat is non-random selection. If older participants in a cross-sectional study tend to be more positively selected than younger participants are, aging-related deficits could actually be masked in cross-sectional data. A related validity threat, selective attrition, involves lower-functioning participants being less likely to return for a longitudinal assessment (due either to lack of interest or to a relation between cognitive functioning and illness or death; Lindenberger, Singer, & Baltes, 2002), which would lead to an underestimation of aging-related deficits in longitudinal data. A final validity threat, and the one that we believe is the largest contributor to the empirically observed discrepancies between cross-sectional and longitudinal age trends, is that longitudinal research inherently requires the repeated testing of individuals and is therefore contaminated by practice-related learning as a result of individuals' accumulating experiences with the tests (Salthouse & Tucker-Drob, 2008).

How can we evaluate the contributions of each of these possibilities and their alternative implications for the validity of cross-sectional versus longitudinal research? A tremendous amount of work has been published on this topic (Baltes, Reese, & Nesselroade, 1977; Baltes & Schaie, 1976; Horn & Donaldson, 1976; Yang, Schullhofer-Wohl, Fu, & Land, 2008), and we cannot possibly attempt to summarize it all here. We do make the following observations. First, the validity threats do not all bias inferences in the same directions. That is, while some threats (e.g. cohort differences) imply that cross-sectional comparisons may overestimate decline, other threats (e.g. non-random selection) imply that cross-sectional comparisons may underestimate decline, and yet others (e.g. practice effects) imply that longitudinal comparisons may underestimate decline. Second, a number of different approaches have been used to correct for the validity threats, and each tends to be consistent with the proposition that cognitive decline begins in early adulthood. For example, when Rönnlund et al. (2005) corrected cross-sectional data for cohort differences in educational attainment and corrected longitudinal data for experience-related practice effects, results were consistent with early life declines in episodic memory. Salthouse (2009) has provided evidence that, when practice effects are removed either by comparing twice-tested individuals to once-tested individuals or by statistically correcting for the number of previous testing occasions that individual participants have experienced, aging-related deficits were apparent in early adulthood for episodic memory, spatial visualization, processing speed, and abstract reasoning. Third, neurobiological

indices thought to be related to cognition, such as brain size, begin declining in early adulthood in both cross-sectional and longitudinal data (Dennis & Cabeza, 2008). Fourth, continuous aging-related cognitive deficits have been documented in controlled studies of animals (Herndon, Moss, Rosene, & Killiany, 1997; Le Bourg, 2004), in which the threats to validity that are common to studies with human participants are not applicable. On the basis of these observations, we believe that there is conclusive evidence that, on average, aging-related declines in processing abilities begin in early adulthood, as suggested by cross-sectional age trends. Nevertheless, we value longitudinal approaches for the information they provide about individual differences in change, particularly when statistical methods for controlling for practice effects are applied.

To What Extent Do Individual Differences Exist in Aging-Related Cognitive Changes?

The most basic question of direct relevance to the topic of individual differences in cognitive aging is the question of whether appreciable individual variation actually exists in aging-related cognitive changes. That is, are there some people who decline more steeply than others, or, put differently, are there some people who experience little decline (or even increase) and others who experience much decline? We make two points of clarification here. First, this section is concerned with the simple existence of individual difference in changes in processing abilities. We address predictors of these individual differences in later sections. Second, we focus on the continuous distribution of individual differences in cognitive aging across normal healthy adults. We acknowledge that there are, very likely, large differences in cognitive declines between healthy adults and those who experience dementia. However, this chapter is only concerned with how normal adults differ from one another, not with how they differ from patient populations.

Cross-sectional evidence

One simple, albeit fairly crude, means of examining whether individual differences exist in cognitive aging is to examine whether there are age differences in the magnitude of between-person variation in cognitive performance. That is, one might expect the differences between individuals to increase with age, as some maintain high levels of performance while others experience large declines (note, however, that, if the most able decline the steepest, one might actually expect a pattern of decreasing variation in cognitive performance with age). Evidence appears to be mixed for the existence of age-related increases in between-person variation in adulthood. Morse (1993) analyzed data from studies published in *Psychology and Aging* and the *Journal of Gerontology* over a five-year period and concluded that adult age was related to increased variability in reaction time, memory, and reasoning, but not in verbal knowledge. On the basis of data from the WAIS–III standardization sample and of scaling standard deviations relative to mean performance (which we are critical of, because it confounds variation with performance level), Ardila (2007, p. 1010)

similarly concluded that aging-related declines in test scores were associated with increased test score heterogeneity. However, in analyzing data from a community sample of 1,424 adults, Salthouse (2004a, p. 141) alternatively concluded that the variation in speed, reasoning, and memory scores evidenced nearly constant variability, and that the entire distributions of scores shifted downward with advancing adult age. Moreover, in analyzing data from the Berlin Aging Study, Lindenberger and Baltes (1997) similarly found no evidence for age-related differences in variation in perceptual speed, fluency, memory, or general intelligence. Finally, in surveying the published statistics from the nationally representative norming samples from a number of standardized cognitive testing batteries, Salthouse (2010a) was unable to find clear evidence for systematic cross-sectional age trends in between-person variation in cognitive test performance. To judge from these findings, there does not appear to be much evidence that between-person variation in cognitive test performance increases with adult age. We do note, however, that cross-sectional differences in between-person variability are likely to be quite sensitive to age differences in the participation rates of adults of different levels of functioning (a validity threat known as "selectivity") and to failures of the assumption of interval measurement of the cognitive tests (of which ceiling and floor effects can be considered severe examples).

Longitudinal evidence

Given the limitations of cross-sectional approaches for making inferences about individual differences in cognitive changes, we turn to evidence derived from longitudinal data. In a longitudinal study, individual differences in cognitive aging would be directly reflected by individual differences in (i.e. variation in) rates of cognitive change. While variation in simple difference scores is likely to be disproportionally attributable to the existence of measurement error (Cronbach & Furby, 1970), new growth curve modeling and latent difference score modeling approaches enable researchers to produce estimates of variation in changes that are theoretically error-free. On the basis of these new methods, there is accumulating evidence for systematic and statistically significant variation in longitudinal cognitive change (e.g. Wilson et al., 2002). Even with measurement error removed, however, it is possible that individual differences in longitudinal change reflect a mixture of individual differences in true maturational change and individual differences in practice-related learning. We therefore emphasize studies that have examined whether between-person variation in longitudinal change persists after statistically correcting for estimates of between-person variation in practice effects. These include McArdle, Ferrer-Caja, Hamagami, and Woodcock (2002), Tucker-Drob, Johnson, and Jones (2009), and Tucker-Drob (in press a). Each study has reported significant variation in longitudinal slopes that is independent of variation in the practice effects (interestingly, variation in the practice effects was in many cases not statistically significant). What is the magnitude of this variation? Tucker-Drob (in press a) has reported that, in longitudinal data from VCAP, the ratio of the standard deviation of yearly maturational change to the standard deviation of individual differences at baseline was 9 percent, 8 percent, 12 percent, and 8 percent for reasoning, spatial

visualization, episodic memory, and processing speed respectively. While this variation in yearly change may appear to be modest, it is important to realize that compounding it across multiple years or decades can result in substantial heterogeneity in the cognitive aging process.

Finally, we call attention to evidence that individual differences in maturational cognitive change are reliable and systematic. Evidence comes from recent studies by Ferrer, Salthouse, McArdle, Stewart, and Schwartz (2005), Wilson et al. (2002), Tucker-Drob et al. (2009), and Tucker-Drob (in press a), all of which have reported moderate correlations (approximately r = .5 in magnitude) among rates of change in different cognitive variables, even after accounting for practice effects. Because correlations can only exist in the presence of systematic variability (see e.g. Hertzog, von Oertzen, Ghisletta, & Lindenberger, 2008), this is strong evidence that individual differences in cognitive change are systematic. We discuss the topic of correlated longitudinal changes in further detail in the next section.

How Many Explanations Are Needed for Cognitive Aging?

That age-related deficits are apparent on multiple measures representative of multiple domains of cognitive functioning raises the question of whether each of these deficits reflect a distinct developmental process, or they are all simply symptomatic of a fewer number of more general deficits. The former, *multidimensional* possibility would suggest the operation of a heterogeneous variety of causes of cognitive aging, with different causes affecting different functions. The latter, *few-dimensional* or *unidimensional* possibility would suggest a relatively smaller set of "common causes" (Baltes & Lindenberger, 1997), each one of which influences many different functions.

Shared influence approaches

Shared influence approaches derive from two observations. First, many different cognitive variables evidence moderate to large negative correlations with adult age. Second, all reliably measured cognitive variables evidence moderate to large positive correlations with one another. These two observations allow for the possibility that mean age differences on each of the different cognitive variables can be accounted for by way of the influences of age on just a few common factors.

Salthouse and colleagues have tested shared influence models in a number of large cross-sectional data sets (Salthouse, 2004b, 2009; Salthouse & Davis, 2006; Salthouse & Ferrer-Caja, 2003). The general finding is that the mean age-related deficits that are observed on a variety of different cognitive variables can be parsimoniously accounted for by way of age differences on a very small number of dimensions. This is illustrated as a path diagram in Figure 9.3 for cross-sectional data from the Virginia Cognitive Aging Project. In this case, the negative effects of age on 12 different cognitive variables can be well accounted for by the influences of age on three dimensions: a common factor (often termed "*g*"), an episodic memory factor, and a speed of processing factor.

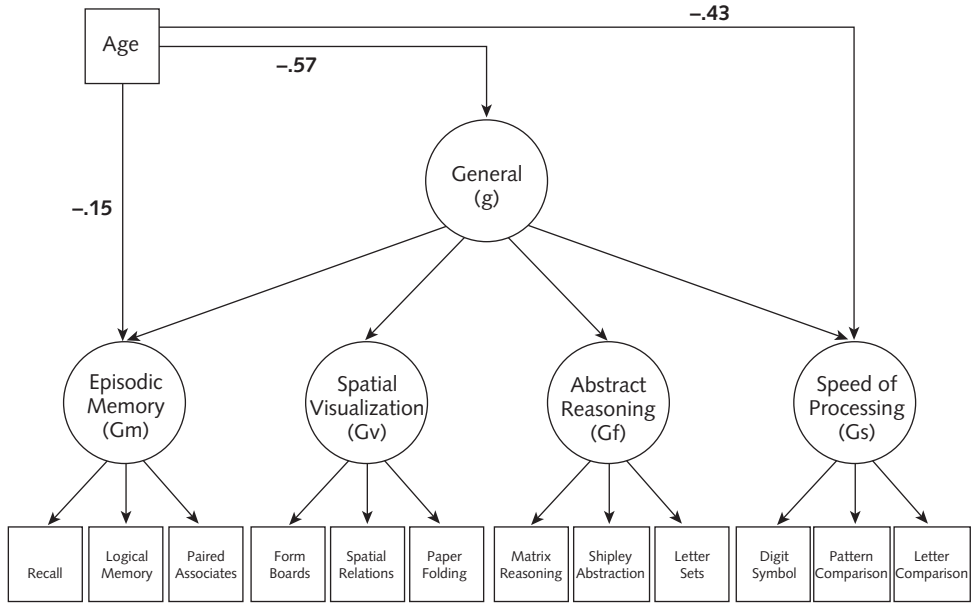

Figure 9.3 Localizing cross-sectional aging-related differences in a hierarchical structure

Correlated changes approaches

In the past decade, researchers have begun to estimate correlations amongst individual differences in longitudinal changes in different cognitive variables. In contrast to cross-sectional shared influences models, which examine the extent to which mean age differences are shared across different cognitive variables, these correlated change approaches examine the extent to which individuals' rates of cognitive changes relative to their peers tend to be similar for different variables. Correlated changes approaches help to answer a question posed most plainly by Rabbitt (1993): "Does it all go together when it goes?"

Evidence is beginning to accumulate to suggest that the answer to Rabbitt's question is a qualified "yes." Rates of change in a variety of different indices of cognitive functioning tend to be moderately correlated with one another, such that a large proportion (although not all) of the individual differences in changes in different cognitive domains are shared. Such correlations have been reported by Anstey, Hofer, and Luszcz (2003), Lemke and Zimprich (2005), Sliwinksi and Buschke (2004), and Sliwinski, Hofer, and Hall (2003). Ferrer et al. (2005), Tucker-Drob (in press a), Tucker-Drob et al. (2009), and Wilson et al. (2002) have reported that these correlations largely persist when practice effects are statistically controlled for.

Five studies (Hertzog, Dixon, Hultsch, & MacDonald, 2003; Lindenberger & Ghisletta, 2009; Reynolds Gatz, & Pederson, 2002; Tucker-Drob, in press a; and Wilson et al., 2002) have employed factor-analytic methods to examine the extent to which the changes in a broad variety of cognitive variables can be attributable to a common underlying dimension of individual differences in changes. Results have been quite consistent with one another, with a single common factor accounting for

between approximately 35 percent and 60 percent of individual differences in cognitive changes. Tucker-Drob (in press a) has, moreover, demonstrated that a hierarchical factor model can be fit to longitudinal cognitive changes. In such a hierarchical factor model, approximately 40 percent of individual differences in longitudinal changes in 12 tests of cognitive processing from VCAP (the same tests depicted in Figure 9.3) could be accounted for by a domain-general change factor, approximately 30 percent could be accounted for by domain-specific factors (reasoning, spatial visualization, memory, or processing speed), and approximately 30 percent was variation in change specific to the individual tests. These results, together, suggest that individual differences in cognitive aging are attributable to a mixture of a domain-general factor and multiple domain-specific factors.

It is of note that nearly all examinations of correlated changes have been based on data from middle-aged and older adults. The question of whether correlated changes also exist in young adulthood is, however, relevant to at least two major issues. First, as was described earlier, there is still some controversy regarding whether meaningful age-related deficits indeed begin in early adulthood. If abilities remain stable and do not decline during early adulthood, one would not expect individual differences in change to exist in healthy young adults. Alternatively, establishing that similar patterns of individual differences in change pertain to younger and older adults would suggest that the meaning of change does not differ with age (see Salthouse, 2010c), and therefore it would undermine the view that cognitive aging does not begin until middle to late adulthood. Second, a number of researchers (Baltes & Lindenberger, 1997; de Frias, Lövden, Lindenberger, & Nilsson, 2007; Lövdén & Lindenberger, 2005; McDonald, 2002) have argued that, even though idiosyncratic function-specific cognitive declines may indeed begin in young adulthood, general deficits that pervade many domains of functioning are only prominent in later life. Tucker-Drob (in press a) produced one of the first examinations of the extent to which global patterns of correlated cognitive changes are evident in younger adults. Participants were divided into three groups, the younger group containing adults between 18 and 49 years of age, the middle group containing adults between 50 and 69 years of age, and the older group containing adults between 70 and 95 years of age. A common factor model was fit to longitudinal slopes representing changes in four domains of cognition: fluid reasoning, spatial visualization, episodic memory, and processing speed. The resulting patterns were consistent across age groups, with moderate to large positive loadings on a global change factor in all three groups. Furthermore, constraining the unstandardized factor variances and factor loadings to be equivalent across groups did not significantly decrease model fit—in other words, there was no evidence that the pattern was significantly different across the three groups. These findings suggest that the global and pervasive patterns of cognitive declines that are typically experienced in older adulthood originate in early adulthood.

What Are the Moderators of Cognitive Aging?

One question that is of great interest not only to cognitive aging researchers, but to the public at large, is: *Who* are the people who stave off decline, and how do they differ from those who do not? Here we follow the lead of Hertzog, Kramer, Wilson,

and Lindenberger (2009) and focus on social environments and individual behaviors that have been hypothesized to protect against cognitive declines. We do not consider hypotheses relating chronic illness or unhealthy behaviors (e.g. smoking) to individual differences in cognitive decline, nor do we review work on the roles of nutrition or pharmaceuticals. Instead, we focus on two broad classes of popular hypotheses. The first hypothesis has often been termed the *cognitive reserve* hypothesis. It predicts that advantages afforded by educational and socioeconomic opportunities in early life can serve to slow the rates of aging-related cognitive decline. The second hypothesis has frequently been termed the *"use it or lose it"* hypothesis. It predicts that mental exercise and maintenance of an engaged lifestyle can help to slow the rates of aging-related cognitive declines.

Before reviewing the scientific evidence pertaining to the two above-described hypotheses, it is important to make a conceptual clarification. Relations between hypothesized protective factors and late-life cognitive function might be observed for one of two distinct possible reasons. The first possibility is what Salthouse and colleagues (Salthouse 2006; Salthouse, Babcock, Skovronek, Mitchell, & Palmon, 1990) have referred to as "differential preservation." Differential preservation, which is illustrated in the left panel of Figure 9.4, describes a situation in which individuals who differ in their level of a hypothesized protective factor also predictably differ in their rate of cognitive decline (i.e. the preservation of cognitive function is differential). The second possibility is what Salthouse and colleagues (Salthouse 2006; Salthouse et al., 1990) have referred to as "preserved differentiation." Preserved differentiation, which is illustrated in the right panel of Figure 9.4, describes a situation in which individuals who differ in their level of a hypothesized protective factor begin adulthood at different levels of cognitive ability, but do not differ in their rate of cognitive decline (the differentiation between people is preserved across time). Therefore, under preserved differentiation, the differences that exist between groups at the beginning of adulthood are preserved into later adulthood, but do not widen.

Consider the implications of differential preservation and preserved differentiation for interpreting the finding relating a risk factor (e.g. education) to the incidence rate

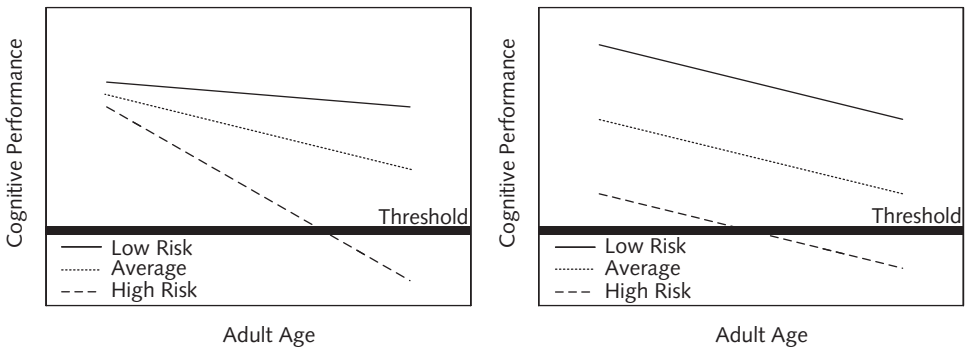

Figure 9.4 Illustration of differential preservation (left) and preserved differentiation (right) scenarios. The horizontal line depicts a diagnostic threshold beyond which the level of cognitive functioning is considered pathological

of dementia, or otherwise clinically severe levels of functioning. Dementias and related disorders are often identified by using cognitive tests: if real-world functioning is deemed to be impaired and performance on the cognitive test falls below a diagnostic threshold, diagnosis is probable (American Psychiatric Association, 2000). An increased risk of dementia is therefore likely to reflect at least one of two general possibilities. The first is that individuals high on the risk factor decline more steeply in their cognitive performance than those low on the risk factor (this possibility reflects differential preservation). The second possibility, however, is that individuals high on the risk factor decline in their cognitive performance at similar rates to those of individuals who are low on the risk factor (a possibility that reflects preserved differentiation), but they begin adulthood at lower levels of cognitive performance, such that they are closer to the diagnostic threshold. To illustrate these possibilities, a threshold is superimposed atop the differential preservation and preserved differentiation patterns in Figure 9.4. It can be seen that those who are high on the risk factor surpass the threshold the earliest, regardless of whether the risk factor is related or not to the rate of cognitive decline. In the preserved differentiation scenario, the risk factor is related to the incidence of dementia simply because high-risk individuals begin adulthood closer to the threshold beyond which their performance is considered clinically severe or pathological. Because of the ambiguity associated with examining prevalence and incidence rates for inferring differential preservation versus preserved differentiation, we only review here studies that examine cognition measured on a continuous scale, and we do not review studies that focus on presence versus absence outcomes.

The *cognitive reserve hypothesis* generally refers to the prediction that those who have experienced more enriched socioeconomic environments during childhood and early adulthood have more resilient cognitive and/or neurobiological architectures, which protect against aging-related cognitive deficits. The number of years of educational attainment is among the most popular indices of such advantages. Multiple versions of the cognitive reserve hypothesis currently exist, and they can generally be classified either as passive models or as active models (Stern, 2009). Passive models are more frequently conceptualized at the neurobiological level. These models generally view high-reserve (i.e. more educated) individuals as having more resilient brains, whose functions are less affected by neurodegeneration than those of low-reserve (less educated) individuals are. One such basis for these models is the hypothesis that high-reserve individuals have more redundant brain networks. Therefore, if a single network is damaged but the redundant network is not, functioning is unaffected. Active models, alternatively, are most often—although not exclusively—conceptualized at the cognitive level. These models generally view high-reserve individuals as better able to compensate for neurodegeneration, through a reorganization of information-processing networks and/or through a shift in reliance on unaffected cognitive processes or knowledge structures to support functions that were previously supported by the now-affected processes. Under active models, high-reserve individuals should have more flexible brain structures, cognitive processes, and/or knowledge structures.

The cognitive reserve hypothesis has frequently been tested by examining the relation between educational attainment and rates of longitudinal cognitive changes.

While some studies have reported statistically significant positive relations, more highly educated individuals exhibiting smaller declines than less educated individuals, many of these studies suffer from major methodological limitations (see Tucker-Drob et al., 2009 for a discussion). The main limitation is that studies have relied upon measures that are not very sensitive to the task of discriminating between individuals at the higher ranges of functioning. Because education is consistently related to levels of functioning at the beginning of a longitudinal study, the change amongst the more highly educated will be harder to detect with crude instruments, and an artifactual positive relation between educational attainment and cognitive change will arise. We therefore emphasize the results from studies that have made use of sensitive cognitive measures. Such studies include those by Christensen et al. (2001), Hofer et al. (2002), Mackinnon, Christensen, Hofer, Korten, and Jorm (2003), Tucker-Drob et al. (2009), and Van Dijk, van Gerven, van Boxtel, van der Elst, and Jolles (2008), all of which have failed to find positive education–cognitive changes relations. We therefore conclude that there currently exists little persuasive evidence that educational attainment (or any factors for which it may act as a surrogate) protects against normative cognitive declines. We do emphasize, however, that there is substantial evidence that those with higher levels of education have higher average levels of cognitive functioning throughout adulthood (likely as the result of preserved differentiation). Educational attainment may therefore still have important real-world implications for cognitive functioning in adulthood, even if it does not protect against aging-related cognitive decline.

The *"use it or lose it" hypothesis*, also known as the mental exercise hypothesis, predicts that those who maintain a mentally engaged and mentally active lifestyle will experience relatively less cognitive decline than those who do not. Mentally stimulating activities that have been hypothesized as protective against cognitive aging include recreational activities such as doing crossword puzzles and playing chess, learning a new skill such as how to play an instrument or speak a foreign language, and having an intellectually demanding job.

Salthouse (2006) has comprehensively reviewed cross-sectional evidence for the "use it or lose it" hypothesis. As he explains, observing that older adults who are more mentally active tend to have higher cognitive functioning is not very informative, because (1) the mental activity–mental ability relation may have existed in childhood and therefore have nothing to do with aging; and (2) mental activity may be an outcome of ability level rather than a determinant of ability level. Examination of mental activity-related differences in aging trajectories is therefore much more informative than examination of simple mental activity-cognitive function correlations. Such examinations of mental activity-related differences in aging trajectories can help to distinguish between the preserved differentiation and differential preservation scenarios with respect to the "use it or lose it" hypothesis. Salthouse (2006) has reviewed a large body of such evidence, comparing pre-existing groups known to engage in different levels of mental activity. One exemplary study (Salthouse et al., 1990) found that architects, who regularly employ spatial reasoning in their day-to-day jobs, exhibited age-related deficits in the visual–spatial test performance comparable to those of unselected adults. Another representative study (Hambrick, Salthouse, & Meinz, 1999) found no statistically significant differences in age-related cognitive

trends as a function of time spent per week completing crossword puzzles. It is of note that there was a large degree of variation in the amount of time spent completing crossword puzzles, with the bottom quartile completing 1.1 hours per week, and the top quartile completing 10.2 hours per week. Salthouse (2006) reviewed work on age differences in cognitive performance as a function of self-reports on the time spent being engaged in cognitively demanding activities (sometimes scaled by the participant's subjective demands of each activity), and additionally as a function of self-reported dispositions toward engaging in cognitively stimulating activities. He concluded that there was little evidence supportive of a differential preservation pattern. In a 2009 paper, Hertzog and colleagues criticized Salthouse's conclusion for its overreliance on cross-sectional data. They cited six longitudinal studies that, they argued, produced evidence consistent with a differential preservation pattern. We note that, for the majority of these studies, the differential preservation pattern only held for small subsets of the hypothesized risk factors and cognitive outcomes examined (and therefore may have been spurious); the cognitive outcomes were measured with tests of questionable validity; or large portions of participants who were in the process of converting to dementia were included. Our view is therefore that there does not currently appear to be persuasive evidence for the differential preservation of cognitive abilities with respect to mental activity in normal healthy adults.

What Can Improve Cognitive Performance in Adulthood?

Related to the question of what individual characteristics and behaviors might moderate the rate of cognitive change, there is also the question of what interventions might be applied to boost an overall level of cognitive performance. Here the question is not whether the rate of cognitive change can be altered, but whether overall performance can be improved. Research on interventions is relevant to individual differences in cognitive aging for at least two reasons. First, individual differences in late-life cognition can arise because some people have undergone an effective (naturally occurring) intervention, whereas others have not. Second, individual differences in late-life cognition can arise because some benefit more from an intervention than others do. While not much research has currently been done on the latter topic to date, we anticipate that this topic will gain more attention with the increasing appreciation of individual differences in cognitive aging, combined with recent methodological developments for examining individual differences in experiments (Muthén & Curran, 1997; Tucker-Drob, in press b). Here we focus on two categories of interventions: (1) cognitive training interventions; and (2) physical activity interventions. Medical and pharmacological interventions are beyond the scope of the current chapter.

Cognitive training interventions

In the history of cognitive aging research, cognitive training interventions have been popular among researchers seeking to determine whether declining cognitive functions in old age can be remediated (see e.g. Schaie & Willis, 1986; Willis & Schaie, 1986, 1994). The Advanced Cognitive Training for Independent and Vital Elderly

(ACTIVE; Ball et al., 2002; Willis et al., 2006) serves as a recent and representative example of some of the latest attempts at improving late-life cognition through training.

Cognitive training interventions have conventionally been based on the premise that older adults can be taught skills and strategies that can be used to increase cognitive performance. In ACTIVE, 2,832 participants were randomized either to a no-contact control condition or to one out of three different cognitive training interventions, each one of which was conducted in small groups of 10, for 60- to 75-minute sessions, over up to six weeks. The memory-training intervention involved learning mnemonic strategies for remembering word lists, sequences of items, text material, and main ideas and details of stories. Application of these mnemonics was practiced on lab-based memory tasks and on everyday memory tasks (such as recalling a list of groceries), which were similar to those used as outcome measures. The reasoning training involved learning strategies designed to identify serial patterns. Application of these strategies was practiced on abstract reasoning tasks and on everyday reasoning tasks similar to those used as outcome measures. Speed of processing training involved learning visual search skills and strategies for identifying and localizing visual information quickly. Participants practiced speeded tasks that varied in complexity on the computer. A subset (60 percent) of the intervention group participants were offered booster training after 11 months. Booster training consisted of four 75-minute sessions over up to three weeks. Outcomes were assessed at pretraining baseline, posttest, one year, two years, three years, and five years. Outcomes included psychometric tests of memory, reasoning, and processing speed, self-reports on activities of daily living, and ecologically face-valid tests of everyday problem-solving, activities of everyday living, and everyday processing speed.

At face value, results from ACTIVE might appear to indicate that the training was a success. Relative to controls, participants improved on the psychometric tests of the abilities on which they were trained (participants trained in memory improved in memory, participants trained in reasoning improved in reasoning, and so forth). Moreover, the differences between control and intervention groups were still detectable after over five years. However, these results are not very surprising, as the skills and strategies taught as part of the training were tailored toward these specific outcomes. For example, participants who received the reasoning intervention were taught strategies to identify the pattern in a letter or word series, and indeed they improved on psychometric measures of letter series and word series completion. We believe that a more interesting and important question is whether the skills transferred, such that performance would improve on psychometric tasks that were not trained, or on ecologically face-valid measures of everyday functions. Results suggest that such a transfer did not occur. Training did not transfer across domains (e.g. participants trained in reasoning did not improve in memory relative to controls), nor did it transfer to objective measures of everyday functioning (e.g. relative to controls, participants receiving training interventions did not improve in their abilities to understand medication directions, to pay bills, or to follow food recipes). It is of note that, at the fifth-year follow-up, participants in the reasoning training group reported less difficulty with everyday tasks relative to controls. Given that this effect was only found on self-report measures of everyday functions but not on objective

measures of everyday functions, it is likely that it represents an effect of training on personal beliefs about functioning, rather than actual functioning. A major challenge for future cognitive training intervention work will be to demonstrate the transfer of benefits to objective indices of cognitive performance and everyday functioning that may not share the same superficial qualities as the tasks on which the training occurred (McArdle & Prindle, 2008).

Physical activity interventions

Over the past decade, results from randomized experiments have provided evidence supportive of a causal effect of aerobic exercise on cognitive function in older adults. A particularly rigorous study on this topic was conducted by Kramer et al. (1999), who randomly assigned 124 previously sedentary older adults to an aerobic walking intervention group or to a stretching and toning control group. They found that, compared to those in the stretching and toning group, those in the walking group exhibited enhanced performance on switching, distracter interference, and response inhibition tasks. Colcombe and Kramer (2003) later identified 18 articles reporting on cognitive change during randomized controlled fitness interventions. They meta-analyzed these studies, which in total included 197 effect sizes for a total of 96 control group participants and 101 exercise group participants. Exercise-related gains were observed in all cognitive domains: executive functioning, controlled processing, spatial visualization, and processing speed, with the largest gains (d = approximately .6) observed in executive functioning, and the smallest gains (d = approximately .2) observed in processing speed. It is not yet clear what mechanisms underlie the cognitive benefits of increased exercise, but some possibilities may include enhanced cerebral blood flow, stimulation of neurotransmitter activity, enhanced hormonal activity or regulation, stabilized mood, or automation of physical functions that would otherwise require effortful cognitive resources. We discuss research on the neurobiological bases of cognitive aging in a later section. However, one interesting possibility is that the mechanisms that underlie exercise-related cognitive benefits might be the mechanisms that degrade with age. In other words, exercise may help to restore functions that deteriorate as a result of normal aging. On the basis of this assumption, one might expect larger exercise benefits for older participants and for those who have exhibited particularly pronounced declines. Finally, of interest is whether these effects are simply immediate, or also result in altered rates of cognitive decline. Unfortunately, there is currently little work on this topic.

How Does Cognitive Aging Relate to Real-World Functioning?

One is likely to wonder about the real-world implications of the rather dramatic age-related decreases in performance on cognitive tasks that occur during adulthood. Because the jobs, decisions, and even everyday activities that people perform in their lives often involve high levels of complexity and sophisticated thought, the conclusion that real-world functioning decreases substantially with old age might appear to be

rather straightforward. Alternatively, studies of the self-appraisals of real-world functioning by older adults, as well as the observations of a number of cognitive aging researchers, suggest that the effects of cognitive aging on real-world function are rather minimal. For example Park (1998, p. 61) has written that "older adults function well and that cognitive declines documented in the lab do not impact as negatively as one would expect on everyday domains of behavior."

Do everyday functions, such as balancing a checkbook, following a food recipe, looking up a telephone number, or understanding medication adherence directions, decline along with cognitive declines in adulthood? These functions have received a great deal of attention from researchers, because they are crucial for independent living, and because failures of these functions (following medication instructions in particular) can have major negative consequences. Interestingly, contradictory findings are often produced by studies in which everyday functions are subjectively measured versus studies in which everyday functions are objectively measured. Self-reports of the subjective difficulty that adults experience in performing daily tasks typically exhibit only very weak relations to age, or to cognitive abilities for that matter. Alternatively, objectively measured performance on ecologically face-valid tests of everyday functions typically exhibit strong relations to age and to cognitive abilities. A recent study by Tucker-Drob illustrates these contradictory findings and produces evidence that may help to resolve them. Tucker-Drob (in press c) analyzed five-year longitudinal data from adults aged 65 years and older, who were living independently and were dementia-free at enrollment. He found that, although self-reports of everyday functions were indeed only weakly related to cognitive abilities and to age, objective, ecologically valid measures of everyday functions were negatively related to age, strongly related to cognitive abilities, and, most importantly, they declined in tandem with cognitive abilities (i.e. individual differences in changes in everyday functions were strongly correlated with individual differences in changes in cognitive abilities). These new results suggest that the reason why the effects of cognitive aging are not apparent on everyday functions is that people are poor at appraising their own levels of functioning, not that cognitive aging and everyday functioning are truly independent.

A related question concerns whether on-the-job performance declines with adult age. Rather than surveying many specific studies of many different types of job performance, we summarize this issue conceptually. Industrial/organizational psychologists have established that the efficient and successful performance of different jobs requires different mixtures of knowledge, skills, abilities, and "other" (Schmitt & Chan, 1998). "Other" includes aspects of personality, such as conscientiousness, extraversion, motivation, curiosity, and interests. Therefore, whether a person's job performance decreases, remains stable, or even increases with age is likely to be determined by a combination of the extent to which that individual changes in his or her levels of knowledge, skills, abilities, and "other," weighted by the extent to which that person's job requires each of these four factors (cf. Salthouse & Maurer, 1996). It is important to appreciate that, as individuals age and their processing abilities decline on average, their experience on the job accumulates. For many jobs, this experience results in the accumulation of knowledge and skills that positively impact

performance, and overall job performance may continue to increase for much of adulthood (Skirbekk, 2004). Alternatively, for jobs that are especially high in cognitive demands, accumulating experience may not be sufficient to offset declines in processing abilities, and job performance may not increase with age, or may even begin to decline early in adulthood. Job performance may also decline early in adulthood for jobs that require cognitive effort but little knowledge or skill. These statements of course simplify the situation, as "other" factors (e.g. personality factors) may also change with age and therefore play roles in age-related changes in job performance.

What Are the Neurobiological Substrates of Individual Differences in Cognitive Aging?

A complete understanding of the factors that underlie individual differences in cognitive aging will certainly require an understanding of individual differences in changes in the neurobiological factors that underlie cognitive performance. Indeed, numerous aspects of brain physiology have been found to change with age (Dennis & Cabeza, 2008; Raz et al., 2005; Raz & Rodrigue, 2006). Age-related decreases in overall brain volume and in regional brain volumes have been reported for both cross-sectional and longitudinal data. Gray matter shrinkage appears to be most pronounced for the frontal lobes, followed by the parietal lobes, and then by the medial temporal lobes (Dennis & Cabeza, 2008). Age-related degradation of white matter volume and integrity and of dopamine function appear to be similarly disproportionately concentrated in the frontal brain regions. There is evidence that each of these measures is correlated with cognitive function in older adults, suggesting that they are indeed plausible substrates of cognitive aging. However, future work will need to be done to link individual differences in longitudinal changes in the various neurobiological indices with those in various cognitive functions. Moreover, given the evidence that different cognitive functions change together (Tucker-Drob, in press a) and emerging evidence that different aspects of brain anatomy change together (Raz et al., 2005), it will be important to take multivariate approaches to the neurobiology–cognition link, such that commonalities among predictors and among outcomes can be taken into account.

What Are the Genetic Risk Factors for Cognitive Aging?

A complete treatment of the topic of individual differences in cognitive aging necessitates that some attention be paid to the extent to which between-person genetic variation underlies individual differences in cognitive aging trajectories. As Turkheimer (2000) has stated, the finding that psychological traits are heritable is so pervasive that it can be considered "the first law of behavioral genetics." Cognitive abilities in adulthood are no exceptions to this law. In fact, genetic influences have been estimated to account for as much 80 percent of individual differences in cognitive

abilities during later adulthood (Pederson, Plomin, Nesselroade, & McLearn, 1992). However, the finding that cognitive ability is highly heritable in adulthood is not a direct indication of the extent to which cognitive aging is genetically influenced. That is, because individual differences on cognition in adulthood reflect a combination of individual differences in cognitive development in addition to individual differences in cognitive aging, the heritability of cognitive ability potentially reflects the combination of genetic influences on development and genetic influences on cognitive aging. As such, it is much more informative to examine the heritability of individual differences in longitudinal cognitive changes that actually occur during adulthood. Only a few longitudinal twin studies of cognitive aging exist, and conclusions with respect to the heritability of cognitive changes are therefore somewhat tentative. Some of the best data come from the Swedish Adoption/Twin Study of Aging, for which Reynolds, Finkel, McArdle, Gatz, Berg, and Pedersen (2005) fit quadratic growth curve models to 13-year longitudinal data on 10 different cognitive variables representative of either verbal, spatial, memory, or processing speed abilities. The median reported heritability of the linear component of change was 16 percent, and of the quadratic component of change the median reported heritability was 41 percent. In the same data, Finkel, Reynolds, McArdle, and Pedersen (2005) reported the heritability of the linear components of change in verbal ability, spatial ability, memory, and processing speed composite scores to be 5 percent, 19 percent, 23 percent, and 32 percent respectively, and heritabilities for the quadratic components to be 5 percent, 57 percent, 69 percent, and 82 percent respectively. While these heritability estimates of aging-related cognitive changes are somewhat lower than the corresponding estimates for levels of cognitive functioning, there still appears to be ample room for genes to contribute to individual differences in cognitive aging.

A number of specific genetic polymorphisms have been identified as potential risk factors for cognitive decline. McGue and Johnson (2008) provide an accessible review of research on candidate genes for aging-related cognitive changes. As they explain, the gene for which the most robust and compelling evidence exists for a link to late-life cognition is the Apolipoprotein E (APOE) gene, which has been implicated in lipid transport and neuronal repair. The ε4 allele of APOE, which is present in approximately 15 percent of individuals with European ancestry, has been identified as a potential risk for cognitive decline. The ε4 allele of APOE has been robustly associated both with the age of dementia onset and with normal-range variation in cognitive functioning during later adulthood. Significant meta-analytic associations between APOE variation and general cognitive ability, episodic memory, and executive functioning in cognitively intact adults have been reported (Small, Rosnick, Fratiglioni, & Bäckman, 2004), and evidence is accumulating from studies to the effect that APOE is related to the rate of cognitive decline (Bretsky, Guralnik, Launer, Albert, & Seeman, 2003; Deary et al., 2004; Hofer et al., 2002). A number of other genetic polymorphisms have been proposed as candidates for risk of cognitive decline. These include Angiotensin I Converting Enzyme (ACE; implicated as a risk factor for hypertension), Catechol-O-Methyltransferase (COMT; involved in the degradation of released catecholamines), and Methionine Synthase (MTR; involved in the metabolism of homocysteine). McGue and Johnson (2008), however, conclude that

current evidence for a systematic association between these genes and late-life cognitive functioning is inconsistent.

It is important to note that the population-genetic and molecular–genetic research on cognitive aging that has been conducted to date has primarily been concerned with the main additive effects of genes and has paid comparatively little attention to the possibilities of gene-by-environment interaction (i.e. of genetic influences varying as a function of specific environmental conditions) and of gene–environment correlation (i.e. of different environmental protective or risk factors varying systematically with different individual genotypes). There is, however, growing emphasis on gene-by-environment interaction and on gene–environment correlation in current research and theory (Deater-Deckard & Mayr, 2005; Shanahan & Hofer, 2005).

Conclusions, Outlook, and Future Directions

Given that the ultimate goal of research in the psychological sciences is to understand, and perhaps even ultimately to affect, processes that occur for individuals, it is appropriate that research on cognitive aging is moving toward an increased appreciation of individual differences. In this chapter we have presented and summarized the progress that has been made toward answering seven major questions that we believe to be fundamental to the study of individual differences in cognitive aging. Much progress has already been made, but the answers to the questions are far from complete. Here we describe what we believe to be the next major steps that need to be taken in this important area of inquiry.

First, an increasing focus on individual differences in cognitive aging will entail an increased reliance on longitudinal data derived from sensitive measures. High-quality longitudinal data, paired with appropriate analytical methodologies for modeling change and for removing retest effects, will serve as the basis for better characterizing the progression of cognitive aging and for robustly identifying its correlates and consequences.

Second, in light of recent findings that large proportions of individual differences in aging-related changes in many different cognitive functions are overlapping, it will be important for future work to integrate the diverse findings and models that have been established for individual tasks and functions across those tasks and functions. This will entail increased collection of multivariate data and increased application of multivariate methodologies.

Third, there is a need to integrate findings from cognitive aging with findings from cognitive development. Although we do not believe that there is currently strong evidence consistent with differential preservation patterns with respect to popularly hypothesized moderators of cognitive aging, we note that there are ubiquitous socoidemographic correlates of levels of cognitive function at all stages of adulthood (i.e. preserved differentiation is well supported for many popularly hypothesized risk factors). It will therefore be crucial to understand the developmental processes that give rise sociodemographic disparities in cognitive functioning in childhood, which in turn persist throughout adulthood. In fact, it has even been suggested that the most cost-effective interventions to boost adult levels of cognitive

functioning are likely to be those that target cognitive development during childhood (Heckman, 2006).

Fourth, crucial to intervention work will be the construction and evaluation of interventions that do not simply have proximal effects on test performance, but which reliably result in far transfer to many different abilities, and most importantly to real-world outcomes. Training adults in specific strategies that can be applied to specific sorts of tasks is not likely to produce gains that generalize to many functions. For cognitive training interventions, far transfer may be more likely to occur when general skills and functions, rather than specific strategies, are targeted.

Fifth, it will be crucial to empirically link the neurobiological changes that are thought to underlie cognitive aging with actual cognitive changes. That neurobiological variables degrade on average with age does not necessarily imply that such declines underlie aging-related cognitive declines, even if the neurobiological variables correlate with cognitive functions at a given period of time. Rather, it will be crucial to examine the longitudinal relations between individual differences in neurobiological variables and in cognitive variables. Longitudinal relations can take the form of (1) level of one variable preceding and predicting change in another variable; (2) changes in two or more variables being concurrently related; or (3) change in one variable preceding and predicting later change in another variable. Moreover, given that multiple neurobiological variables change with age, it will be important to examine the unique influences of different neurobiological variables on cognition, controlling for other neurobiological variables. This will help to map different aspects of cognitive aging to their specific neurobiological substrates.

Sixth, it is clear that conceptualizing genetic influences as uncorrelated and additive with environmental influences on cognition grossly oversimplifies reality. Future population–genetic and molecular–genetic work should test specific hypotheses regarding gene-by-environment interaction and the gene–environment correlation. The existence of gene-by-environment interaction may help to explain why candidate environmental risk factors are inconsistently linked with cognitive decline. That is, the relation between risk factor and outcome may be different for different people.

Finally, while the current chapter has primarily treated cognitive aging as an outcome in need of explanation, there is much work on how individual differences in cognitive functioning and cognitive aging predict individual differences in health and epidemiological outcomes. *Why* individual differences in cognitive functioning and in cognitive change relate to individual differences in health outcomes is a fundamental issue, which will need to be resolved in future research (Deary, 2008).

Note

1 $r_{full\ sample}$ refers to the correlation between age and the ability in the full sample. $r_{MMSE \geq 27}$ = refers to the correlation between age and the ability when individuals with scores below 27 out of 30 on the Mini Mental State Examination are excluded.

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