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Textbook of Geriatric Psychiatry >

## **Chapter 7. Psychological Aspects of Normal Aging**

## **PSYCHOLOGICAL ASPECTS OF NORMAL AGING: INTRODUCTION**

Research on psychological aspects of normal aging is now mature and middle-aged. Major longitudinal studies have collected data for up to 50 years of repeated observations, versions of the Handbook of the Psychology of Aging and chapters reviewing the psychology of normal aging have been written for up to 30 years, and the handbook is now in its 6th edition. In this chapter we take a life span approach that is focused on adult development and aging (Siegler 2007a). Thus, it is possible to cite previously published chapters on the normal psychology of aging for geriatric psychiatry (Poon and Siegler 1991; Siegler 1980; Siegler and Poon 1989; Siegler et al. 1996, 2004) and to focus here on the exciting data that recently have become available from mature longitudinal studies and from cross-sectional studies in cognition and neuropsychology, as well as personality and behavioral medicine, that routinely cross disciplinary boundaries. Since the last edition of this textbook, a series of edited volumes have presented reviews in areas of the psychology of normal aging (Birren and Schaie 2006; Carstensen and Hartel 2006; Costa and Siegler 2004) and of health and aging (Aldwin et al. 2007; Markides 2007); these reviews have updated the detailed information presented in the previously published chapters cited above. Attention to how ethnic factors may shape normal psychology of aging are now well recognized (Jackson et al. 2004), and ethnic variations in dementia are being considered (Dilworth-Anderson et al. 2005a, 2005b), as are discussions of cognitive changes with aging (integrated with neuroimaging and neurosciences [Madden and Whiting 2004; Pierce et al. 2004]), including work in motivation, emotion, and social functioning (Carstensen et al. 2006). Summaries from these recent volumes are presented in this chapter.

These summaries will allow us to use the pages allotted to highlight new findings from ongoing research programs. We start with the Seattle Longitudinal Study (SLS) (Schaie 1996, 2005), which focuses on understanding adult intellectual development at its core with a much broader and more complete picture of multiple cohorts of aging persons ages 18–88. Next we discuss findings from the Georgia Centenarian Studies (Poon et al., in press), a series of studies of the extremely aged with comparison populations in their 60s and 80s, presenting data primarily on cognition, personality, coping, and the role of health status and psychological functioning. As we move to studies of personality, we present 40-year follow-up data from the UNC Alumni Heart Study (Brummett et al. 2006b; Siegler 2007b), which examine whether young adult measures predict midlife status and whether detailed measures during midlife help explain health and survival as members of the baby boom cohort reach their age 60 transition. We then look at personality predictors of midlife hypertension (Siegler 2007b) and review findings from the Maine-Syracuse Studies of the impact of hypertension on cognitive and neuropsychological measures (Dore et al. 2007; Elias et al. 2004). We also review some new findings in the coping literature and consider the major common stressor of caregiving as a way to illustrate important research in ethnic differences and models of stress that relate psychosocial variables to disease outcomes.

One might reasonably ask why such a chapter on normal psychology of aging is still needed in a modern 21st-century view of geriatric psychiatry. Clinicians will always need to know the limits of expectable behavior with age in terms of their own expectations as well as expectations of patients and their families. With the benefit of longitudinal findings and particular attention to what we have learned from centenarian studies, we hope to provide a useful set of benchmarks.

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#### INTELLECTUAL AND COGNITIVE DEVELOPMENT

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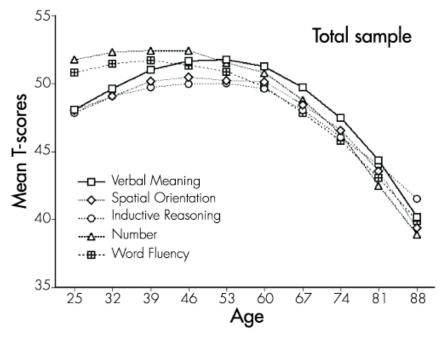
# **Course of Adult Intellectual Development**

The development of intellectual competence in childhood and adolescence follows a fairly uniform path, with new stages of competence and differentiation of functioning occurring within a relatively narrow age band. By contrast, there are widely divergent individual trajectories over the life course of adult intelligence. However, over the 50-year course of the SLS (Schaie 1996, 2005), sufficient evidence has been gathered to reach some rather definitive conclusions on a number of core questions:

Does intelligence change uniformly through adulthood, or are there different life course ability patterns? The answer remains quite unambiguous: Uniform patterns of developmental change across the entire ability spectrum are not observed for the tests actually given or for the inferred latent ability constructs. Hence, it is only fair to warn those who would like to assess change in intellectual competence by means of an omnibus IQ-like measure that such an approach will not be very helpful to either thoughtful clinicians or basic researchers. Such global measures have little practical utility in monitoring changes (or differences) in intellectual competence for individuals or groups.

From the extensive longitudinal data on the primary mental abilities used in the SLS, it can be concluded that the abilities of verbal meaning (recognition vocabulary), spatial orientation, and inductive reasoning reach a peak plateau in midlife from the 40s to the early 60s, whereas number and word fluency peak earlier and show very modest decline beginning in the 50s. The steepness of late-life decline is greatest for number and least for the reasoning ability. Verbal meaning declines last but also shows steeper decline than the other abilities from the 70s to the 80s (see Figure 7–1). More limited data on the multiply marked latent construct estimates (obtained only in the fifth through seventh study cycles) suggest that a shift in peak ages of performance has been seen and is continuing, and that we now see these peaks occurring in the 50s for inductive reasoning and spatial orientation and in the 60s for verbal ability and verbal memory. By contrast, perceptual speed peaks in the 20s and numeric ability in the late 30s. Even by the late 80s, declines for verbal ability and inductive reasoning are modest, but they are severe in very old age for perceptual speed and numeric ability, with spatial orientation and verbal memory in between (see Figure 7–2).

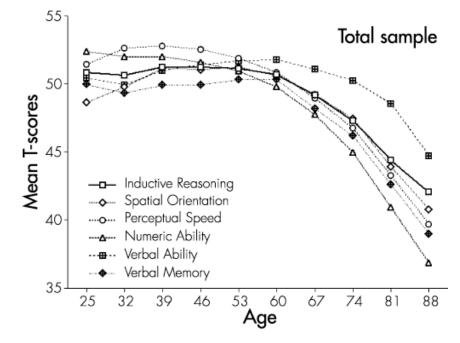
FIGURE 7–1. Longitudinal age changes for the primary mental abilities.



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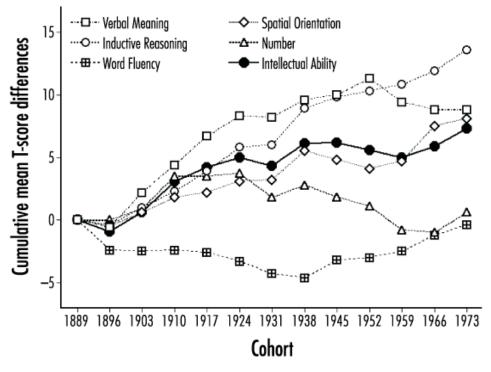
*Source.* Schaie KW: *Developmental Influences on Adult Intelligence: The Seattle Longitudinal Study.* New York, Oxford University Press, 2005, p. 127. Reprinted with permission.

At what age is there on average a reliably detectable decrement in ability, and what is its magnitude? For some ability markers, statistically significant but extremely modest average changes have been observed in the 50s. Nevertheless, it should be stressed that individual decline before age 60 is likely to represent a symptom of or a precursor to neuropathological age changes. On the other hand, it is clear that by the mid-70s significant *average* decrement can be observed for all abilities and that by the 80s average decrement is severe except for verbal ability. In the SLS, statistically significant decrement was found for number and word fluency by age 60 and for space and reasoning by age 67, but for verbal meaning only by age 81. At the latent construct level, statistically significant decrement is first observed by age 60 for spatial ability, numeric ability, and perceptual speed; by age 67 for inductive reasoning; and by age 74 for verbal ability and verbal memory.

The SLS data suggest that it is during the period of the late 60s and 70s that many people begin to experience noticeable ability declines. Even so, it is not until the 80s are reached that the average older adult will fall below the middle range of performance for young adults. Hence, it turns out that for decisions relating to the retention of individuals in the workforce, chronological age is not a useful criterion for groups and is certainly not useful for individuals. This conclusion has of course been the rationale for largely abandoning mandatory retirement in the United States.

What are the patterns of generational differences, and what is their magnitude? The facts of individual aging must also be considered within the context of profound changes over time in environmental and social support systems. In the SLS, the impact of these changes on intellectual development has been documented by charting cohort (generational) differences on the intellectual performance measures. These studies have clearly demonstrated that there are substantial generational trends in intellectual performance. The form of these generational trends has been positive for verbal meaning, space, and reasoning, but it is concave for number (with peak performance for the 1924 cohort and decline thereafter) and convex for word fluency (with lowest performance for the 1931 cohort and return to the 1889 baseline thereafter) (see Figure 7–3).

FIGURE 7–3. Cumulative cohort differences for the primary mental abilities.



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An understanding of cohort differences is important in order to account for the discrepancy between longitudinal (within-group) age changes and the cross-sectional (between-group) age differences. In general, it was found that cross-sectional findings will overestimate within-individual declines whenever there are positive cohort gradients and will underestimate decline in the presence of negative cohort gradients. Curvilinear cohort gradients will lead to temporary dislocations of age-difference patterns and will over- or underestimate age changes, depending on the direction of differences over a particular time period. The slowing of the cohort difference trend suggests that in the next 20 or 30 years concurrently measured age differences will become substantially smaller over that age range where there is little or no within-participant decline. This is fortunate, because there is a need to retain people to higher ages in the labor force because of the demographic reality of the aging of the baby boomers. Stereotypes about age decline will obviously be reinforced less in the absence of the dramatic shifts in ability base levels that were observed for cohorts entering adulthood in the first half of the twentieth century.

What accounts for individual differences in age-related change in adulthood? Some individuals, either because of the early onset of neuropathology or the experience of particularly unfavorable environments, begin to decline in their 40s, whereas a favored few maintain a full level of functioning into very advanced age. All individuals do not decline in lockstep. Although linear or quadratic forms of decline may best describe the average aging of large groups, individual decline occurs far more frequently in a stair-step fashion. Individuals may have unfavorable experiences, to which they respond with a modest decline in cognitive functioning but then tend to stabilize for some time, perhaps repeating this pattern repeatedly before their demise. Moreover, the sequence of decline of abilities is not uniform across individuals but may depend in any one individual on the circumstances of use and disuse of particular skills. Thus, in actuarial studies of the SLS core battery, it was observed that virtually all individuals had significantly declined on one ability by age 60, but virtually no one had declined on all five abilities even by age 88.

Genetic endowment, of course, will account for a substantial portion of individual differences (Schaie 2005, Chapter 16; Schaie and Zuo 2001). Nevertheless, there are many other important sources of individual differences in intellectual aging that have been implicated in our studies. To begin with, the onset of intellectual decline seems to be markedly affected by the presence or absence of a variety of chronic diseases; cardiovascular disease, diabetes, cancers, arthritis, and other inflammatory diseases have all been identified as risk factors for the occurrence of cognitive decline, as is a low level of overall health. On the other hand, high levels of cognitive functioning seem to be associated with survival after treated malignancies and with late onset of cardiovascular disease and arthritis. Those persons who function at high cognitive levels are also more likely to seek earlier and more competent medical intervention in the disabling conditions of late life. They also are more likely to comply effectively with preventive and ameliorative regimens that tend to stabilize their physiological infrastructure. Perhaps even more importantly, they are less likely to engage in high-risk lifestyles, and they will respond more readily to professional advice that maximizes their chances for survival and reduction of morbidity. On the other hand, there does not seem to be a high relation between cognitive competence and systematic adoption of effective health behaviors. However, the more able individuals tend to engage in more effective medication use. Findings from the UNC Alumni Heart Study suggest that some personality factors may also be at work, as discussed later in this chapter.

*Can age-related ability change be modified through behavioral interventions?* Since the 1970s, a number of cognitive training studies have examined the question of the modifiability of age-related decline in independent-living elders without dementia (Ball et al. 2007; Schaie and Willis 1986; Verhagen et al. 1992). The target of these interventions has been abilities (verbal memory, perceptual speed, inductive reasoning) showing early age-related decline in the mid-60s. On the basis of findings of small-scale training studies, the Advanced Cognitive Training in Vital Elders (ACTIVE) (Ball et al. 2002; Jobe et al. 2001) randomized, controlled clinical trial was conducted, and findings of the 5-year follow-up have been recently reported (Willis et al. 2006). Elders were randomly assigned to one of three interventions focusing on the abilities of inductive reasoning, verbal memory, or speed of processing or a control group. Booster training effects for each of the interventions were found immediately after training and maintained at 5-year follow-up; effects were specific to the ability trained. Booster training significantly improved performance on the ability trained above the nonboosted intervention condition. At 5-year follow-up, those trained on reasoning reported significantly less difficulty performing instrumental tasks of daily living; those receiving booster training on speed of processing were faster at performing speeded tasks of daily living. Trainees in all interventions (compared with the control group) reported a higher level of quality of life 5 years after training (Wolinsky et al. 2006).

# **Cognitive Functioning Among the Oldest Old**

Data on the oldest old come from the set of studies included in the Georgia Centenarian Studies (Poon et al. 2007). This section reviews three pertinent questions regarding cognitive functions in very old age: 1) Is dementia inevitable as one ages? 2) Is maintenance of high cognition an important contributor to longevity? and 3) What phenotypes of cognitive abilities can be employed to classify cognition among the oldest old?

#### Dementia

Prevalence of dementia is found to be about 1.5% in adults in their mid to late 60s. Both prevalence and incidence rise to as high as 25%–30% in the oldest old. If one lives to be very old, an interesting question is whether dementia is inevitable. If dementia is inevitable, then the development of dementia may be part of the normative process as one ages. If the development of dementia is found not to be universal, then one may conclude that the development of dementia is pathological and not normal aging.

Empirical data from centenarian studies do not support the assertion that dementia is inevitable in aging (Gondo and Poon 2007). The prevailing finding from centenarian studies is that dementia prevalence ranged from 42% to 80% (Akisaka 2000; Andersen-Ranberg et al. 2001; Asada et al. 1996; Beregi and Klinger 1989; Choi et al. 2003; Gondo et al. 2006; Hagberg et al. 2001; Inagaki 1995; Karasawa 1985; Poon et al., in press; Powell 1994; Ravaglia et al. 1999; Robine et al. 2003; Silver et al. 2001; Sobel et al. 1995). A lower prevalence of 27% was reported by the Swedish Centenarian Study; however, after considering nonparticipants, the investigators estimated that the prevalence could be as high as 42% (Samuelsson et al. 1997). It is interesting to note that only one study to date did report a 100% dementia rate in the assessment of community-dwelling centenarians (Blansjaar et al. 2000). Kliegel et al. (2004) found that about half of their centenarians in the Heidelberg Centenarian Study showed moderate to severe cognitive impairment but that one-quarter were cognitively intact. Results of the Heidelberg study also demonstrated that cognitive decline was slightly but significantly accelerated in the last 6 months before death. Finally, a recent Japanese study reported that 24.3% of their centenarian sample had no dementia, 13.8% were classified to "probably" have no dementia, and 61.8% were classified as having mild to severe dementia (Gondo et al. 2006). Gender effects were reported in the Japanese study, indicating that men were generally functioning cognitively better than women.

Issues surrounding factors contributing to the development of dementia in old age are controversial beyond whether dementia is inevitable. The wide range of reported dementia prevalence in different parts of the world could be due to the use of different criteria in diagnosing dementia, the use of nonrepresentative samples, and differential genetic and

environmental factors affecting dementia in different geographic areas or cultures. Another potential contributor to the varying rates is that the female-to-male ratio among centenarians varied greatly, from 1:1 in Sardinia, Italy, to 12:1 in regions of South Korea. Because women tend to have a higher dementia prevalence (Andersen-Ranberg et al. 2001; Beregi and Klinger 1989; Choi et al. 2003; Gondo et al. 2006; Hagberg et al. 2001; Ravaglia et al. 1999; Robine et al. 2003; Sobel et al. 1995), the gender ratio could significantly affect the dementia prevalence of a sample or population. The time is ripe to better understand contributing factors to dementia prevalence within and between cultures and ethnicities.

### **Cognitive Function and Longevity**

Does a high level of cognitive functioning contribute to longevity? A review by Gondo and Poon (in press) provided supportive evidence in both longitudinal and centenarian studies. A series of studies that collected intelligence test data among children showed a strong relationship between high childhood intelligence and low mortality in middle and old age (Batty et al. 2006; Deary et al. 2006; Hart et al. 2005; Shenkin et al. 2004; Whalley and Deary 2001; Whalley et al. 2000). Similarly, the Terman cohort study (Friedman and Martin 2007), which examined the life course of intellectually gifted children over seven decades, found that mortality rates of these gifted children were significantly lower than those of their birth cohorts in the general population (see also Siegler 1980 for a review of these studies).

Bosworth and Siegler (2002) reviewed nine studies that evaluated the relationship between terminal decline of cognitive function and death. Although they were not able to confirm this relationship in a consistent manner, they did verify that lower cognitive function is predictive of mortality. Ghisletta et al. (2006) and Rabbitt et al. (2006) reported similar relationship of cognitive functioning and mortality among well-controlled, representatively sampled longitudinal studies. Data from the Nun Study (Snowdon et al. 1999) showed that subjects with higher linguistic abilities tended to live 7 years longer than their cohorts with lower linguistic abilities. Wilson et al. (2007) provide data from the Rush Memory and Aging Project and found an increased rate of cognitive decline within the final 3.5 years of life.

The facilitative effect of higher cognitive function on longer survival among the very long lived (centenarians) was also demonstrated. Poon et al. (2000) examined predictors of number of days of survival beyond 100 years among 105 centenarians from the Georgia Centenarian Study. They found cognition was one of four significant predictors. The others were gender, father's age of death, and nutrition sufficiency. Cognitive status measured by the Short Portable Mental Status Questionnaire was one of five significant predictors of survival among 800 centenarians in the French Centenarian Study (Robine et al. 2003). The other predictors were residential condition, health status, activities of daily living, and instrumental activities of daily living. Similarly, data from the Tokyo Centenarian Study (Gondo et al. 2006) showed that Clinical Dementia Rating score had a significant influence on survival. Taken together, cognitive functioning is an important contributor to survival in the general population as well as in the oldest old.

#### Phenotypic Classification of Cognitive Functions Among the Oldest Old

There is large within- and across-subject variability in the cognitive performances of the oldest old (Hagberg et al. 2001). Although the progression of pathological changes is correlated with cognitive performances, recent studies reported a significant amount of variability in the concordance between pathology and performance (Gold et al. 2000; Haroutunian et al. 1998; Nagy et al. 1997). The seminal findings from these studies were that there were excellent correlations between normal and severe dementia with cognitive functions; however, the relationships were ambiguous in the moderate stages.

There are no commonly agreed-upon criteria for the classification of phenotypes of oldest old that take into account their cognitive ability and neuropathology (Gondo and Poon, in press). However, studies that examined premorbid cognitive performances and pathological diagnosis at postmortem autopsy may be helpful with the formulation of criteria. Mizutani and Shimada (1992) autopsied 27 centenarians, 11 of whom had not developed dementia. Some degree of brain degeneration was observed in 8 of the 11 centenarians without dementia, but there were no apparent anatomical changes in the brains of the remaining three. The researchers termed those neuropathologically and behaviorally dementia-free centenarians "supernormal." The autopsies performed with the New England Centenarian Study (Silver et al. 2002) and the Aichi Centenarian Study (Ding et al. 2006) reported, respectively, 4 out of 14 cases and 4 out of 6 cases of centenarians without dementia that met the criteria of supernormal, with the remaining centenarians, although dementia free at time of death, having brain neuropathology that pointed to pathological progression of dementia at autopsy. These centenarians could be classified as maintaining normal cognitive reserves.

The supernormal and "cognitive reserve" centenarians were both dementia free, although the second group presented

some neuropathological degenerations. The second group could perform normally with everyday functions and communication; however, this group may have had difficulty with more complex tasks.

Finally, as noted earlier, 40%–80% of centenarians could be classified as having some degree of dementia. Most of these centenarians would have developed dementia at an advanced age, because early-onset dementia has been estimated to develop on average at 80 years. The final two phenotypes could be identified as "late-onset dementia" (defined as dementia with accompanying neuropathology developed at advanced age) and "early-onset dementia" (defined as dementia accompanying neuropathology developed at earlier age). In conclusion, although there is large individual diversity among the oldest old in both cognitive performance and neuropathological status, the four proposed phenotypes (supernormal, cognitive reserve, late-onset dementia, and early-onset dementia) could provide some guidelines in understanding the diversity.

### **Neuroimaging Data on Normal Aging**

The rapidly developing field of neuroimaging can provide valuable data on the relation between pathological and normal aging. The identification of dementia and other brain disorders from neuroimaging has been described elsewhere (Buckner et al. 2004; Hoffman 1997; Marcus et al. 2007; Steffens 1997). Here we focus on the highlights of current neuroimaging research in normal aging and some implications for the practicing physician.

The goal of neuroimaging research in aging is to characterize structural and functional age-related changes in the brain as well as how these changes are manifest in cognitive performance. Behavioral studies of cognitive performance have yielded a complex pattern of age-related decline in many—but not all—abilities. The state of this field is represented in the recent editions of *The Handbook of Aging and Cognition* (Craik and Salthouse 2000). Within this broad area of cognition, relevant reviews are available in specific areas of perception (Baltes and Lindenberger 1997; Schneider and Pichora-Fuller 2000; Scialfa 2002), processing speed (Madden 2001; Salthouse 1996; Salthouse and Madden 2007), attention (Kramer and Madden 2008; Madden 2007; Madden and Whiting 2004), language (Burke and Shafto 2008), and memory (Pierce et al. 2004; Zacks et al. 2000). A general trend of this research is that cognitive abilities that depend on perceptual speed and contextual memory tend to decline significantly with age, even for healthy adults, whereas abilities that rely on semantic knowledge and highly overlearned patterns decline less or may even improve. This trend has been expressed as different types of distinctions, such as *crystallized* versus *fluid* abilities (Cattell 1971; Horn 1982), *aging-resilient* versus *aging-sensitive* abilities (Lindenberger 2001), and *pragmatics* versus *mechanics* (Baltes and Lindenberger 1997).

A specific illustration of the type of cognitive change to be expected during normal aging is a longitudinal study of Swedish twins, reported by Finkel et al. (2007). These authors obtained estimates of longitudinal change across several testing occasions that were up to 16 years apart from a sample of twins who were 50–88 years of age at initial testing. Participants performed a battery of cognitive tests representing four domains: verbal abilities, spatial abilities, memory, and processing speed, which were each defined by a composite of tests. The results indicated that although some longitudinal decline occurred for all four domains, the decline was most pronounced for the spatial and speed domains. In addition, speed was a leading statistical indicator of change in both the spatial and memory domains (fluid ability) but not of change in the verbal domain (crystallized ability).

Pierce et al. (2004) proposed that when interpreting these types of changes in cognitive ability, it is important to recognize that they represent an adaptation on the part of older adults to a changing neurological environment. These authors classified failures of memory as seven "sins," including three sins of omission—transience (forgetting over time), absent-mindedness, blocking (e.g., tip-of-the-tongue states)—and four sins of commission—misattribution, suggestibility, bias, and persistence. Pierce et al. emphasized that age-related increases that occur in these types of errors can be viewed as useful byproducts of otherwise adaptive features of memory. That is, the goal of memory is to support the encoding, retention, and retrieval of task-relevant information, not to preserve all incidental details of the environment. The neurological changes that occur with advancing age may lead to an increased reliance on adaptive strategies that maximize available cognitive resources but also leave older adults more vulnerable to the resulting loss of some forms of memory information (see also McDaniel et al. 2008).

Neuroimaging studies have characterized the age-related changes in brain structure and function relevant for the cognitive changes expressed in the behavioral measures. One edited volume summarizes current work in this area (Cabeza et al. 2005), as do several individual articles and book chapters (Cabeza 2001, 2002; Dennis and Cabeza 2008; Raz et al. 1998, 2005). Age-related change is prominent in both structural and functional imaging measures. Volumetric studies of gray matter have established that age-related decline occurs in cortical volume, with concomitant increase in ventricular size. A theme across many of these studies is that age-related volumetric decline is more pronounced for

prefrontal regions than for more posterior cortical regions (Raz 2005; Raz et al. 2005). These findings have led to a frontal lobe hypothesis of cognitive aging (Dempster 1992; West 1996), which proposes that the cognitive changes associated with aging are the result of reduced frontal lobe efficiency. The degree to which reduced frontal lobe functioning can serve as an explanatory construct, however, is debated (Greenwood 2000; Tisserand and Jolles 2003). Age-related declines also occur, for example, in the volume and structure of posterior and sensory brain regions, such as gray matter near the primary visual cortex (Salat et al. 2004). In addition, although the division of the cerebral cortex into lobes is a useful pedagogical device, most cognitive tasks appear to rely on widely distributed cortical networks (Mesulam 1990; Tisserand et al. 2005).

Age-related decline in cerebral white matter volume is also observed, although it is not clear whether the trajectory of decline is comparable to that of gray matter. In addition, the magnitude of age-related decline in white matter appears to be sensitive to the proportion of study participants with hypertension or related cardiovascular disease. Increasing the proportion of these individuals tends to increase the degree of estimated age-related decline (Raz 2005). White matter hyperintensities, evident in T2-weighted structural magnetic resonance imaging (MRI), also increase in number and volume with age (Gunning-Dixon and Raz 2000; Raz et al. 2007; Yetkin et al. 1993). These hyperintensities are also correlated with hypertension and cardiovascular risk factors and represent decreased integrity of white matter (Oosterman et al. 2004; Raz et al. 2003; Soderlund et al. 2006; van den Heuvel et al. 2006).

Diffusion tensor imaging (DTI), in which the directionality and rate of molecular diffusion of water are measured (Mori and Zhang 2006), is a structural imaging method that is informative regarding age-related changes in white matter. This imaging modality is valuable because rather than relying on an ordinal-scale measure of pathology (e.g., number of hyperintensities), it provides an interval-scale measure of the range of white matter integrity throughout the brain. Studies using DTI have demonstrated that the integrity of white matter declines with age (Moseley 2002; Sullivan and Pfefferbaum 2006). This decline is also more prominent in the prefrontal regions but occurs posteriorly as well (Head et al. 2004; Salat et al. 2005).

Functional neuroimaging studies of aging complement these structural findings. Functional imaging has been conducted with both positron emission tomography (PET) and functional MRI, which measure cortical activation during task performance. Although many of the technological advances in neuroimaging have occurred in recent years, interest in the effects of normal aging dates to the first studies in the 1950s (Kety 1956). Neuroimaging of simple perceptual tasks, such as passively viewing checkerboards, has suggested that age-related decline occurs in both the amplitude (Buckner et al. 2000) and spatial extent (Huettel et al. 2001) of activation in primary visual (striate) cortex. By using appropriate control tasks, functional neuroimaging studies have identified age-related decline in brain regions associated with specific components of cognitive function. Many of these studies have found that age-related reduction of task-related activation in visual sensory regions is accompanied by age-related increased activation of prefrontal regions (Cabeza et al. 2004; Grady et al. 1994; Madden et al. 2005; McIntosh et al. 1999). This pattern has led to the suggestion that older adults compensate for deficiencies at a sensory/perceptual level by the recruitment of prefrontal regions associated with higher-order cognitive strategies. This type of theory is being investigated currently in a variety of task domains. One important issue is whether age-related increased activation is in fact compensatory, in which case better-performing older adults would exhibit relatively greater activation (Cabeza et al. 2002). In some instances, however, worse-performing older adults exhibit relatively greater activation, which may represent increased effort or task difficulty rather than compensation (Nielson et al. 2002). However this issue is resolved, current neuroimaging research suggests that 1) decline in activation is not the whole story, and 2) there is a high degree of plasticity of function in the aging brain (Craik 2006; Craik and Bialystok 2006; Grady 1998; Grady et al. 2006).

Ultimately, the contribution of neuroimaging will rely on relating the neuroimaging measures to behavioral measures. Although this may be intuitively obvious, the association of a particular brain structure or activation with a behavioral measure is still a correlational approach, and methodological and statistical care is required to identify causal relations in the data. Researchers are currently developing improved methods for analyzing the functional connectivity among brain regions in the context of specific task domains (Grady 2005; Ramnani et al. 2004). Structural imaging measures, such as white matter integrity from DTI, can be included in statistical models of age-related changes in cognitive function (Bucur et al. 2008; Colcombe et al. 2005; Madden et al. 2007). Functional imaging measures are being combined with behavioral measures in novel ways, for example, to distinguish remembered and forgotten items (Daselaar et al. 2006; Dennis et al. 2007).

For the practicing clinician, these theoretical developments are not always directly relevant but do lead to useful implications. First, cognitive change occurs throughout later adulthood; some decline in perceptual speed and fluid abilities will be evident even in healthy individuals. Second, significant changes in brain structure and function may

also occur in individuals without noticeable cognitive impairment, although at some point impaired cognitive function will be reflected in the brain measures. Third, health status is a relevant variable, and to the degree that cardiovascular disease and other comorbidities can be avoided, age-related decline is likely to be minimized. Fourth, the brain and central nervous system are constantly adaptive, and this adaptation is expressed in measures of older adults' brain function as well as in behavioral measures of cognitive performance.

Work in psychology of aging is becoming integrated across traditional areas. Work in cognition generally reports some decrements, although typically there is maintenance of emotional functioning. Although this is not surprising to the practicing psychiatrist, it is a new approach in psychology that comes from attempts to understand the aging mind. Carstensen et al. (2006) review the relevant literature, and the nub of their argument is that older persons are motivated to be selective and use their cognitive processing resources to meet emotional needs. Carstensen et al. provide a framework that can accommodate gains as well as losses seen in cross-sectional aging studies.

## PERSONALITY, COPING, AND BEHAVIORAL MEDICINE DEVELOPMENTS

#### **Personality Developments**

The unequivocal assertion that personality does not change over time is beginning to be challenged, particularly with the advent of more sophisticated statistical methods that allow for the test of individual growth curves and trajectories. A number of studies have pointed out that neuroticism appears to decline with age (Mroczek and Spiro 2003; Small et al. 2003) and that agreeableness and conscientiousness appear to increase over time (Helson et al. 2002; Small et al. 2003). Terracciano et al. (2005) reported that openness declined across adulthood, neuroticism declined up to age 80, and for extraversion there was first stability and then decline, whereas there was an increase in agreeableness and conscientiousness up to age 70.

Additional attention is being paid to possible cohort differences in personality. Twenge (2000), for example, reported an increase of neuroticism in more recent cohorts, but this has not been replicated in other studies (Terracciano et al. 2006). The Terracciano et al. (2006) study, however, did report cohort effects for personal relations, with later-born cohorts declining more than one T-score point per decade. In relation to this finding, Robinson and Jackson (2001) also reported a decline in trust among Americans born after the 1940s.

Continuity of personality and social preferences is expected across the adult life course; thus, changes have potential diagnostic significance and make knowledge about expected trajectories important. Although the work on cognitive development reviewed previously finds generally good patterns by domain of performance, individual differences in personality predict physical disease, which in turn has consequences for cognitive performance, which then leads to greater incidence of disease. This can be well illustrated with work on hypertension.

Findings from the UNC Alumni Heart Study indicate a relationship, dependent on covariates in the model, between personality in early middle age (approximately age 40) and incident hypertension 11–15 years later. The behavioral predictors of hypertension are well known and include age, education, exercise, family history, overweight, and obesity in the UNCAHS cohort. Hostility also predicted hypertension, but this effect was mediated only by overweight and obesity. This same pattern was seen for Neuroticism facet scale score findings of N5 (Impulsiveness) for overall N (Neuroticism). Because hypertension is a silent disease, it was also more likely in more conscientious persons and in Conscientiousness facets of C1 (Competence) and C3 (Dutifulness). Aside from Conscientiousness and its facets, only job strain and A4 (Anger) score predicted hypertension with all traditional covariates in the model. Because UNCAHS is a mail survey, hypertension was tested on 2 days—when first reported and when treatment first reported—to model how it would have been defined had we been able to measure blood pressure directly, where normal pressures with treatment are considered hypertension (Siegler 2007b). Midlife hypertension confers increased risk for later coronary heart disease and stroke and vascular dementia.

Elias et al. (2004) present 30 years of research on the impact of age and hypertension on normal cognitive functioning, a study that started in 1974. A summary of the findings is presented here. They found that almost all abilities are affected by hypertension and that antihypertensive treatments may not prevent this decline. After 30 years, questions remain about subtypes of disease and of treatments. Overall estimates of the impact of changes in blood pressure on summary indicators of Wechsler Adult Intelligence Scale performance and speed indicate that being hypertensive carried a 74% increased risk of poor performance, with a 67% increase in risk of poor speeded performance compared with estimates for 10 years of age at 58% and 85%, respectively, with a 20 mmHg increase in systolic blood pressure conferring 18% and 22% increases in risk. (Cross-sectional normative data are presented in Dore et al. 2007.) These are useful data derived from the Maine-Syracuse Longitudinal Study stratified by age and education showing level of

mean -1.5 standard deviation of change, indicating an estimate of the level of performance that could be considered mild cognitive impairment, which may represent a heightened risk for Alzheimer's disease. Cognitive and neuropsychological measures in the battery were also evaluated by proportion of variance accounted for by age, education, and gender separately and together, as well as additional variance caused by disease indexed by depression and health indicators including risky behaviors like smoking and prevalent disease. On average, health variables added about 3%. These data underscore the importance of long-term chronic disease assessment and management for geriatric psychiatry.

Mroczek et al. (2006) cast traditional concerns of stability and change in personality with age into theoretical terms and note that the changes can be both positive and negative and respond to developing health conditions in adaptive ways. Work in this area still looks to see if nonnormative changes have medical consequences that should raise the level of suspicion in an insightful clinician. Theoretically, Hooker and McAdams (2003) have incorporated social processes into trait psychology, although empirical findings will take time to emerge. Latent growth curves are providing new techniques to evaluate sophisticated developmental patterns of change. Our own work on hostility (Siegler et al. 2003) finds the normative pattern of declining hostility with age is replicated longitudinally and cross-sectionally (Siegler 2007b) but reflects only 75% of the population; in a very small group (3.5%), hostility actually increases from age 18 to 60 years, whereas the remainder decline slowly. Differences in such trajectories have definite health consequences. At age 60, those who increased in hostility were more likely to be hypertensive, to be depressed, and to have cancer (Siegler 2007b), as shown in Figure 7–4.

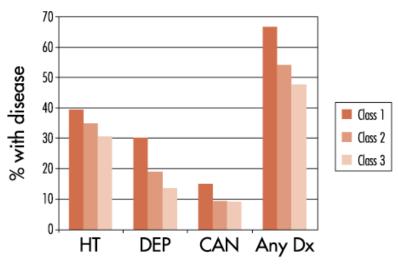


FIGURE 7–4. Classes of hostility and cumulative disease.

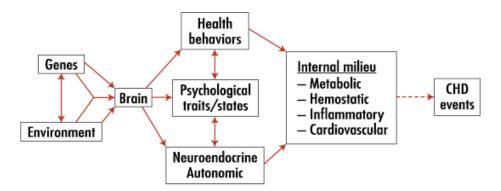
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HT = hypertension; DEP = depression; CAN = cancer.

*Source.* Siegler IC: "Psychology of Aging and the Public Health." 2007 Developmental Health Award, Division 20 and Division 38. Invited address presented at the Annual Meeting of the American Psychological Association, San Francisco, CA, August 2007. Reprinted with permission.

Work in behavioral medicine is based on a search for the biological and physiological mechanisms that relate psychosocial constructs such as personality to disease (Siegler 1989). All of these are variants of a stress model that involves neuroendocrine, immune, inflammatory, and cardiovascular reactivity paradigms. These are illustrated in a model by R.B. Williams (2007) (see Figure 7–5).

FIGURE 7–5. A model of psychosocial mediators of health events.



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CHD = coronary heart disease.

*Source.* Williams RB: "Coping Skills Training in Different Cultures: The LifeSkills Experience." Poster presented at the First Conference of the Central Eastern European Society of Behavioral Medicine, Pecs, Hungary, August 20–22, 2007. Reprinted with permission.

There are normative age differences in all of these biological indicators (see Hazzard et al. 1999; Markides 2007) and fewer longitudinal age change data to evaluate. Recent published chapters have worked to integrate aging data into these frameworks for cardiovascular and social risk domains (Berg et al. 2007), neuroendocrine parameters (Epel et al. 2007), and all of the systems that respond to chronic stressors such as caregiving (Young and Vitaliano 2007). Research is moving toward personalized medicine that will take genes and gene environment interactions into effect (R.B. Williams 2007).

A useful illustration of how this model works in an aging population is provided by emerging findings from our recently completed study of the impact of caregiving (Duke Caregiving Study: Brummett et al. 2005, 2006a, 2007b, 2008; Dilworth-Anderson et al. 2005a). The broad objectives of this research are to identify factors in the social (e.g., being a caregiver for a relative with Alzheimer's disease) and physical (e.g., neighborhood characteristics) environments that interact to affect biological and behavioral characteristics that lead to poorer physical and mental health and to evaluate variants in genes that regulate function of the neurotransmitter serotonin as moderators of the impact of these environmental factors on health and disease.

We found that caregivers who expressed a higher level of concern about crime in their neighborhood had higher levels of fasting blood glucose and glycosylated hemoglobin (a measure of average blood glucose over the past 2–3 months) than either caregivers with low crime concerns or matched control subjects with high or low crime concerns. These findings suggest that among the millions of Americans with caregiving responsibilities for a relative with Alzheimer's disease, those who live in neighborhoods that engender concerns about crime are at higher risk for developing type 2 diabetes and the other diseases, such as heart disease, to which it leads (Brummett et al. 2005). In a structural equation model, we found that caregivers of a relative with Alzheimer's disease report poorer sleep quality indirectly through reduced social support and increased levels of negative emotions, compared with matched control subjects who do not have caregiving responsibilities (Brummett et al. 2006a) and that these differences in sleep quality are related to monoamine oxidase-A alleles associated with less transcriptional activity and with depression (Brummett et al. 2007b) in caregiving men. Poor sleep quality in women was associated with the S allele in the serotonin transporter gene (5-HTTLPR) (Brummett et al. 2007a). Gender effects in response to caregiving were seen in the UNCAHS, where for middle-aged caregivers, caregiving was associated with diabetes for men and depression for both men and women in models controlling for age and income. Age increased risk of disease, whereas income was protective (Siegler et al. 2006).

A literature is developing that finds consistent personality mortality associations. Not only does hostility in college predict premature mortality in the UNCAHS (Siegler 2007), we have also found that optimists compared with pessimists were more likely to survive 40 years after college entry (Brummett et al. 2006b). Friedman and Martin (2007) review conscientiousness as a critical construct in survival and an integrated way to think about personality as a system, whereas our own work is finding new implications for the facets of openness to experience in coronary patients (Jonassaint et al. 2007). The behavioral medicine literature focuses more on negative constructs (hostility, neuroticism, and pessimism), whereas survival studies focus more on the more positive traits. Whether the individual

constructs or the broader domains prove more useful (R.B. Williams et al. 2003; Suls and Bunde 2005), the findings are starting to show the general trends seen above. What do long-term survivors actually look like?

## **Personality in Centenarians**

In order to survive successfully into very old age, individuals appear to need a highly resilient or robust personality. Several centenarian studies appear to point this out. For example, the New England Centenarian Study noted that centenarians were very stress-resilient individuals (Perls and Silver 1999). Findings from the Georgia Centenarian Study also noted that a particular cluster of personality traits was more likely to be found among centenarians: relatively high levels of extraversion, emotional stability, and conscientiousness (Martin et al. 2006). High ratings of emotional stability also were found in centenarian studies in Sweden (Samuelsson et al. 1997) and Japan (Shimonaka et al. 1996). A longitudinal follow-up showed that centenarians had decreased scores in sensitivity but higher scores in openness (Martin et al. 2002) after an 18-month follow-up testing. The results suggest that centenarians may compensate for physical and functional decline by having robust personality traits and by becoming less sensitive and more open-minded.

#### **Coping Developments**

There is considerable interest to study coping behaviors in centenarians. This group of "expert survivors" (Poon et al. 1992) faces accelerated changes in a number of functioning domains, such as activities of daily living, and considerable losses of peers and family members. How do individuals at such an advanced age cope with these changes? The results obtained so far suggest that centenarians are less likely to use "active behavioral" coping styles (Martin et al., in press). Active behavioral coping refers to all specific actions individuals take when being confronted with stressors or events. For example, seeking professional advice and talking with family and friends constitute active strategies. It is not surprising that centenarians are less likely to use active behavioral coping, because their resources are more limited. Although centenarians are restricted in their active behaviors, the level of active cognitive coping does not appear to diminish (Martin et al., in press). Centenarians may not be able to *do* something about a problem, but they surely can *think* about it as much as any other age group. Along the same lines, Martin et al. (2001) pointed out that it may not be the general coping modes (i.e., active behavioral, active cognitive, or avoidance) that play an important role. Rather, it may be specific "molecular" coping behaviors that distinguish the oldest old from other age groups. For example, centenarians are more likely to use religious coping and acceptance, whereas they are less like to worry about a problem (Martin et al. 2001). A centenarian study in Barbados also noted that successful adaptation and coping among centenarians were positively related to high levels of religiosity (Archer et al. 2005).

# **Coping With Caregiving in Diverse Populations**

In the face of the growing numbers of people with Alzheimer's disease and related issues such as testing, assessment, and care, Peggye Dilworth-Anderson, Ramón Valle, Sam Fazio, and Teresa Radebaugh convened a conference in 2004 and published papers from this conference that address the 5 million Americans who have Alzheimer's disease. Of particular concern in the discussions and published papers is that what is known about aging and Alzheimer's disease is a function of the people studied, and currently little is known about diverse populations. It is important to further understand the heterogeneity of Alzheimer's disease, because heterogeneity may be within the disease as well as within the population. Including diverse populations in Alzheimer's disease research can provide opportunities for diagnosis, care, and treatment for everyone.

Further, despite many years of Alzheimer's disease research, our understanding of the effects of this disease on family caregivers is still limited for ethnic minorities. For example, we know, based on current evidence, that 1) the burden of Alzheimer's disease is greater among African Americans, among whom age-specific prevalence of dementia is 14%–100% higher than that found among European Americans; 2) first-degree relatives of African Americans who have Alzheimer's disease have a 43.7% cumulative risk of getting the disease compared with 26.9% for whites, and among blacks, spouses have an 18.5% cumulative risk of getting the disease compared with 10.4% for whites (Green et al. 2002); 3) African Americans are less likely to institutionalize relatives with dementia (43.7%), compared with whites (89.6%) (Stevens et al. 2004), and 29% of African American families provide care for their older family members compared with 24% of white families (Dilworth-Anderson et al. 2006); 4) African American caregivers are more likely to care for more than one dependent adult in their families, spending an average of 20.6 hours per week providing care; 5) African American caregivers tend to underutilize formal services; 6) 66% of African American caregivers are as likely to be a spouse as an adult child (Hinrichsen and Ramirez 1992). These conditions would suggest that caregivers of African American elders are particularly vulnerable

to poor emotional and physical health outcomes. Using data published from the Resources for Enhancing Alzheimer's Caregivers Health (REACH), investigators addressed these vulnerabilities, as well as those in other groups, through a multicomponent intervention (Belle et al. 2006). Their findings show that compared with minimal support provided in a control group, their multicomponent intervention statistically significantly improved the quality of life (as measured by indicators of depression, burden, social support, self-care, and patient problem behaviors) for white and Hispanic caregivers but not for African American caregivers. However, they found statistically significant quality-of-life changes with this intervention among African American spouse caregivers, in contrast to African American adult children in the caregiver role. Given that adult children provide the majority of care in black families (unlike in white and Hispanic families), additional research is needed to better identify and address their emotional and physical health vulnerabilities through interventions.

Limited information on psychological coping poses further concerns for understanding how diverse groups respond to and address the stress and strain of caregiving. Evidence shows that caregivers suffer emotionally from a variety of stressors because of the physical demands of assisting care recipients with daily activities (Alzheimer's Association and National Alliance for Caregiving 2004). Of particular concern is the type and degree of caregiver stress associated with caring for elders with dementia who often have behavioral and physical health problems (Haley et al. 2004; Hooker et al. 2002; Schulz and Martire 2004). Information on addressing emotional coping and well-being among dementia caregivers in diverse groups suggests that a sociocultural perspective is needed to understand the diversity issues that are involved. A sociocultural perspective takes into consideration an ethnic and cultural group's history, values, beliefs, and ways of thinking. It is also characterized by what is often described as the "historical memory" of a group as evidenced by customs, rituals, and ways of expressing themselves. Work from our Duke Caregiving Study found that African Americans have different cultural reasons for providing care for relatives with Alzheimer's disease and that this varies by educational level (Dilworth-Anderson et al. 2005a). Findings show that race and ethnicity appear to influence significantly the expression of depression, and depression is not always synonymous across cultures. Hence, it has been suggested that the application of standard mood inventories in African American groups may contribute to the observation of lower prevalence rates of depression in this group when compared with white samples (Harrelson et al. 2002). Studies of depression in caregivers of patients with Alzheimer's disease have also underscored the racial and ethnic differences in depressive symptomatology.

In some studies, it appears that African American caregivers of patients with Alzheimer's disease are often reported as less depressed when compared with white caregivers (Haley et al. 1996); however, both groups show other negative health outcomes from caregiving over time, such as increased physical symptoms (Roth et al. 2001). Findings by Dilworth-Anderson et al. (1999) show that very few African American caregivers experience depression assessed by the Center for Epidemiologic Studies Depression Scale (Radloff 1977); however, by using Derogatis's (1993) global index on distress, their findings did document that about 18% of the caregivers were emotionally distressed. These distressed caregivers received less social support, were in poorer physical health, and experienced more caregiving problems than caregivers who were not distressed (Dilworth-Anderson et al. 1999). Thus, to be appropriately sensitive to depression among African Americans and possibly other racial and ethnic groups, researchers need to rethink how best to measure depression with culture in mind. Both conceptual and methodological issues, therefore, will need to be revisited as we approach understanding emotional well-being among diverse groups of caregivers.

# **Behavioral Interventions**

Research on the role of social factors in aging has benefited from the flowering of integrated theoretical work in emotion and motivation by Carstensen and her colleagues and has been the basis for behavioral intervention studies. Not only has there been great progress in basic research in the psychology of normal aging, but major intervention studies also have been completed and reported. Willis et al. (2006) present the results of a cognitive training intervention for normally aging persons (ACTIVE) that suggests that cognitive training can be beneficial; Gitlin et al. (2003) present REACH for interventions to reduce the stress of caregiving; and Berkman et al. (2003) and Lett et al. (2007) present ENRICHD, which attempted to modify depression and social support to reduce the impact of coronary heart disease. These three large clinical trials show the beginnings of applications of decades of findings in psychology to help mitigate the impact of age-related changes in the population. Williams LifeSkills (V.P. Williams and Williams 1999) teaches coping skills and has been found to reduce coronary heart disease risk indicators (Bishop et al. 2005). This approach is currently being tested as a framework to help caregivers. Randomized clinical trials with behavioral interventions are difficult to conduct because individuals who are randomly assigned to the control group can sometimes provide an intervention for themselves. The results of these behavioral interventions are less important than the fact that they are entering the realm of tested scientific practice. This represents an important acknowledgment of the role of psychosocial factors in disease as well as an optimism that something can be done to reduce the burden.

## IMPLICATIONS FOR THE PRACTICE OF GERIATRIC PSYCHIATRY

The practice of geriatric psychiatry is healthy (Cohen 2005). There are still major gaps in our knowledge of how to define *normal* aging in frail institutionalized populations that have been defined as "abnormally" aging—however, this work is beginning (Buckman et al. 2007; Tyas et al. 2007; Welsh-Bohmer et al. 2006). Population-based national studies generally do not have sufficiently rich measurement batteries; thus, there is a growing group of older impaired persons and their caregivers who could benefit from more study. Similarly, we do not have a "psychology of aging with Alzheimer's disease" or "aging with multi-infarct dementia." If these disorders are soon cured, we will not need one. Until then, multiple generations of aging persons can be expected to live longer, more complex lives, and geriatric psychiatrists may have two or three generations in the same family as patients, needing to understand multiple trajectories of normal aging processes.

#### **KEY POINTS**

- Individual decline in cognitive performance before age 60 generally is not normal aging. By the mid-70s, average
  decrement is observed for all abilities, and by the 80s this decrement is severe except for verbal ability.
- Empirical data from centenarian studies suggest that dementia is not inevitable.
- Cognitive abilities that depend on perceptual speed and contextual memory tend to decline with age, even for healthy adults, whereas abilities that rely on semantic knowledge and highly overlearned patterns decline less or may even improve.
- Continuity of personality and social preferences is expected across the adult life span; thus, changes have potential diagnostic significance.
- The effects of Alzheimer's disease and of caregiving for relatives with Alzheimer's disease vary in diverse populations.

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