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Education and Cognitive Decline in Older Americans: Results From the AHEAD Sample

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Abstract

Although education is consistently related to better cognitive performance, findings on the relationship between education and age-associated cognitive change have been conflicting. Using measures of multiple cognitive domains from four waves of the Asset and Health Dynamics of the Oldest Old study, a representative sample of Americans aged 70 years and older, the authors performed growth curve modeling to examine the relationships between education, initial cognitive score, and the rate of decline in cognitive function. More years of education were linked to better initial performance on each of the cognitive tests, and higher levels of education were linked to slower decline in mental status. However, more education was unrelated to the rate of decline in working memory, and education was associated with somewhat faster cognitive decline on measures of verbal memory. These findings highlight the role of early-life experiences not only in long-term cognitive performance but also in old-age cognitive trajectories.

Keywords

education; socioeconomic status; cognition; memory; growth curve modeling

It is well known that higher educational attainment is related to higher levels of cognitive performance in late life (Albert 1995; Schaie 1996; Wechsler 1981); however, it remains unclear whether more education slows the rate of cognitive decline over time in late life. Most longitudinal studies linking education and cognitive change in old age have found a direct relationship, such that lower education levels are associated with a greater risk for poor cognitive performance (Albert et al. 1995; Colsher and Wallace 1991; Evans et al. 1993; Farmer et al. 1995; Jacqmin-Gadda et al. 1997; Lee et al. 2003; Lysetkos, Chen, and Anthony 1999). However, some research contradicts these findings, suggesting that higher education does not protect against cognitive decline (Christensen et al. 2001; Hultsch et al. 1998; Leibovici et al. 1996) or may even result in a greater rate of decline (Ardila et al. 2000; Butler, Ashford, and Snowdon 1996; Stern et al. 1999; Unverzagt et al. 1998). Relationships appear to differ by cognitive task, age, and educational group. Further research is needed to address this inconsistency and to clarify the ways in which education influences cognitive stability and decline. The purpose of this study was to assess the relationship between higher education and

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change across multiple domains of cognitive function in a representative sample of older Americans.

Education and Cognitive Function

Previous research suggests that education may improve cognitive function in a variety of ways. Education may increase cultural competency, improving reading, math, and reasoning skills, as well as test-taking abilities. At the same time, education may actually improve brain function. Because enriched environments result in a greater number of synapses (Diamond 1988; Jacobs, Schall, and Schiebel 1993), individuals with higher education may enter old age with a greater synaptic density. These hypotheses are roughly related to two principal dimensions of intelligence: crystallized intelligence, representing knowledge gained from culture relating to judgment, understanding, and reasoning, and fluid intelligence, representing the influence of biological factors on an individual's ability to perceive, recall, and think about patterns, relationships, and implications (Horn and Cattell 1966). Crystallized intelligence remains fairly intact or even increases across the life span, whereas fluid intelligence deteriorates with advancing age (Baltes 1993). Although education is related to both higher crystallized and higher fluid intelligence, crystallized intelligence appears to be particularly sensitive to education.

Education early in life is likely to affect late-life cognitive outcomes in part through its association with adult socioeconomic status and social behavior. Education is related to adult occupation and lifestyle, and higher education early in life may result in greater mental activity in occupation and leisure pursuits throughout life (Andel et al. 2006; Kramer et al. 2004; Richards, Hardy, and Wadsworth 2003; Richards and Sacker 2003). Thus, education may have a direct influence on cognitive function early in life through its role in promoting cognitive growth, and education may play an indirect role in maintaining cognitive function later in life through its relationships with adult socioeconomic status and social behavior.

Education and Cognitive Change

Untangling the relationship between education and age-associated cognitive change has potentially important implications for our broader understanding of cognitive reserve and age-associated cognitive decline. Although researchers have suggested pathways through which education may bolster the level of cognitive function one has entering old age, they have focused less on its role in cognitive decline at older ages. Three competing hypotheses could offer insight about the effect of education on the rate of cognitive change.

If education slows the rate of cognitive decline, this finding would support an *active cognitive reserve* hypothesis. In this case, individuals with higher education are hypothesized to process tasks more efficiently (Stern 2002); because they make more efficient use of brain networks, the same amount of organic cognitive damage results in a smaller decline in cognitive function relative to those with less education. Alternately, if education does not modify the rate of cognitive decline, this supports a *passive cognitive reserve* (Stern 2002) or a *common cause* (Salthouse 1999) hypothesis. If aging individuals begin to lose cognitive function from a common cause, such as declines in processing speed, people with higher education will decline at a rate similar to the total population but will continue to perform at a higher level at any given age because of greater baseline brain reserve. Finally, if education increases the rate of cognitive decline, this could suggest a *compensation* hypothesis (Christensen et al. 1997; Stern 2002). If older adults rely on intact cognitive domains to compensate for declines in other cognitive abilities, this compensatory behavior would slow decline until these other domains also begin to deteriorate. Research suggests that more highly educated older persons may capitalize on their increased crystallized abilities to supplement declining fluid abilities (Christensen et al. 1997; Compton et al. 2000). Therefore, if organic brain deficits eventually

began to interfere with this compensation strategy, one would expect a faster rate of decline among educated older adults.

Previous Research

Inconsistencies in earlier studies appear to derive at least in part from differences in methods, measurement, and samples. First, most investigators have used methods that define cognitive decline either as change in performance scores or decline in performance below a specified threshold value between two time points (Albert et al. 1995; Evans et al. 1993; Farmer et al. 1995; Jacqmin-Gadda et al. 1997; Lysetkos et al. 1999). Ideally, to estimate a true trajectory of cognitive decline, cognitive ability should be assessed at multiple time points rather than using a simple difference in two test administrations. Analyses from the Baltimore cohort of the Epidemiological Catchment Area study using difference scores found that those with more than eight years of formal education experienced less decline (Lyketsos et al. 1999). However, a recent longitudinal study of older Australians found no relationship between education and cognitive decline on the basis of multiple time points (Christensen et al. 2001).

Second, the majority of studies that have reported lower education to be a risk factor for cognitive decline have used mental status measures that assess the most basic level of crystallized cognitive abilities and may be insensitive to change among well-educated adults (Albert et al. 1995; Evans et al. 1993; Farmer et al. 1995; Jacqmin-Gadda et al. 1997; Lysetkos et al. 1999). At the same time, studies reporting no relationship between education and cognitive decline have primarily focused on either fluid abilities, which are considered to be the product of biological traits, or composite measures that do not differentiate between abilities (Anstey and Christensen 2000).

Third, the literature on the relationship between education and cognitive change has been inconsistent because study samples have been confined to specific subpopulations of older adults, such as religious clergy, high-functioning older adults, and/or older adults in geographically defined areas; these groups may vary greatly in educational attainment and opportunities, as well as other environmental exposures (Albert et al. 1995; Butler et al. 1996; Carmelli et al. 1997; Christensen et al. 2001; Evans et al. 1993; Lee et al. 2003; Lysetkos et al. 1999). A clear example is the observation that Black and Hispanic older Americans consistently perform more poorly on cognitive tests compared with non-Hispanic White Americans, largely as a function of lower levels of educational attainment and fewer educational opportunities early in the twentieth century (Cagney and Lauderdale 2002; Manly and Jacobs 2001; Whitfield 2002). Even though sample composition may be a critical factor in determining study outcomes, no study to date has compared trajectories of cognitive decline within a nationally representative sample of older Americans. The purpose of the current analysis was to determine whether greater educational attainment is associated with the rate of cognitive decline across multiple testing occasions for a representative sample of Americans over the age of 70 years.

Methods

Data

Data came from the Asset and Health Dynamics of the Oldest Old (AHEAD) study, a nationally representative longitudinal study of older Americans living in the community at the first interview and born in 1923 or earlier. The first four waves of the AHEAD study, a biennial, nationally representative data set, were collected by the Survey Research Center at the University of Michigan (Soldo et al. 1997). Blacks and Hispanics were oversampled in AHEAD to ensure adequate representation of these groups for analysis (Soldo et al. 1997). Because the AHEAD sample represents the educational and ethnic heterogeneity of the older

American population aged 70 years and older, we were able to investigate the role of education and race and ethnicity in cognitive change simultaneously.

The sample at the first data collection in 1993 consisted of 7,443 persons aged 70 to 103 years. Growth curve analysis allowed us to make full use of data for all subjects with at least one cognitive interview. Respondents were included if they had at least one self-response on cognitive performance items across the four testing occasions. For this analysis, 638 respondents were excluded because they never completed the cognitive interview and all data were supplied by a proxy, and another 154 were missing cognitive data. The 6,651 remaining respondents had at least one cognitive interview, 81% had two cognitive interviews, and 66% and 53% had three and four interviews, respectively. By the fourth wave, 2,306 participants had died, although data on these subjects were included for available waves. At the final data collection in 2000, the sample consisted of 3,541 respondents aged 77 to 101 years. Respondents who died or were missing at follow-up scored lower on baseline cognitive measures and were more likely to be older, to be less educated, and to have a chronic health condition (see Herzog and Wallace 1997 for a more detailed discussion of missing data on cognitive measures).

Factors known to affect cognitive decline are treated as covariates in the analysis. Baseline age and subsequent aging are analyzed because age explains a substantial proportion of the population variance in cognitive functioning and decline over time (Albert 1994; Zelinski and Burnight 1997). Previous research has found that women are at greater risk for cognitive impairment (Barrett-Connor and Kritz-Silverstein 1999; Fratiglioni et al. 1997; Ott et al. 1998). Additionally, several chronic health conditions have been identified as correlates of cognitive impairment, including high blood pressure, diabetes, heart disease, and stroke (MacKnight et al. 2002; Posner et al. 2002; Ruitenberg et al. 2001; Zelinski et al. 1998; Zhu et al. 1997). Because participants could have developed these chronic conditions during the period of study, disease indicators were included as time varying. Given the established relationships between demographic characteristics, health status, and cognitive function in old age, accounting for these varying characteristics is essential to understanding the effect of education on cognitive decline. In addition, prior exposure to cognitive testing may result in subjects' learning the test approach and improving scores, so an indicator of prior testing to control for a practice effect was also included (Hultsch et al. 1998).

The weighted sample for this analysis was 86% non-Hispanic White, 10% Black, and 4% Hispanic (Table 1); fewer than 1% were classified as other ethnicities, including Asians and American Indians. At baseline 63% of the sample was female. Overall, the average level of education was 11.1 years of school completed. Education ranged from no years of formal education to greater than 17 years; 42.8% of respondents had between 10 and 12 years of education. As expected, education differed by race, with African Americans completing an average of 8.6 years of school, Hispanics completing 6.3, and non-Hispanic Whites completing 11.7. Education was coded as a continuous variable. At baseline, 50% of respondents had hypertension, 12% had diabetes, 31% had heart disease, and 7% had histories of stroke. Another 11% of subjects developed hypertension, 5% diabetes, 12% heart disease, and 7% stroke over the study period.

The average age at baseline was 77.2 years, with a range of 70 to 103. To create an easily interpretable model, a centered variable for age was used to represent the difference between a respondent's age and the mean age for the sample at baseline. By centering age, we assumed that the intercept represented a person of average age at baseline; subsequently, we could determine how differences in age from the group average affected cognitive function. Using this specification, time and age were represented by the same variable, allowing us to construct growth curves across multiple age groups.

Cognitive Measures

The same cognitive tests were administered at all four waves of data collection and used to construct cognitive trajectories for individuals on each test (Herzog and Wallace 1997). Four tests that tap different cognitive abilities were used in this analysis: delayed and immediate verbal recall to assess verbal memory (fluid intelligence), the Serial 7's to assess working memory (fluid intelligence), and the Telephone Interview for Cognitive Status (TICS) to assess general mental status (a composite measure reflecting very basic crystallized intelligence). Baseline descriptive data for each of these measures are included in Table 1.

The verbal recall tests consisted of 10 common nouns read aloud by the interviewer, followed by a request for respondents to recall as many nouns as possible from the list (immediate recall). Delayed recall was tested after approximately five minutes (spent on other areas of test administration) by asking respondents to again recall as many words as they could. Scores represent the number of correctly remembered words, with a range from 0 to 10. If a respondent replied, “I don't know” when asked to recall any words from the list, a score of 0 was assigned.

The TICS is derived from the Mini-Mental State Examination and is specifically designed for administration over the telephone (Brandt, Spencer, and Folstein 1988; Folstein, Folstein, and McHugh 1975). The TICS is designed to assess the most basic level of cognitive functioning, general mental status, including language skills and orientation to place and time (Albert 1994). These abilities are assessed through questions on object identification, date identification, and naming the president and vice president. The TICS has a score range of 0 to 10, determined by summing the number of correct answers.

The Serial 7's test asks respondents to continuously subtract 7 for five trials, starting at 100 and ending at 65; scores are determined by summing the correct number of trials for a score range of zero to five. The number of respondents who declined to participate in the Serial 7's test was significantly higher than refusals on other cognitive tests. At baseline, 12% (n = 769) of the 6,651 respondents refused to answer Serial 7's questions; fewer than 1% of participants refused the word-recall lists or individual items on the TICS. Although the number of refusals changed across subsequent data collection points, the pattern of disproportionately greater refusals on the Serial 7's test was consistent at every time point. Because these data were not missing at random (Herzog and Wallace 1997), it was important to take account of missing data rather than to delete missing cases (Rodgers, Ofstedal, and Herzog 2003). Serial 7's values for participants who refused were imputed cross-sectionally on the basis of values on the other three cognitive tests of the same wave. The results shown below were based on analysis of imputed values. Imputation did not significantly change the sample mean or the significance or direction of the relationships between education and cognition in regressions relative to a coding scheme that defined refusals as missing.

In longitudinal analyses with repeated administrations of cognitive tests, there is a risk of practice effects because of prior exposure to the tests, resulting in improved performance on later testing occasions. To control for practice effects in the analysis, a variable was included in the models to represent prior exposure to the tests; respondents were assigned zeroes for baseline participation in the cognitive test and ones at each subsequent test administration. Thus, the coefficient for practice effect represents variation due to prior experience with the tests, or the average increase due to practice between the first and second testing occasions. Prior research on this methodological approach has found this technique to adequately account for the effects of repeated test exposure (Hultsch et al. 1998).

Statistical Analysis

The use of cognitive measurements at multiple time points has several advantages, especially when cognitive change is analyzed using growth curve models. First, multiple measurements provide a more reliable assessment of long-term cognitive change. Second, growth curve models allow researchers to examine differences in baseline scores and slope separately using growth curve modeling. A variety of risk factors may affect differences in cross-sectional cognitive performance but do not affect cognitive decline over time in late life. These cross-sectional differences are likely to represent fixed differences established earlier in life and may not represent the same risk factors that determine decline, but these differences are likely to be missed in simple models predicting change score. Third, modeling cognitive change using growth curve models accounts for the correlation of multiple measurements within subjects and minimizes the underestimation of standard errors typical of analyses predicting change (Glymour et al. 2005; Raudenbush and Bryk 2002). Fourth, growth curve analysis makes full use of available data, incorporating subjects who have data missing at later waves. Although this procedure cannot fully address potential bias due to attrition, it minimizes bias relative to the use of change scores. Finally, multiple measures of cognition allow for the testing of nonlinear models of change.

Growth curve models were used in this study to estimate the relationship between education, the initial level of cognitive function, and the rate of age-associated cognitive decline. Growth curve models have been used in longitudinal designs to model individual change in a variety of outcomes as a function of time (Littell et al. 1996; Raudenbush and Bryk 2002; Reynolds, Gatz, and Pedersen 2002). Estimating a growth curve of cognitive decline depends on the specification of a mixed model, one that contains both fixed effects (estimates for the entire sample) and random effects for a respondent (individual estimates). The utility of this approach is that it not only estimates the average path of cognitive change for the entire sample but considers baseline cognitive function as a source of individual random variability that affects the subsequent rate of cognitive decline (Singer 1998). This was especially important for the current analyses to determine whether well-educated older adults have a differential rate of cognitive decline or simply better baseline cognitive performance than persons with less education.

Separate models were estimated for each of the four cognitive measures: immediate verbal recall, delayed verbal recall, Serial 7's, and the TICS. In each case, analysis began with the simplest person-level model to establish sufficient variability in the intercept and slopes and to provide model fit comparisons (results not shown). Later models included gender, years of education, Black and Hispanic ethnicity, the presence of hypertension, diabetes, heart disease, history of stroke, the practice effect, and a variable that represented baseline age and time over the study period. After main-effects models were constructed to determine the baseline effects of the variables on cognitive functioning, interaction terms for Age × Education were entered into the models to examine differences in the rate of change by education. Interaction terms were also tested for each covariate to determine if the rate of cognitive decline was affected by other characteristics. Quadratic terms for age were also included as interaction terms with education to test for the presence of a curvilinear pattern of cognitive decline.

Results

After adjusting for covariates, scores on each test declined significantly as age increased (Tables 2 and 3). There were no significant nonlinear associations between age and word-recall or Serial 7's performance; scores on these tests declined progressively with age (Table 2). However, there was a significant nonlinear effect of age on TICS scores. On this test, scores initially remained fairly stable but began to decline more rapidly at older ages (Table 3).

As expected, years of education were positively related to higher baseline scores on each of the cognitive tests; the effect of an additional year of education was large, particularly relative to the effect of an additional year of age. For each additional year of education, baseline scores on each of the cognitive tasks were about 0.1 to 0.2 points higher. Substantively, this translates into an effect such that an older adult with 16 years of schooling or a college education scored about 0.4 to 0.8 points higher at baseline than a respondent with only 12 years of education.

Education was also related to the rate of longitudinal decline in cognitive performance, although the direction of this relationship varied by cognitive task. For delayed and immediate recall, the significant negative coefficient representing the interaction of age and education indicates that higher education was related to faster decline on these tasks. In contrast, the lack of significance of the Age × Education coefficient on the Serial 7's task suggests that education had no effect on the rate of cognitive decline. Finally, the positive sign on this interaction in the TICS model indicates that education was related to a slower decline on this task. There was also a significant interaction between education and age squared. The resulting trajectory in the TICS score among those with more years of education was more stable than that of their less educated counterparts, whose TICS scores declined more rapidly after age 80. These differences were statistically significant even when controlling for robust predictors of cognitive decline such as age, physical health, and, most important, baseline cognitive ability.

To illustrate these different trajectories, Figures 1 and 2 display estimated age-specific scores on the basis of fixed effects of the delayed verbal recall and TICS models at four different education levels. Comparing older adults with 4 versus 16 years of education, initial adjusted mean scores were 2.5 and 4.9, respectively, on the delayed-recall test. By age 90, those older adults with 4 years of education remembered only 1.0 words on the delayed recall test, whereas their peers with 16 years of education recalled 2.0 words. Thus, persons with more education scored higher on delayed verbal recall at all ages, but they also experienced a more rapid rate of decline than respondents with less education, averaging a loss of 2.9 words versus 1.5 words over the 20 years. However, on the TICS, persons with more education scored higher and experienced a slower rate of decline relative to respondents with less education. Older persons with 4 years of education demonstrated an initial average score of 8.0, declining to 6.6 by age 90, whereas those with 16 years of education demonstrated an initial average score of 9.7 and only declined by 1 point, to an average score of 8.7. Essentially, cognitive performance among education groups converged with age on the delayed recall task, whereas cognitive performance on the TICS diverged with age.

Race and ethnicity, gender, health conditions, and the practice effect were also related to baseline cognitive score. Blacks had lower baseline scores on each cognitive task relative to Whites, and Hispanics had lower baseline scores on the Serial 7's task. Women had higher baseline scores on verbal recall tasks but lower scores on the Serial 7's. History or onset of diabetes was related to lower baseline scores on verbal recall measures, and stroke was related to lower baseline scores on each cognitive task. Hypertension was related to higher mental status, but this relationship may be related to the inclusion of hypertension as a time-varying covariate. Those who were diagnosed with hypertension during the study period may have represented a healthier population than those who entered the study with hypertension, but this measure assessed variation in the group with either prevalent or incident hypertension.

In an effort to investigate other sources of variance in the slope of cognitive change, we tested interactions between other all independent variables and time. Gender was a significant predictor of cognitive change in verbal recall and Serial 7's, such that cognitive decline occurred at a faster rate in women. Racial and ethnic differences varied by cognitive task, with Blacks experiencing a slower decline relative to Whites on tests of immediate word recall and delayed word recall and Hispanics experiencing a faster decline on the TICS. No racial differences

were present in the rate of decline on the Serial 7's test. Finally, heart disease was the only health indicator that resulted in differential cognitive decline; individuals who had or developed heart disease experienced a slower decline on the delayed word recall.

Discussion

The purpose of this analysis was to determine whether education resulted in differential cognitive change among a representative sample of Americans over 70 years of age. Results across a range of cognitive domains suggest that education is related to significantly higher baseline cognitive function; however, the link between education and cognitive decline differed by cognitive domain. Because this study is the first to investigate a relationship between education and the rate of cognitive decline within a nationally representative sample of older adults that accounts for the racial and educational heterogeneity of the older population in the United States, our findings provide another perspective as to whether educational achievement is “protective” against cognitive decline in old age, as some other published studies have suggested (Albert et al. 1995; Evans et al. 1993; Lysetkos et al. 1999). Rather, these findings support observations made by Anstey and Christensen (2000) in a review of existing research on the relationship between education and cognitive change, suggesting that education was more consistently protective on mental status measures, but not on measures of fluid intelligence.

Education appears to slow decline in general mental status, supporting an active cognitive reserve hypothesis, such that individuals with higher educational attainment may process the simple tasks included in the TICS more efficiently. However, although they had higher ability at any given age, individuals with higher levels of education actually experienced greater absolute decline than those with less education in the more complex verbal and working memory tasks, including immediate and delayed word recall and the Serial 7's. The proportion of cognitive ability lost was similar across educational groups, but those with the highest education experienced greater absolute decline. For instance, although those who began with higher ability experienced a greater loss of performance over time and those with lower baseline ability experienced a smaller loss of performance over time, all group scores dropped by 23% to 25% over a 7-year period on delayed word recall. Education is clearly not protective in this area, because cognitive function declines similarly across all groups, and the greatest effects are observed among those with the highest education and highest initial performance. This finding suggests that educated adults over 70 years of age may begin to lose the ability to draw on education to compensate for declines in organic cognitive function, supporting the compensation hypothesis.

However, there are several alternate explanations for these findings. It may be possible that the less educated respondents in our sample showed a greater rate of decline in cognitive performance before age 70, prior to baseline data collection. In this scenario, the rate of cognitive decline for less educated older adults could be greater earlier in life (compared with well-educated older adults), but slower after age 70. On the basis of the same rationale, it is possible that well-educated respondents had very minimal declines in verbal recall or general mental status earlier in life (before baseline) but that their greatest rate of decline is observable after age 70. Therefore, our findings may reflect an observed “delayed onset” of degeneration in performance for well-educated older adults, if decline was postponed past age 70. A longer study observation period, including age groups younger than 70, could result in different outcomes. This possibility corresponds with what we know about deterioration in different cognitive abilities. For instance, working memory, as measured by the Serial 7's, is likely to show the first age-related changes in cognitive function. If adults in their 60s were included in our analysis, we might have observed a greater rate of decline in this measure among those with lower educational attainment.

Additionally, results may have been affected by methodological limitations of the study. Specifically, the effects of education may be limited by ceiling and floor effects inherent in cognitive measures that narrow the potential range of decline. For example, the TICS is a general mental status measure tapping orientation and language skills; changes in these abilities tend to have a later age of onset and are most pronounced in older adults with organic brain damage or dementing conditions (Albert 1994). Therefore, our observation that older adults with fewer years of education demonstrated a curvilinear decline on the TICS, whereas decline for those with more years of education remained relatively constant, could be the result of ceiling effects in this test that limit the variability of change for well-educated older adults with higher baseline scores. More sensitive measures that tap greater variability in cognitive function might better address educational differences in future research.

Furthermore, the exclusion of proxy reports may have affected results. A significant number of respondents ($n = 638$) were excluded because they lacked at least one time point of cognitive performance measurement, and another 15.6% of the sample ($n = 1,207$) had at least one data point missing because of proxy reporting. Education was associated with proxy status, so that those with fewer years of education were more likely to have at least one proxy report. In an effort to assess potential confounding in the Age × Education relationship by proxy status, we first attempted to control for proxy status by running regressions with a variable indicating whether a participant ever had a proxy report. Participants who ever had proxy reports had lower baseline cognitive scores in all four domains. However, the Age × Education relationship was not significantly different among these individuals. To further investigate the effect of including those with proxy reports in our sample, we excluded participants with at least one proxy report. The exclusion of these individuals did not change the significance or direction of the relationship between education and cognitive change. Unfortunately, we cannot estimate the effect of excluding those 638 participants with exclusively proxy reports. If those with the lowest education were both more likely to have proxy reports and to have a faster rate of cognitive decline, we may have underestimated the effect of education on cognitive decline in this group. Furthermore, if proxy status was associated with rapid declines not observed in previous waves, we would underestimate the effect of education on cognitive decline. Although sensitivity analysis suggests that the inclusion of those with at least one complete cognitive assessment and one or more proxy reports does not appear to have biased results, differential attrition due to proxy reporting may in part account for the lack of a protective effect of education on cognitive decline observed in this sample.

Similarly, we cannot completely account for differential selection due to mortality. By controlling for sociodemographic and health characteristics closely associated with mortality, we attempted to minimize the potential confounding effects of mortality. However, if those with lower education levels had faster rates of cognitive decline and were more likely to die, we might not observe the true effects of lower levels of education on cognitive decline.

Finally, we should note that although this sample is generally representative of Americans aged 70 years and older, there are some limitations to the generalizability of this analysis. First, because we included only respondents who answered questions assessing cognitive ability, our data likely do not reflect the relationship of education to cognitive decline among older adults with initially moderate to severe cognitive impairment who were interviewed through proxy respondents at all waves. Second, the initial AHEAD sample consisted of community-dwelling older adults, eliminating nursing home residents, a group with significant cognitive deficits. Although in later waves, interviews were attempted with persons in nursing homes, the declines observed here probably do not reflect those of an institutionalized population.

Unfortunately, our ability to examine the pathways through which education influences cognitive decline is limited. Education may be related both to cognitive development early in

life and to the maintenance of cognitive skills later in life through its associations with socioeconomic status. It is difficult without better data on early life to determine which of these periods of influence is more closely related to the rate of cognitive decline. Furthermore, education occurs in a social environment, and the meaning of education may be different for different individuals. Other measures, such as literacy, may better address the meaning of education in a population in which the quality of education is highly variable (Manly et al. 2003). Finally, there are a number of biological correlates that may modulate or mediate the relationship between age and education that we were unable to address in this study. Health status may play an important role, but the self-reports of disease used were likely to be confounded by education and access to health care. The use of objective biomarkers of disease processes would improve our ability to estimate these relationships. Education also appears to change brain function with age (Springer et al. 2005), and recent articles suggest potential interactions between genetic background and education (Seeman et al. 2005; Winnock et al. 2002) in predicting cognitive decline.

Despite these limitations, this study adds to the present understanding of cognitive decline by examining longitudinal cognitive change across multiple domains in a sample representative of the U.S. older population. In addition to educational variation, gender, race and ethnicity, health status, and prior exposure to the test were important predictors of baseline cognition. Interestingly, prior test exposure had a relatively large effect, particularly on the verbal recall tasks. Results also suggest that the rate of cognitive change varies by gender, race and ethnicity, and history of heart disease, issues that should be explored more fully in future research. Differences by gender and race may in part reflect differences in the quality and meaning of education for women and minority groups, particularly among older cohorts.

This study highlights the fact that early-life educational experiences are related not only to long-term cognitive performance but also to differential cognitive trajectories in old age. The effect of education on age-associated cognitive change varied by cognitive task, suggesting a loss of compensation ability among more educated older adults on verbal and working memory tasks but continued active reserve that can be applied to boost general mental status. The results demonstrate that cognitive performance across education groups converged with age on the fluid intelligence tasks, whereas cognitive performance diverged with age on the TICS. More highly educated adults scored higher at any given age on all tasks, and education continued to be protective of declines in mental status, which are closely related to dementia.

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References

Albert, Marilyn. Age Related Changes in Cognitive Function. In: Albert, Martin L.; Knoefel, Janice E., editors. Clinical Neurology of Aging. New York: Oxford University Press; 1994. p. 315-28.

Albert, Marilyn. How Does Education Affect Cognitive Function? Annals of Epidemiology 1995;5:76–78. [PubMed: 7728289]

Albert, Marilyn S.; Jones, Kenneth; Savage, Cary R.; Berkman, Lisa; Seeman, Teresa; Blazer, Dan; Rowe, JW. Predictors of Cognitive Change in Older Persons: Macarthur Studies of Successful Aging. Psychology & Aging 1995;10:578–89. [PubMed: 8749585]

Andel, Ross; Vigen, Cheryl; Mack, Wendy J.; Clark, Linda J.; Gatz, Margaret. The Effect of Educational and Occupational Complexity on Rate of Cognitive Decline in Alzheimer's Patients. Journal of the International Neuropsychological Society 2006;12:147–52. [PubMed: 16433954]

Anstey, Kaarin; Christensen, Helen. Education, Activity, Health, Blood Pressure and Apolipoprotein E as Predictors of Cognitive Change in Old Age: A Review. Gerontology 2000;46:163–77. [PubMed: 10754375]

Ardila, Alfredo; Ostrosky-Solis, Feggy; Rosselli, Monica; Gomez, Cesar. Age-Related Cognitive Decline during Normal Aging: The Complex Effect of Education. Archives of Clinical Neuropsychology 2000;15:495–513. [PubMed: 14590204]

Baltes, Paul. The Aging Mind: Potential and Limits. The Gerontologist 1993;33:580–94. [PubMed: 8225002]

Barrett-Connor, Elizabeth; Kritz-Silverstein, Donna. Gender Differences in Cognitive Function with Age: The Rancho Bernardo Study. Journal of the American Geriatrics Society 1999;47:159–64. [PubMed: 9988286]

Brandt, Jason; Spencer, Miriam; Folstein, Marshal F. The Telephone Interview for Cognitive Status. Neuropsychiatry, Neuropsychology, and Behavioral Neurology 1988;1:111–17.

Butler, Steven M.; Ashford, J Wesson; Snowdon, David A. Age, Education, and Changes in the Mini-Mental State Exam Scores of Older Women: Findings from the Nun Study. Journal of American Geriatrics Society 1996;44:675–81.

Cagney, Kathleen A.; Lauderdale, Diane S. Education, Wealth, and Cognitive Function in Later Life. Journals of Gerontology: Psychological Sciences 2002;57B:163–72.

Dorit, Carmelli; Swan, Gary E.; LaRue, Asenath; Eslinger, Paul J. Correlates of Change in Cognitive Function in Survivors from the Western Collaborative Group Study. Neuroepidemiology 1997;16:285–95. [PubMed: 9430128]

Christensen, Helen; Hofer, Scott M.; MacKinnon, Andrew J.; Korten, Aillsa E.; Jorm, Anthony F.; Henderson, A Scott. Age Is No Kinder to the Better Educated: Absence of an Association Investigated Using Latent Growth Techniques in a Community Sample. Psychological Medicine 2001;31:15–28. [PubMed: 11200953]

Christensen, Helen; Korten, Ailsa E.; Jorm, Anthony F.; Henderson, A Scott; Jacomb, Patricia A.; Rodgers, Bryan; Mackinnon, Andrew J. Education and Decline in Cognitive Performance: Compensatory but Not Protective. International Journal of Geriatric Psychiatry 1997;12:323–30. [PubMed: 9152716]

Colsher, Patricia L.; Wallace, Robert B. Longitudinal Application of Cognitive Function Measures in a Defined Population of Community-Dwelling Elders. Annals of Epidemiology 1991;3:215–30. [PubMed: 1669503]

Compton, David M.; Bachman, Lara D.; Brand, Darren; Avet, Traci L. Age-Associated Changes in Cognitive Function in Highly Educated Adults: Emerging Myths and Realities. International Journal of Geriatric Psychiatry 2000;15:75–85. [PubMed: 10637408]

Diamond, Marian C. Enriching Heredity: The Impact of the Environment on the Anatomy of the Brain. New York: Free Press; 1988.

Evans, Denis A.; Beckett, Laurel A.; Albert, Marilyn S.; Herbert, Liesi E.; Scherr, Paul A.; Funkenstein, H Harris; Taylor, James O. Level of Education and Change in Cognitive Function in a Community Population of Older Persons. Annals of Epidemiology 1993;3:71–77. [PubMed: 8287159]

Farmer, Mary E.; Kittner, Steven J.; Rae, Don S.; Bartko, John J.; Regier, Darrel A. Education and Change in Cognitive Function: The Epidemiologic Catchment Area Study. Annals of Epidemiology 1995;5:1–7. [PubMed: 7728280]

Folstein, Marshal F.; Folstein, Susan E.; McHugh, Paul R. Mini-Mental State: A Practical Guide for Grading the Cognitive State of Patients for the Clinician. Journal of Psychiatric Research 1975;12:189–98. [PubMed: 1202204]

Fratiglioni, Laura; Viitanen, Matti; von Strauss, Eva; Tontodonati, V.; Herlitz, Agneta; Winblad, Bengt. Very Old Women at Highest Risk of Dementia and Alzheimer's Disease: Incidence Data from the Kungsholmen Project, Stockholm. Neurology 1997;48:132–38. [PubMed: 9008508]

Glymour, M Maria; Weuve, Jenniver; Berkman, Lisa F.; Kawachi, Ichiro; Robins, James M. When Is Baseline Adjustement Useful in Analysis of Change? An Example with Education and Cognitive Change. American Journal of Epidemiology 2005;162:267–78. [PubMed: 15987729]

Herzog, A Regula; Wallace, Robert B. Measures of Cognitive Functioning in the AHEAD Study. Journals of Gerontology: Psychological and Social Sciences 1997;52B(Suppl):37–48.

Horn, John L.; Cattell, Raymond B. Refinement and Test of the Theory of Fluid and Crystallized Intelligence. Journal of Educational Psychology 1966;57:253–70. [PubMed: 5918295]

Hultsch, David F.; Hertzog, Christopher; Dixon, Roger A.; Small, Brent J. Memory Change in the Aged. Cambridge, MA: Cambridge University Press; 1998.

Jacqmin-Gadda, Helene; Fabrigoule, Colette; Commenges, Daniel; Dartigues, Jean-Francois. A 5-Year Longitudinal Study of the Mini-Mental State Examination in Normal Aging. American Journal of Epidemiology 1997;145:498–506. [PubMed: 9063339]

Jacobs, Bob; Schall, Matthew; Scheibel, Arnold B. A Quantitative Dendritic Analysis of Wernicke's Area in Humans. II. Gender, Hemispheric, and Environmental Changes. Journal of Comparative Neurology 1993;327:97–111. [PubMed: 8432910]

Kramer, Arthur F.; Bherer, Louis; Colcombe, Stanley J.; Dong, Willie; Greenough, William T. Environmental Influences on Cognitive and Brain Plasticity during Aging. Journal of Gerontology: Medical Sciences 2004;54A:940–57.

Lee, Sunmin; Kawachi, Ichiro; Berkman, Lisa F.; Grodstein, Francine. Education, Other Socioeconomic Indicators, and Cognitive Function. American Journal of Epidemiology 2003;157:712–20. [PubMed: 12697575]

Leibovici, Didier; Ritchie, Karen; Ledesert, Bernard; Touchon, Jacques. Does Education Level Determine the Course of Cognitive Decline? Age and Ageing 1996;25:392–97. [PubMed: 8921146]

Littell, Ramon C.; Milliken, George A.; Stroup, Walter W.; Wolfinger, Russel D. SAS System for Mixed Models. Cary, NC: SAS Institute; 1996.

Lykestos, Constantine G.; Chen, Li-Shiun; Anthony, James C. Cognitive Decline in Adulthood: An 11.5 Year Follow-Up of the Baltimore Epidemiological Catchment Area Study. American Journal of Psychiatry 1999;156:58–65. [PubMed: 9892298]

MacKnight, Chris; Rockwood, Kenneth; Awalt, Erin; McDowell, Ian. Diabetes Mellitus and the Risk of Dementia, Alzheimer's Disease and Vascular Cognitive Impairment in the Canadian Study of Health and Aging. Dementia and Geriatric Cognitive Disorder 2002;14:77–83.

Manly, Jennifer J.; Jacobs, Diane. Future Directions in Neuropsychological Assessment with African Americans. In: Ferrarro, F Richard, editor. Minority and Cross-Cultural Aspects of Neuropsychological Assessment. Bristol, PA: Swets & Zeitlinger; 2001. p. 79-96.

Manly, Jennifer J.; Touradji, Pegah; Tang, Ming-Xin; Stern, Yaakov. Literacy and Memory Decline among Ethnically Diverse Elders. Journal of Clinical and Experimental Neuropsychology 2003;25:680–90. [PubMed: 12815505]

Alewign, Ott; Breteler, Monique MB.; van Harskamp, Frans; Stijnen, Theo H.; Hofman, Albert. Incidence and Risk of Dementia: The Rotterdam Study. American Journal of Epidemiology 1998;147:574–80. [PubMed: 9521184]

Posner, Holly B.; Tang, Ming X.; Luchsinger, Jose; Lantigua, Rafael; Stern, Yaakov; Mayeux, Richard. The Relationship of Hypertension in the Elderly to AD, Vascular Dementia, and Cognitive Function. Neurology 2002;53:1175–81. [PubMed: 11971083]

Raudenbush, Stephen W.; Bryk, Anthony S. Hierarchical Linear Models: Applications and Data Analysis Methods. Vol. 2nd. Thousand Oaks, CA: Sage; 2002.

Reynolds, Chandra A.; Gatz, Margaret; Pedersen, Nancy L. Individual Variation for Cognitive Decline: Quantitative Methods for Describing Patterns of Change. Psychology and Aging 2002;17:271–87. [PubMed: 12061412]

Richards, Marcus; Hardy, Rebecca; Wadsworth, Michael EJ. Does Active Leisure Protect Cognition? Evidence from a National Birth Cohort. Social Science & Medicine 2003;56:785–92. [PubMed: 12560011]

Richards, Marcus; Sacker, Amanda. Lifetime Antecedents of Cognitive Reserve. Journal of Clinical and Experimental Neuropsychology 2003;25:614–24. [PubMed: 12815499]

Rodgers, Willard L.; Ofstedal, Mary Beth; Herzog, A Regula. Trends in Scores on Tests of Cognitive Ability in the Elderly U.S. Population, 1993-2000. Journal of Gerontology: Social Sciences 2003;58B:S338–46.

Ruitenberg, Annemieke; Skoog, Ingmar; Ott, Alewign; Aevarsson, Olafur; Witteman, Jaqueline CM.; Lernfelt, Bodil; van Harskamp, F.; Hofman, A.; Breteler, MM. Blood Pressure and Risk of Dementia:

Results from the Rotterdam Study and the Gothenburg H-70 Study. Dementia and Geriatric Cognitive Disorders 2001;12:33–39. [PubMed: 11125239]

Salthouse, Timothy A. Theories of Cognition. In: Bengtson, Vern L.; Schaie, K Warner, editors. Handbook of Theories of Aging. New York: Springer; 1999. p. 196-208.

Schaie, K Warner. Intellectual Development in Adulthood: The Seattle Longitudinal Study. New York: Cambridge University Press; 1996.

Seeman, Teresa E.; Huang, Mei-Hua; Bretsky, Philip; Crimmins, Eileen; Launer, Lenore; Guralnik, Jack. Education and ApoE-e4 in Longitudinal Cognitive Decline: MacArthur Studies of Successful Aging. Journals of Gerontology: Psychological Sciences 2005;60B:P74–83.

Singer, Judith D. Using SAS PROC MIXED to Fit Multilevel Models, Hierarchical Models, and Individual Growth Curve Models. Journal of Educational and Behavioral Studies 1998;24:323–55.

Soldo, Beth J.; Hurd, Michael J.; Rodgers, Willard L.; Wallace, Robert B. Asset and Health Dynamics among the Oldest-Old: An Overview of the AHEAD Study. Journals of Gerontology 1997;52B (Suppl):1–20. [PubMed: 9215354]

Springer, Mellanie V.; McIntosh, Anthony R.; WInocur, Gordon; Grady, Cheryl L. The Relation between Brain Activity during Memory Tasks and Years of Education in Young and Older Adults. Neuropsychology 2005;19:181–92. [PubMed: 15769202]

Stern, Yaakov. What Is Cognitive Reserve? Theory and Research Application of the Reserve Concept. Journal of the International Neuropsychological Association 2002;8:448–60.

Stern, Yaakov; Albert, Steven; Tang, Ming-Xin; Tsai, Wei-Yen. Rate of Memory Decline in AD Is Related to Education and Occupation. Neurology 1999;53:1942–47. [PubMed: 10599762]

Unverzagt, Frederick W.; Hui, Sui L.; Farlow, Martin R.; Hall, Kathleen S.; Hendrie, Hugh C. Cognitive Decline and Education in Mild Dementia. Neurology 1998;50:181–85. [PubMed: 9443477]

Wechsler, David. Manual for the Wechsler Adult Intelligence Scale Revised. New York: Psychological Corporation; 1981.

Whitfield, Keith E. Challenges in Cognitive Assessment of African Americans in Research on Alzheimer Disease. Alzheimer Disease and Associated Disorders 2002;15:S80–81. [PubMed: 12351919]

Winnock, Maria; Letenneur, Luc; Jacqmin-Gadda, Helene; Dallongeville, Jean; Amouyel, Phillipe; Dartigues, Jean-Francois. Longitudinal Analysis of the Effect of Apolipoprotein E e4 and Education on Cognitive Performance in Elderly Subjects: The PAQUID Study. Journal of Neurology, Neurosurgery, & Psychiatry 2002;72:794–97.

Zelinski, Elizabeth M.; Burknight, Kerry P. Sixteen Year Longitudinal and Time Lag Changes in Memory and Cognition in Older Adults. Psychology & Aging 1997;12:503–13. [PubMed: 9308097]

Zelinski, Elizabeth M.; Crimmins, Eileen; Reynolds, Sandra; Seeman, Teresa. Do Medical Conditions Affect Cognition in Older Adults? Health Psychology 1998;17:504–12. [PubMed: 9848800]

Zhu, Li; Fratiglioni, Laura; Guo, Zhenchao; Aguero-Torres, Hedda; Winblad, Bengt; Viitanen, Matti. Association of Stroke with Dementia, Cognitive Impairment, and Functional Disability in the Very Old. Stroke 1997;29:2094–99. [PubMed: 9756588]

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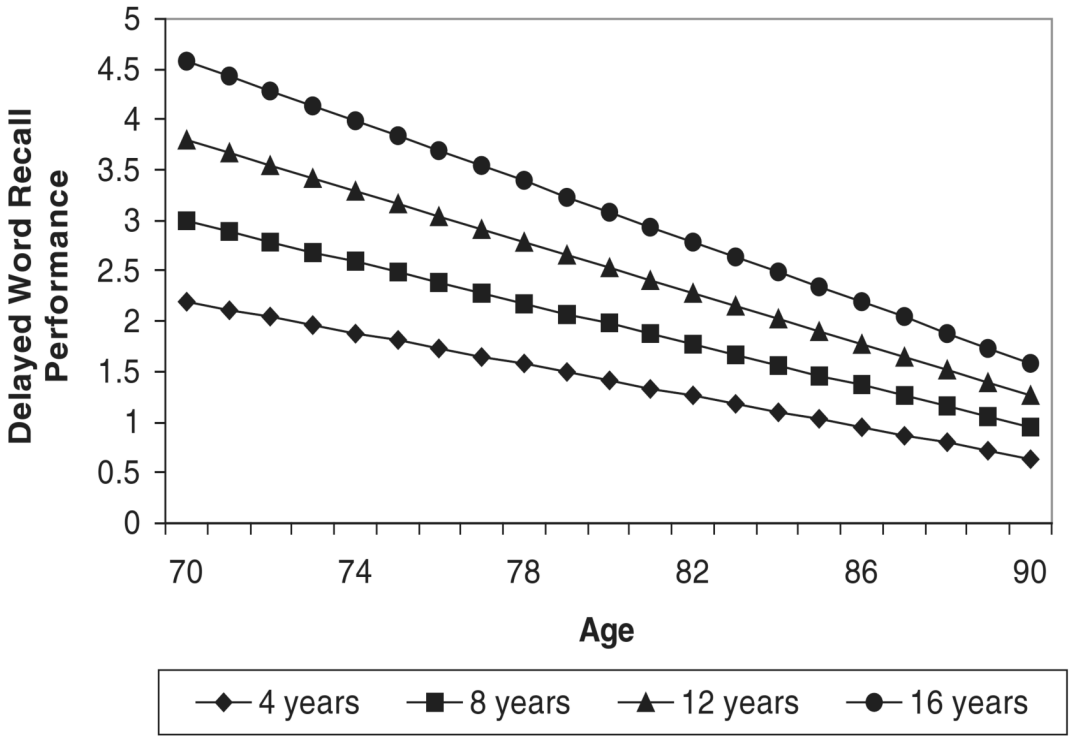

Figure 1. Estimated Score on Delayed Verbal Recall by Age at Four Levels of Education (years of school completed)

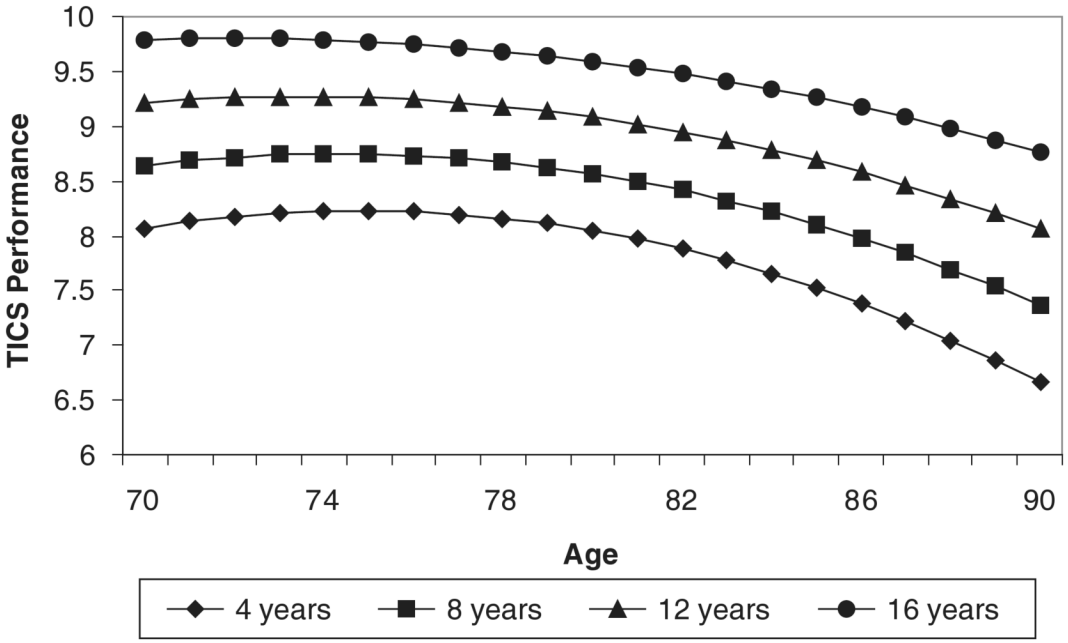

Figure 2. Estimated Score on the Telephone Interview for Cognitive Status (TICS) by Age at Four Levels of Education (years of school completed)

Table 1

Weighted Baseline Characteristics of the Asset and Health Dynamics of the Oldest Old Sample: *M* ± *SD* or Percentage (*n* = 6,805)

Characteristic	Value
Demographics	
Age (years)	77.2 ± 5.6
Education (years)	11.1 ± 3.5
Female	62.9%
Married	49.6%
Black	9.8%
Hispanic	3.5%
Hypertension	50.3%
Diabetes	12.3%
Heart disease	31.2%
Stroke	7.4%
Cognitive performance	
Verbal recall	4.6 ± 1.9
Delayed recall	3.2 ± 2.2
Telephone interview for cognitive status	8.9 ± 1.6
Serial 7's	3.1 ± 1.9

Table 2

Regression Estimates Associated With Level and Slope of Verbal and Working Memory in the Asset and Health Dynamics of the Oldest Old Sample: Linear Trajectory

	Delayed Verbal Recall (n = 6,351)			Immediate Verbal Recall (n = 6,441)			Serial 7's (n = 6,404)		
Effect	**Estimate**	***SE***	***Pr*** > |***t***|	**Estimate**	***SE***	***Pr*** > |***t***|	**Estimate**	***SE***	***Pr*** > |***t***|
Intercept	0.869	0.107	<.0001	2.076	0.090	<.0001	2.267	0.090	<.0001
Intercept variance	1.477	0.046	<.0001	1.046	0.330	<.0001	1.021	0.030	<.0001
Age	−0.054	0.009	<.0001	−0.066	0.008	<.0001	−0.036	0.008	<.0001
Age variance	0.010	0.009	<.0001	0.032	0.007	<.0001	0.003	0.001	<.0001
Education	0.156	0.007	<.0001	0.161	0.005	<.0001	0.172	0.005	<.0001
Age × Education	−0.006	0.001	<.0001	−0.003	0.001	<.0001	0.000	0.001	.9496
Black	−0.686	0.063	<.0001	−0.521	0.054	<.0001	−1.140	0.055	<.0001
Hispanic	−0.106	0.101	.2930	−0.055	0.085	.5186	−0.346	0.084	<.0001
Female	0.384	0.040	<.0001	0.463	0.034	<.0001	−0.455	0.033	<.0001
Hypertension	0.001	0.035	.9847	0.016	0.030	.5978	0.011	0.029	.6948
Diabetes	−0.161	0.050	.0013	−0.165	0.042	.0001	−0.069	0.040	.0885
Heart disease	−0.063	0.034	.0697	−0.052	0.029	.0761	−0.042	0.030	.1547
Stroke	−0.391	0.008	<.0001	−0.393	0.048	<.0001	−0.368	0.047	<.0001
Practice	0.399	0.027	<.0001	0.257	0.023	<.0001	−0.047	0.021	.0258

Table 3

Regression Estimates Associated With Level and Slope of General Mental Status in the Asset and Health Dynamics of the Oldest Old Sample: Nonlinear Trajectory

	Telephone Interview for Cognitive Status (*n* = 6,651)		
Effect	**Estimate**	***SE***	***Pr*** **>** **|*t*|**
Intercept	7.837	0.074	<.0001
Intercept variance	0.620	0.023	<.0001
Age	–0.032	0.010	.0007
Age variance	0.109	0.008	<.0001
Age^2	–0.008	0.001	<.0001
Education	0.127	0.004	<.0001
Age × Education	–0.0008	0.001	.9231
Age^2 × Education	0.0003	0.0001	.0003
Black	–1.002	0.044	<.0001
Hispanic	–0.377	0.069	<.0001
Female	–0.051	0.028	.0659
Hypertension	0.077	0.024	.0014
Diabetes	–0.042	0.035	.2280
Heart disease	–0.067	0.024	.0049
Stroke	–0.319	0.041	<.0001
Practice	0.080	0.018	<.0001