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Individual Differences in Cognitive Aging

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Handbook of Individual Differences

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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)
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> "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)

There are seven questions that we believe to be foundational to this burgeoning area of inquiry. They 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?* 6) *What are the Neurobiological Substrates of Individual Differences in Cognitive Aging?* and 7) *What are the genetic risk factors for cognitive aging?* In this chapter we summarize the progress that has been made towards answering each of these questions and discuss prospects for future research. First, we describe the basic phenomenon in question at the population level.

When Does Cognitive Aging Begin and For What Cognitive Abilities?

Before addressing questions of 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 declines only occur for memory, do not appear until later in life, and only transpire for the small segment of the population that experience neurological disease. However, there is now strong evidence that cognitive declines occur for a number of different abilities besides memory (e.g. reasoning, speed of processing, and spatial visualization), begin in early adulthood, and occur in 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 1200 rural New England residents between 10 and 60 years of age. Jones and Conrad observed that on nearly all of the subtests 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* (i.e. antonym vocabulary) subtest, and the *General Information* subtest, 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' 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 knowledge, and/or highly automatized forms of processing (a.k.a. procedural knowledge), mean levels of performance peak in middle adulthood after which point they remain relatively stable. These trends are illustrated in Figure 1, which is based on data from the Virginia Cognitive Aging Project at the University of Virginia (VCAP; N = 3,560; Salthouse, 2004b; Salthouse, Pink, & Tucker-Drob, 2008; Tucker-Drob, 2010a; Tucker-Drob & Salthouse, 2008; Tucker-Drob & Salthouse, 2009), for 16 tests representative of 5 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 trends are not attributable to age trends in the prevalence of dementia, as the correlations between the abilities and age are very similar before and after individuals with scores below 27 out of 30 on the Mini Mental State Examination (a popular dementia screening instrument; Fosltein, Folstein, & McHugh, 1975) are excluded. For composite scores representing each ability, they are: Spatial Visualization ($r_{full\ sample}$ = -.474, $r_{MMSE\geq27}$ = -.477), Abstract Reasoning ($r_{full\ sample}$ = -.482, $r_{MMSE\geq27}$ = -.477), Episodic Memory ($r_{full\ sample}$ = -.433, $r_{MMSE\geq27}$ = -.427), Processing Speed ($r_{full\ sample}$ = -.629, $r_{MMSE\geq27}$ = -.627), and Verbal Knowledge ($r_{full\ sample}$ = .245, $r_{MMSE\geq27}$ = .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

[1] $r_{full\ sample}$ refers to the correlation between age and the ability in the full sample. $r_{MMSE\geq27}$ = 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.

adulthood. Because logistic issues make it very difficult for longitudinal studies to span an entire lifetime, longitudinal evidence typically comes from what have been 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 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 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, in press - a). 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 (in, e.g., nutrition or education). A second potential validity threat is nonrandom selection. If older participants in a cross-sectional study tend to be more positively selected than are younger participants, 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 to either disinterest or a relation between cognitive functioning and illness or death; Lindenberger, Singer, & Baltes., 2002), which would lead to an

underestimation 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.

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 (Yang, Schullhofer-Wohl, Fu, & Land, 2008; Baltes, Reese, & Nesselroade, 1977; Baltes & Schaie, 1976; Horn & Donaldson, 1976), 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. nonrandom 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 Ronnlund 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 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 et al, 1997; Le Bourg, 2004), in which the threats to validity that are common to studies with human participants are not applicable. Based on these observations, we believe that there 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 that they provide about individual differences in change, particularly when the 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 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 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 four year period and concluded that adult age was related to increased variability in reaction time, memory, and reasoning, but not verbal knowledge. Based on data from the WAIS-III standardization sample, and scaling standard deviations relative to mean performance (which we are critical of, because it confounds variation with performance level), Ardilla (2007, p. 1010) similarly concluded 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 variation in speed, reasoning, and memory scored 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 & 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 (2010) was unable to find clear evidence for systematic cross-sectional age trends in between-person variation in cognitive test performance. Based on 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 (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 the age differences in variation approach, and the fact that the approach is a rather indirect way of gauging individual differences in cognitive aging to start with, 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 (Crobach & Furby), new growth curve modeling and latent difference score modeling approaches enable researchers to produce estimates of variation in changes that are theoretically error-free. Based on these new methods, there is accumulating evidence for systematic and statistically significant variation in longitudinal 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 et al (2002), Tucker-Drob, Johnson, & Jones (2009), and Tucker-Drob (2010). Each study has reported significant variation in longitudinal slopes independent of variation in practice effects

(interestingly, variation in the practice effects was in many cases not statistically significant). What is the magnitude of this variation? Tucker-Drob (2010) 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%, 9%, 13%, and 9% 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 this variation 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 recent studies by Ferrer et al. (2005), Wilson et al. (2002), Tucker-Drob et al. (2009), and Tucker-Drob (2010), 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 these deficits each 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) that each influence 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 datasets (Salthouse, 2004b; Salthouse, 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 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.

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 et al. (2003), Lemke & Zimprich (2005), Sliwinksi & Buscke (2004), and Sliwinski, Hofer, & Hall (2003). Ferrer et al. (2005), Tucker-Drob, Johnson, & Jones (2009), Tucker-Drob (2010a), and Wilson et al. (2002) have reported that these correlations largely persist when practice effects are statistically controlled for.

Five studies (Hertzog et al., 2003; Lindenberger & Ghisletta, 2009; Reynolds et al., 2002; Tucker-Drob, 2010a; 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% and 60% of individual differences in cognitive changes. Tucker-Drob (2010a), has moreover demonstrated that a hierarchical factor model can be fit to longitudinal cognitive changes. In such a hierarchical factor model, approximately 43% of individual differences in longitudinal changes in 12 tests of cognitive processing from VCAP (the same tests depicted in Figure 3) could be accounted for by a domain general change factor, 35% could be accounted for by domain specific (reasoning, spatial visualization, memory, or processing speed) factors, and the remaining 22% 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 both 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 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 health normal young adults. Alternatively, establishing that similar patterns of individual differences of change pertain to younger and older adults would suggest that the meaning of change does not differ with age (c.f. Salthouse, in press-b), and therefore would undermine perspectives that cognitive aging does not begin until middle to late adulthood. Second, a number of researchers (de Frias et al., 2007; Baltes & Lindenberger, 1997; Lövdén and 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 inlater life. Tucker-Drob (2010a) 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 97 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. 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 likely 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 that are able to stave off decline, and how do they differ from those that do not? Here we follow the lead of Hertzog et al. (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 early life educational and socioeconomic opportunities can serve to slow the rates of aging-related cognitive decline. The second hypothesis 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 (2006; Salthouse, Babcock, Skovronek, Mitchell, & Palmon, 1990) has referred to as differential preservation. Differential preservation,

which is illustrated in the left panel of Figure 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 (2006; Salthouse et al., 1990) has referred to as preserved differentiation. Preserved differentiation, which is illustrated in the right panel of Figure 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 of dementia, or otherwise clinically severe levels of functioning. Dementias and related disorders are often identified 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 for 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 (i.e. differential preservation). The second, however, is that individuals high on the risk factor decline in their cognitive performance at similar rates to those who are low on the risk factor (i.e. preserved differentiation) but begin adulthood at lower levels of cognitive performance, such that they are closer to the diagnostic threshold. To illustrate this latter possibility, a threshold is superimposed atop the differential preservation and preserved differentiation patterns in Figure 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 to rate of cognitive decline. In the preserved differentiation scenario, the risk factor is related to dementia incidence 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 studies that examine cognition measured on a continuous scale here, 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 that protect against the aging-related cognitive deficits in adulthood. 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 as either passive models or 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 are those of low reserve (less educated) individuals. 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, models 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 most frequently been tested by examining the relation between educational attainment and rates of longitudinal cognitive changes. While some studies have reported statistically significant relations with higher educated individuals exhibiting smaller declines than less educated individuals, many of these studies suffer from major methodological limitations (see Tucker-Drob, Johnson, & Jones, 2009 for a discussion). The main limitation is that studies have relied upon measures that are not very sensitive to 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. We therefore emphasize the results from studies that have made use of sensitive cognitive measures. Such studies include those by Christensen et al. (2001), Van Dijk et al. (2008), Hofer et al., (2002), Mackinnon et al., (2003), and Tucker-Drob et al. (2009), 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 function have higher average levels of cognitive function 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 cognitive change.

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 function is not very informative because a) the mental activity-mental ability relation may have existed in childhood and therefore have nothing to do with aging, and be b) 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. Such examinations 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 comparable age-related deficits in visual-spatial test performance as unselected adults. Another representative study (Hambrick, Salthouse, & Meinz, 1999) who 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) additionally reviewed work on age differences in cognitive performance as a function of self reports of time spent engaged in cognitive demanding activities (sometimes scaled by the participant's subjective demands of each activity), and additionally as a function of self-reported dispositions towards 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 may have therefore 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 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 rate of cognitive change, is 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 more than do others. While not much research has currently been done on the latter topic to date, we anticipate that this topic will gain more attention with increasing appreciation of individual differences in cognitive aging combined with recent methodological developments for examining individual differences in experiments (Muthén & Curran, 1997; Tucker-Drob, 2010b). 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; Willis & Schaie, 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 to either a no contact control condition, or one of three different cognitive training interventions, each of which was conducted in small groups in ten 60 to 75 minute sessions over up to six weeks. The memory training intervention involved teaching 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 task and everyday memory tasks (recalling a list of groceries) similar to those used as outcome measures. The reasoning training involved teaching strategies to identify serial patterns. Application of these strategies was practiced on abstract problem reasoning tasks and everyday reasoning tasks similar to those used as outcome measures. Speed of processing training involved teaching visual search skills and strategies for identifying and locating visual information quickly. Participants practiced speeded tasks that varied in complexity on the computer. A subset (60%) of 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 pre-training baseline, posttest, one year, two years, three years, and five years. Outcomes included psychometric tests of memory, reasoning, and processing speed, self reports of activities of daily living, and ecologically face valid tests of everyday problem solving, activities of everyday living, and everyday processing speed.

At face, 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 (i.e. participants trained in memory improved in memory, participants trained in reasoning improved in reasoning, and so forth). Moreover, these 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 towards 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 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 improved on psychometric tasks that were not trained, or on ecologically face-valid measures of

everyday functions. Results suggest that such 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. participants receiving training interventions did not improve in their abilities to understand medication directions, pay bills, or follow food recipes relative to controls). It is of note that, at the five 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 objective measures of everyday functions, it is likely that the effect 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 do 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 or a stretching and toning control condition. They found that compared to those 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 whatever mechanisms underlie the exercise benefits might be those mechanisms that degrade with age. In other words, exercise may help to restore functions that deteriorate as a result of normal aging. Based on this assumption, one might expect larger exercise benefits for older participants and 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 by 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 those 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 (2010c) analyzed five year longitudinal data from adults 65 years of age and older who were living independently and 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 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 that the effects of cognitive aging are not apparent on everyday

functions is because people are poor at appraising their own levels of functioning, not because 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 efficient and successful performance of different jobs require 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 experiences 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 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 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 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 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, 2010a) and emerging evidence that different aspects of brain anatomy change together (Raz et al., 2005) it will be imperative 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% 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, & Pedersen (2005) fit quadratic growth curve models to thirteen year longitudinal data on ten 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%, and of the quadratic component of change the median reported heritability was 41%. In the same data, Finkel, Reynolds, McArdle, & Pedersen (2005) reported the heritability of the linear components of change in verbal, spatial, memory and processing speed composite scores to be 5%, 19%, 23%, and 32% respectively, and heritabilities for the quadratic components to be 5%, 57%, 69%, and 82% respectively. While these heritability estimates of aging-related cognitive changes are somewhat lower than 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 polymorphism 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 Apolipoprotien E (APOE) gene, which has been implicated in lipid transport and neuronal repair. The ε4 allele of APOE, which is present in approximately 15% of individuals with European ancestry, has been identified as a potential risk for cognitive decline. Robust associations have between the ε4 allele of APOE and both the age of dementia onset, and 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 there is accumulating evidenc from studies that APOE is related to rate of cognitive decline (Bretsky et al., 2003; Deary et al., 2004; Hofer et al., 2002). A number of other genetic polymorphisms that have been proposed as candidates for risk of cognitive decline. These include Angiotensin I Converting Enzyme (ACE; implicated as a risk factor for hypotertension), Catechol-O-methyltransferase (COMT; involved in the degredation 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. genetic influences varying as a function of specific

environmental conditions) and gene-environment correlation (i.e. different environmental protective or risk factors varying systematically with different individual genotypes). There is, however, a growing emphasis on gene by environment interaction and gene-environment correlation in current research and theory (Deater-Deckard & Mayr, 2005; Shanahan and Hofer, 2005).

Conclusions, Outlook, & Future Directions

Given that the ultimate goal of research in the psychological sciences is to understand, and perhaps even ultimately affect, processes that occur for individuals, it is appropriate that research on cognitive aging is moving towards an increased appreciation of individual differences. In this chapter we have presented, and summarized the progress that has been made, towards 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 removing retest effects, will serve as the basis for better characterizing the progression of cognitive aging, and 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 or 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 those from cognitive development. Although we do not believe that there is currently strong evidence supportive of 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 man 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 affects of 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 relating 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 cognitive variables. Longitudinal relations can take the form of level of one variable preceding and predicting change in another other variable, changes in two or more variables being concurrently related, or 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 tests specific hypotheses regarding gene-by-environment interaction and 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 an outcome in need of explanation, there is much work on how these individual differences in cognitive aging predict individual differences in health and epidemiological outcomes (see Deary Chapter for an overview). *Why* individual differences in cognitive functioning and cognitive change relate to individual differences in health outcomes is a fundamental issue that will need to be resolved in future research (Deary, 2008).

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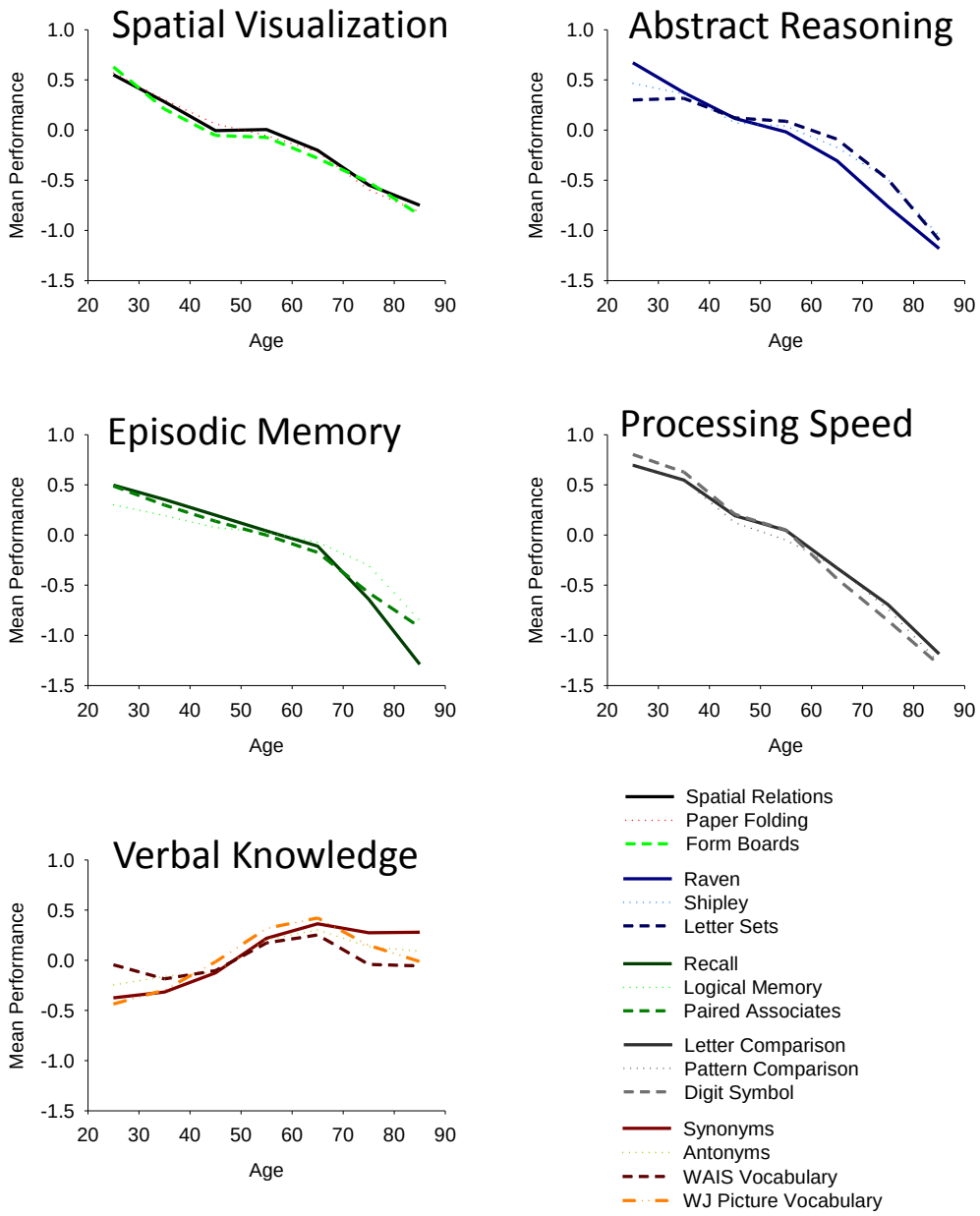
Figure Captions

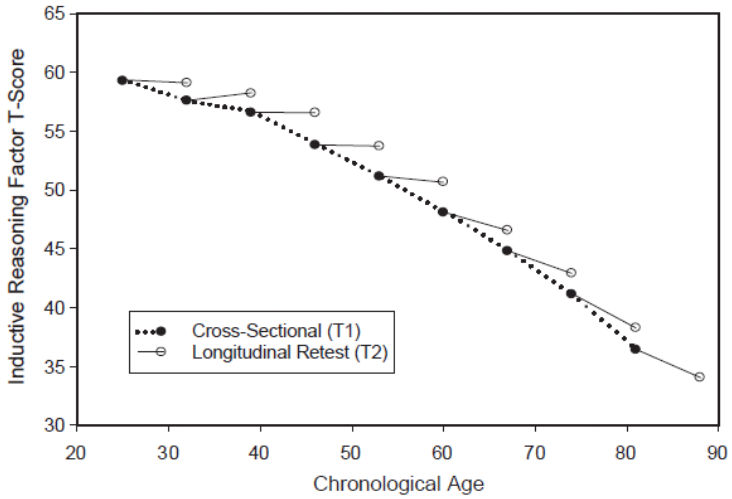
Figure 1. Cross-sectional age trends from the Virginia Cognitive Aging Project at the University of Virginia (VCAP; N = 2,541). All variables have been standardized to have a mean of 0 and standard deviation of 1 in the entire sample.

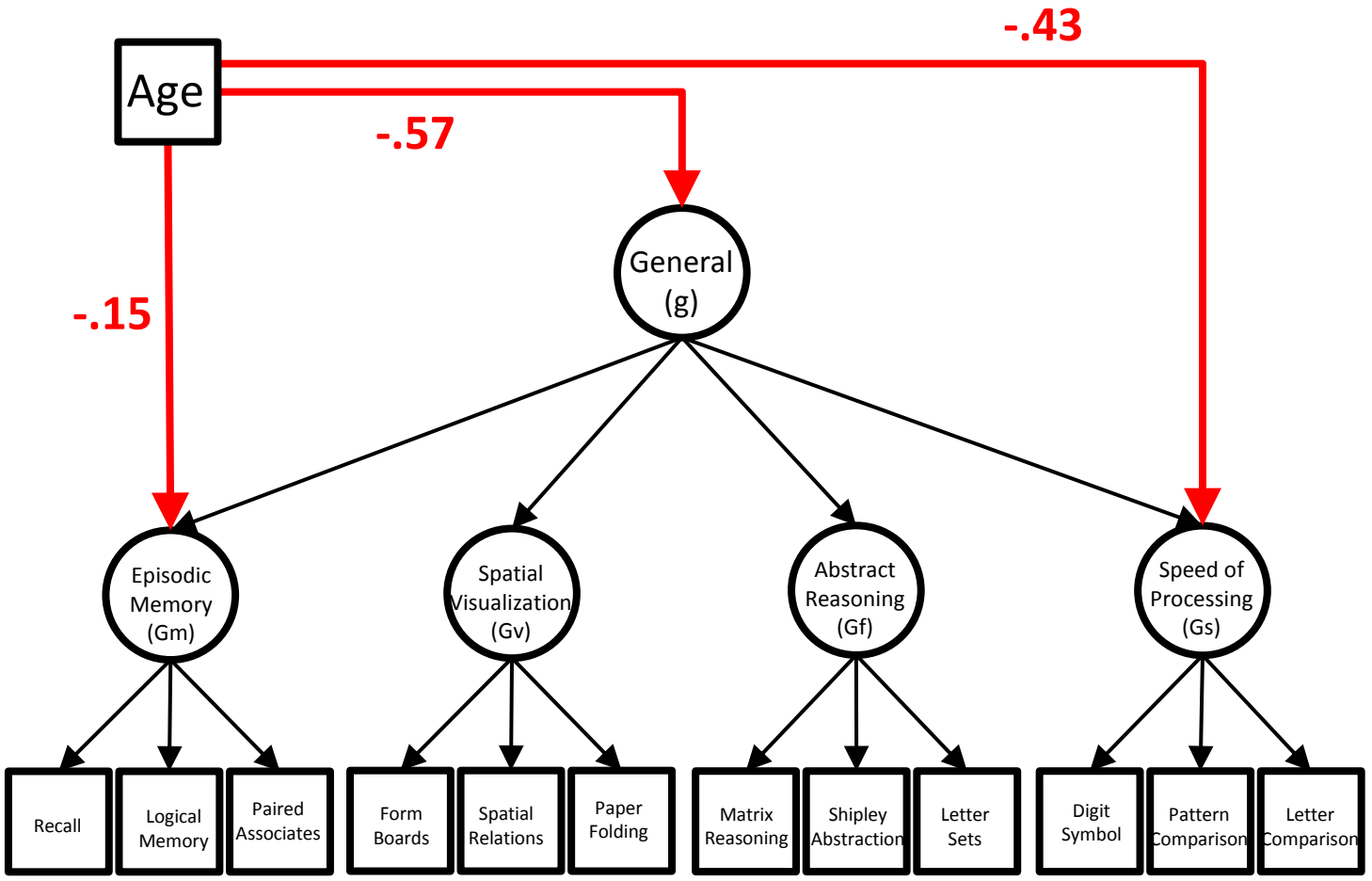
Figure 2. Cross-sectional and Longitudinal age trends in Inductive Reasoning from the Seattle Longitudinal Study (reproduced from Salthouse, 2005). The factor has been standardized to have a mean of 50 and standard deviation of 10 in the entire sample.

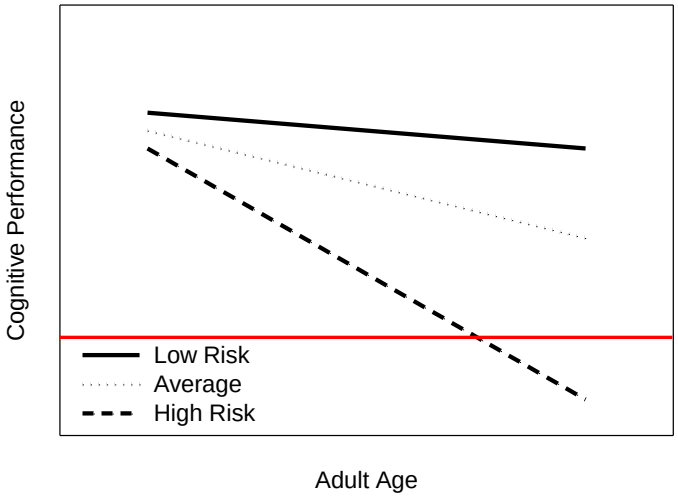
Figure 3. Localizing cross-sectional aging-related differences in a hierarchical structure.

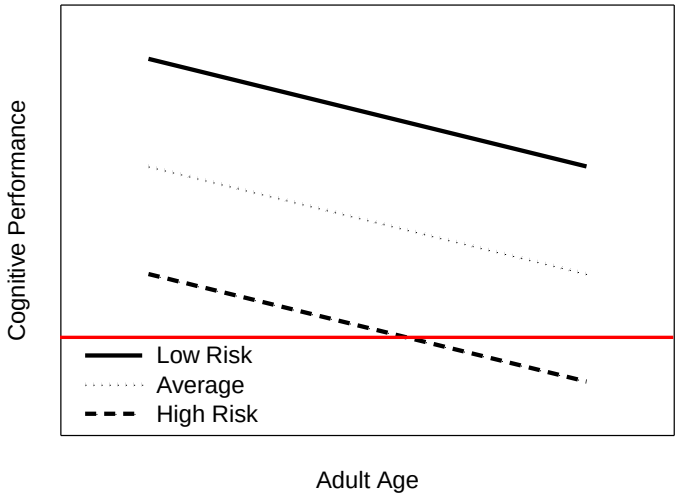
Figure 4. Illustration of differential preservation (left) and preserved differentiation (right) scenarios. The horizontal line depicts a diagnostic threshold beyond which the level of cognitive functioning is considered pathological.


Spatial Visualization
Abstract Reasoning
Episodic Memory
Processing Speed
Verbal Knowledge
Mean Performance
Age
1.0
0.5
0.0
-0.5
-1.0
-1.5
20
30
40
50
60
70
80
90
Spatial Relations
Paper Folding
Form Boards
Raven
Shipley
Letter Sets
Recall
Logical Memory
Paired Associates
Letter Comparison
Pattern Comparison
Digit Symbol
Synonyms
Antonyms
WAIS Vocabulary
WJ Picture Vocabulary


Inductive Reasoning Factor T-Score
65
60
55
50
45
40
35
30
20
30
40
50
60
70
80
90
Chronological Age
Cross-Sectional (T1)
Longitudinal Retest (T2)


Age
-.43
-.57
-.15
General (g)
Episodic Memory (Gm)
Spatial Visualization (Gv)
Abstract Reasoning (Gf)
Speed of Processing (Gs)
Recall
Logical Memory
Paired Associates
Form Boards
Spatial Relations
Paper Folding
Matrix Reasoning
Shipley Abstraction
Letter Sets
Digit Symbol
Pattern Comparison
Letter Comparison


Cognitive Performance
Low Risk
Average
High Risk
Adult Age


Cognitive Performance
Low Risk
Average
High Risk
Adult Age