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Cohorts based on Decade of Death: No Evidence for Secular Trends Favoring Later Cohorts in Cognitive Aging and Terminal Decline in the AHEAD Study

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Abstract

Studies of birth-year cohorts examined over the same age range often report secular trends favoring later-born cohorts, who are cognitively fitter and show less steep cognitive declines than earlier-born cohorts. However, there is initial evidence that those advantages of later-born cohorts do not carry into the last years of life, suggesting that pervasive mortality-related processes minimize differences that were apparent earlier in life. Elaborating this work from an alternative perspective on cohort differences, we compared rates of cognitive aging and terminal decline in episodic memory between cohorts based on the year participants had died, earlier (between 1993 and 1999) or later in historical time (between 2000 and 2010). Specifically, we compared trajectories of cognitive decline in two death-year cohorts of participants in the Asset and Health Dynamics among the Oldest Old (AHEAD) Study that were matched on age at death and education and controlled for a variety of additional covariates. Results revealed little evidence of secular trends favoring later cohorts. To the contrary, the cohort that died in the 2000s showed a less favorable trajectory of age-related memory decline than the cohort who died in the 1990s. In examinations of change in relation to time-to-death, the cohort dying in the 2000s experienced even steeper terminal declines than the cohort dying in the 1990s. We suggest that secular increases in “manufacturing” survival may exacerbate age- and mortality-related cognitive declines among the oldest old.

Keywords

Episodic memory; Asset and Health Dynamics among the Oldest Old (AHEAD); longitudinal; cognitive aging; terminal decline; cohort differences

Lifespan research has long been interested in how individual development is shaped by historical and societal changes (Baltes, Cornelius, & Nesselroade, 1979; Riley, 1973; Ryder,

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1965; Schaie, 1965). Across birth-year cohorts, considerable increases in performance on measures of fluid intelligence over the past century are widely documented (e.g., Flynn, 1987, 2007) and have been suggested to result from advances in schooling, nutrition, and medical technology (Alwin & McCammon, 2001; Blair, Gamson, Thorne, & Baker, 2005; Hauser & Huang, 1997; Rönnlund, Nyberg, Bäckman, & Nilsson, 2005). Such secular trends favoring later-born cohorts suggest that culture-based efforts have been effective and successful in improving individual functioning at various phases of the life span. However, there is also some initial evidence that those secular trends favoring later-born cohorts do not carry into the last years of life (Gerstorf, Ram, Hoppmann, Willis, & Schaie, 2011). Our goal in the present study is to approach cohort differences from a different perspective. Aiming to examine secular trends in (the effects of) late-life living conditions, we move from the typical examination of birth-year cohorts, where cohorts are defined based on year of birth, towards an examination of death-year cohorts, where cohorts are defined based on year of death. We make use of longitudinal data on cognition obtained in the Asset and Health Dynamics among the Oldest Old (AHEAD) Study and compare a cohort who died earlier in historical time (between 1993 and 1999) with a cohort who died later (between 2000 and 2010). We examine differences between these death-year cohorts in rates of cognitive aging and terminal decline on the available cognitive marker (episodic memory). To control for known individual and cohort differences, we match the death-based cohorts on age at death and education, and (statistically) control for differences in age at first assessment, gender, number of health conditions, retest effects, and frequency of observations.

Differences in Cognitive Aging between Birth-Year Cohorts

When examined over the same age ranges, later-born cohorts are often cognitively fitter than earlier born cohorts. This general pattern of mean-level differences has been reported from a myriad of studies, and for young and old participants alike (Alwin, 2008; Bäckman, Small, Wahlin, & Larsson, 2000; Flynn, 2007; Schaie, 2005). When examining birth-year cohort differences in rates of cognitive change, however, the empirical evidence is less conclusive. Some studies report that later-born cohorts experienced, on average, shallower rates of cognitive aging across a variety of different abilities (Gerstorf, Ram et al., 2011; Schaie, Willis, & Pennak, 2005; see also Bowles, Grimm & McArdle, 2005). As a consequence, birth-cohort differences in cognitive functioning that existed at one age (e.g., age 70) were exacerbated later on (e.g., at age 80). In contrast, other studies found that birth-cohort differences were preserved throughout aging, but did not get larger with age (e.g., memory: Finkel, Reynolds, McArdle, & Pedersen, 2007; Zelinski & Kennison, 2007). That is, cognitive aging, as indicated by rates of change, essentially proceeded in parallel between the cohorts.

The secular trends favoring later birth-year cohorts have been linked to a variety of different factors, including advances in education (e.g., quantity of schooling: Alwin & McCammon, 2001; school curricula: Blair et al., 2005); nutrition and hygiene (Condran & Crimmins-Gardner, 1978; Martorell, 1998); changes in family size and educational attitudes (e.g., fewer children per family: Sundet, Borren, & Tambs, 2008; increased parental education and engagement: Grissmer, Kirby, Berends, & Williamson, 1994); and altered cognitive challenges through modern media and technologies (Greenfield, 1998). Of note is that many of those factors primarily operate in early or middle phases of life and are probably less influential in later phases of life. It remains an open question whether differences between birth-year cohorts who differ in the conditions that shape developmental processes in early-life and mid-life generalizes to death-year cohorts who differ primarily in the conditions that shape developmental processes in late-life (e.g., medical conditions). Late-life is often characterized by mortality-related terminal declines in multiple domains, such as cognition

(Bäckman & MacDonald, 2006) and well-being (Gerstorf, Ram et al., 2008, 2010). The identification of such processes requires moving from an age metric to a time-to-death metric. In the present study, we examine the importance of late-life conditions for shaping developmental outcomes by comparing declines in rates of episodic memory performance across cohorts using both age and mortality time metrics and by defining cohorts based on the decade of death rather than the year of birth.

Differences in Terminal Decline between Birth-Year Cohorts

There is some initial evidence suggesting that conditions very late in life do not operate to sustain the cohort differences observed earlier in life. In particular, although later-born cohorts (1914-1948) in the Seattle Longitudinal Study (SLS) considerably outperformed earlier-born cohorts (1883-1913) at age 70 and showed less steep rates of cognitive aging between 50 and 80 years, those differences were basically eliminated when examined in the last years of life (Gerstorf, Ram et al., 2011). Relative to the earlier-born cohorts, rates of terminal decline were steeper among the later-born cohorts on four of the five cognitive tests examined. The increased steepness may be the fallout (pun intended) of “manufactured survival”. That is, technological advances have helped later-born cohorts survive diseases or impairments that killed earlier-born cohorts (Olshansky, Hayflick, & Carnes, 2002). However, as Olshansky et al. (2002) speculated, the extension of life may have been achieved at the costs of losses in functioning (Crimmins & Beltran-Sanchez, 2011), thereby offsetting or even reversing secular trends favoring later cohorts that have existed at an earlier point in life. In such a scenario, historical improvements in health and cognitive functioning may not outweigh the detrimental effects of aging- and mortality-related processes. In the present study, we specifically examine whether or not people who died in different historical times experienced substantively different trajectories of cognitive aging and cognitive terminal decline.

Differences in Cognitive Aging and Terminal Decline between Death-Year Cohorts

Our major proposal is that cohorts of people differ in both their exposure to factors that shape developmental processes and outcomes in early and middle phases of adulthood and in their exposure to factors that mold the nature of late-life development. What are the factors affecting late-life development that have changed over the past several decades and how are these changing conditions potentially altering late-life cognitive change?

Several historical trends are of note. First, many diseases that are known to undermine cognitive functioning such as cardiovascular diseases, heart disease, heart attack, or stroke (Anstey, Mack, & von Sanden, 2006; Verhaeghen, Borchelt, & Smith, 2003) are nowadays associated with remarkably lower mortality rates than they have been some 20 or 30 years ago (e.g., mortality after heart attack decreased by 3% each year from 1994 to 2005; Setoguchi et al., 2008). As a consequence, the number of life-years people spend living in diseased states is on the rise.

Second, in recent decades there has been a marked increase in the prevalence of many debilitating diseases that are more or less directly linked to cognitive decline. For example, between 1997 and 2008 the mortality rate for Alzheimer’s disease among individuals aged 65 years or older increased by 183% (National Vital Statistics System, 1997-2008). Similarly, cellulites and abscess as causes for emergency room visits by 65+ year olds have increased by 81% over the same timeframe (National Hospital Ambulatory Medical Care Survey, 1997-2008), which could indicate a general weakening of older individuals’ immune systems. Associations between deteriorations of the immune system and brain

aging/cognitive decline have been well documented in animals (Brynskikh, Warren, Zhu, & Kipnis, 2007; Haley, Urbanski, Kohama, Messaoudi, & Raber, 2011, Ziv et al., 2006) and several streams of recent evidence suggest similar associations for human aging (for overview, see Ron-Harel & Schwartz, 2009). As a consequence, more and more people are faced with chronic conditions that also likely affect cognitive functioning (Crimmins & Beltran-Sanchez, 2011).

Third, although there has been an increase in the prevalence of chronic conditions among older adults from the 1990s to the 2000s, these diseases often lead to less severe forms of disability (Christensen, Doblhammer, Rau, & Vaupel, 2009; Freedman, Schoeni, Martin, & Cornman, 2007), probably due to both development of more effective medical treatments and advancement of assistive technologies (e.g., telephone with touchtone dialing). For medical treatments, however, we note that cognitive side effects of medication have been well documented (Ancelin et al., 2006; Gray et al., 1999, Larson et al., 1987). For example, drugs such as statins that are used to lower cholesterol levels are associated with cognitive decrements (Muldoon et al., 2004; Wagstaff et al., 2003). For assistive technologies, we draw on some of the key tenets of lifespan psychology to suggest that late life is characterized by an ever-declining efficiency of cultural resources to overcome age-related decrements (Baltes & Smith, 2003). For example, an assistive device such as a walking stick may suffice to maintain mobility when people are in their mid-seventies, whereas even a walking frame may not be sufficient to facilitate mobility when people are in the late eighties. Similarly, hearing aids often become less and less efficient in accommodating sensory (and cognitive) losses (Lindenberger et al., 2008). In sum, we contend that because of these interrelated historical developments, secular trends in cognitive functioning favoring later cohorts during earlier phases of life (Bowles et al., 2005; Finkel et al., 2007; Flynn, 2007; Gerstorf, Ram et al., 2011; Schaie et al., 2005; Zelinski & Kennison, 2007) are minimized in late-life.

The Current Study

Elaborating and extending previous work towards a perspective on cohort differences that focuses on late-life, the current study compares rates of cognitive aging and terminal decline in episodic memory between death-year cohorts who died earlier (between 1993 and 1999) or later in historical time (between 2000 and 2010). To control for known individual and cohort differences, we both covaried for age at the first measurement occasion, gender, health, retest and frequency of observations and used propensity score matching procedures to equate cohorts on age at death and education. Based on notions of “manufactured survival”, we expected that the secular trends favoring later cohorts documented in early adulthood and middle age are minimized or even reversed when examining late life.

Method

We used longitudinal data from the AHEAD study. Detailed descriptions of participants, variables, and procedures can be found in McArdle et al. (2007) and Soldo et al. (1997). Select details relevant to the present study are given below.

Participants and Procedure

The AHEAD study started in 1993 with a nationally representative probability sample of households in the United States that included non-institutionalized individuals aged 70 or older who were born prior to 1924 ($N = 8,222$). If more than one person in the household was aged 70 or older, one person was selected randomly as the study participant. If this person was married or living with a partner, the spouse or partner was asked regardless of her or his age to participate as well. Thus, the sample also included individuals who were

younger than 70 years old. Data have been collected every other year since in 1993. There was no data collection in 1997, when data collection was shifted to even numbered years (1998, 2000 ...) and joined with the Health and Retirement Study (HRS).

An $n = 5,123$ sample of AHEAD participants have since died. To make sure that the data informed our analysis of terminal decline, we excluded all participants who did not provide any data for the episodic memory measures in the 4 years preceding their death ($n = 801$). In the present study, we thus use data from the remaining subsample of $n = 4,322$ participants who died in earlier (1993-1999; earlier-deceased cohort; $n = 1,775$) or later in historical time (2000-2010; later-deceased cohort; $n = 2,547$)¹. In total, we utilized longitudinal data obtained on eight measurement occasions over 15 years (1993, 1995, 1998, 2000, 2002, 2004, 2006, and 2008).

Measures

Episodic memory was indexed by a unit-weighted composite of performance scores on immediate and delayed free recall tests. Detailed information can be found in Ofstedal et al. (2005). In brief, participants were presented a list of 10 nouns, and asked to recall as many words as possible both immediately after presentation and after a delay of approximately five minutes. Scores were calculated as the proportion (ranging from 0 to 1) of words correctly remembered, with higher scores indicating better performance.

Time metrics: Age and time-to-death—We calculated *age* at each measurement occasion as the number of years since an individual's birth. The age variable was coded as integer numbers and centered at 80 years. The age of 80 was chosen as the centering point as it was close to the mean age across all observations (81.02 years). The dense observations at around this age provided for relatively more stable parameter estimates relative to choosing other centering points (e.g., age 70 or age 90). *Time-to-death* at each occasion was calculated as the difference between the year of assessment and the year of an individual's death obtained either (a) directly from the participants' household or neighbors during the interview or (b) from city or national registries or offices (e.g., US National Death Index). Time-to-death was coded as integer numbers and centered at two years prior to death. We chose two years prior to death as the centering anchor in the time-to-death models because of relatively dense observations at this point. Keeping the intercept relatively near to the time of death means that terminal decline effects were very likely to have set in (e.g. Sliwinski et al., 2006; Wilson et al., 2003, 2007, 2012).

Covariates—A number of variables known to differ between individuals and cohorts were included as covariates into our models. *Age at death* was calculated as the difference between the year of an individual's death and his or her year of birth. *Age at TI* was calculated as the difference between an individual's birth year and the year at which their first assessment appeared in the data. *Education* was measured as the number of years the individual had spent in formal schooling. *Health conditions* were assessed at each wave of the AHEAD study as the sum index of self-reported physician-diagnosed current health problems, including high blood pressure, diabetes, cancer, lung disease, heart condition, stroke, psychiatric problems, and arthritis (Wilson-Genderson, Pruchno, & Cartwright, 2009). For our analyses, we used the number of health conditions obtained at the occasion most proximal to an individuals' death year because health effects are presumably strongest in the years immediately preceding death. Six participants from the later-deceased cohort who did not provide data on health conditions at the last measurement occasion were

¹Follow-up analyses showed that dividing the time period equally and contrasting an earlier-deceased (1993-2001) and a later-deceased (2002-2010) cohort leads to substantively similar findings.

excluded from further analyses. Individual differences in retest effects were modeled at the within-person level, assuming that familiarity with the testing procedure leads to increased performance after the initial testing (McArdle et al., 2007). Specifically, *retest* was coded 0 for the first data point of each individual (i.e. in 1993, when the test was taken for the first time) and 1 for all subsequent data points. *Number of observations* was the total number of observations from an individual that were included in the data (range 1–4).

Data preparation

The left portions of Table 1 provide descriptive statistics on socio-demographic measures for the two death-year cohorts examined in the present study.² As can be seen, the later-deceased cohort had, on average, received more years of education, included a greater proportion of women, and had lived a longer life. Of particular note, the AHEAD's longitudinal design meant that the earlier-deceased cohort could contribute a maximum of three observations, whereas the later-deceased cohort had the opportunity to provide up to eight observations.

We used four procedures to minimize these possible confounds and to equate the cohorts as closely as possible. First, to balance the opportunity to contribute observations to the data stream across cohorts, we selected only the last four observations for each participant. Age at T1 was redefined as the age at the first of these four observations.

Second, we made use of *propensity score matching* procedures (Coffman, 2011; Stuart, 2010; Jackson et al., in press; Thoemmes & Kim, 2011) to obtain matched cohort groups. In particular, we used 1:1 matching methods to pair each participant from the earlier-deceased cohort ($n = 1,775$) with a 'twin' participant from the later-deceased cohort ($n = 2,541$) who had the same (or as similar as possible) age at death and education. The probability of each individual being in the earlier or later-deceased cohort was estimated using a logistic regression including the background variables age at death and education. The propensity scores for each cohort group are given in Table 1. As recommended in the matching literature (e.g., Rosenbaum & Rubin, 1985), we then used logit-transformed propensity scores to calculate a between-group distance matrix. Nearest neighbors were matched via a caliper matching algorithm with a caliper (maximal allowable distance between matched participants), $c = .2 SD$, chosen so that group differences on matching variables became non-significant ($p > 0.05$). Each participant in the earlier-deceased cohort was allocated the nearest neighbor from the later-deceased cohort only if the neighbor fell within the caliper distance. When no suitable neighbor existed, the individual was flagged for removal. For 362 participants in the earlier-deceased cohort, no suitable neighbor was identified, thus reducing sample size to 1,413 participants per cohort group.

Third, we honed the distribution of observations across age and time-to-death. Because of sparse observations at both the low and high ends of the age distribution, we discarded all observations obtained prior to age 65 years (5 participants in the later-deceased cohort and 2 participants in the earlier-deceased cohort, 0.28 % of all observations) and after age 100 years (another 3 participants in the earlier-deceased cohort and 0.12 % of all observations). Final sample size for the matched and honed cohorts was 1,408 participants in the earlier-deceased cohort and 1,408 participants in the later-deceased cohort. Descriptive statistics for the matched samples are shown on the right panel of Table 1. It can be obtained that after the matching procedure, the cohorts did not differ on the matching variables age at death and

²To address the question of how biased the estimates obtained with an unweighted sample are, we reran the analysis using the HRS sample weights to see how much the parameters and standard errors might change. The parameters changed by relatively small amounts and there were no changes in significance. Even though these weights were based on a birth-cohort weighting scheme and not developed for death cohorts, the results suggest that the estimates would be similar in a correctly weighted analysis.

education, while cohort differences still existed in age at T1, gender and, health. Figure 1 illustrates the frequencies of observations over chronological age and time-to-death, separately for both death-year cohorts. It can be seen that our efforts to match observations obtained from the two death-year cohorts were reasonably successful.

Fourth, to control for the remaining (known) differences between the death-year cohort groups, we included age at T1, gender, number of health conditions, number of observations, and retest (time-varying) as additional covariates in our models. We note that these covariates might also have been included as part of the propensity score matching procedure. However, we found there was a substantial tradeoff between number of variables and resulting sample size. Thus, attempting to balance need for statistical control using covariates with sample-sized based statistical power to find group differences, we iteratively chose the variable sets and caliper length to minimize differences while maintaining cohort groups with sample sizes of $N > 1,000$.

The episodic memory score was scaled to a T-score metric ($M = 50$, $SD = 10$) using the first measurement occasion in 1993 of the subsample used in the present study as a reference. Table 2 shows descriptive statistics for episodic memory over age (left panel) and time-to-death (right panel), separately for the two matched cohorts. These data descriptively suggest that average levels of episodic memory decline with increasing age and proximity to death in both cohorts.

Data analysis

To examine cohort differences in how episodic memory changes with advancing age and at the end of life, we used chronological age and time-to-death as the two time metrics in our growth curve models. The models were specified at the within-person level (Level 1) as

$$\text{memory}_{it} = \beta_{0i} + \beta_{1i}(\text{time}_{it}) + \beta_{2i}(\text{retest}_{it}) + \beta_{3i}(\text{time}_{it}^2) + e_{it}, \quad (1)$$

where memory_{it} represents person i 's episodic memory at time t . β_{0i} is a person-specific intercept parameter, β_{1i} is a person-specific linear slope parameter that characterizes the rate of change per year in episodic memory. β_{2i} captures retest effects, β_{3i} is a quadratic slope parameter capturing the acceleration of change, and e_{it} is residual error. Person-specific intercept (β_{0i}) and linear (β_{1i}) and quadratic slope (β_{3i}) parameters, as well as retest effects (β_{2i}) were modeled at the between-person level (Level 2) for the age-related change model with as

$$\begin{aligned} \beta_{0i} = & \gamma_{00} + \gamma_{01}(\text{cohort}_i) + \gamma_{02}(\text{gender}_i) + \gamma_{03}(\text{education}_i) + \gamma_{04}(\text{health conditions}_i) \\ & + \gamma_{05}(\text{number of observations}_i) + u_{0i}, \end{aligned} \quad (2)$$

$$\begin{aligned} \beta_{1i} = & \gamma_{10} + \gamma_{11}(\text{cohort}_i) + \gamma_{12}(\text{gender}_i) + \gamma_{13}(\text{education}_i) + \gamma_{14}(\text{health conditions}_i) \\ & + \gamma_{15}(\text{number of observations}_i) + u_{1i}. \end{aligned} \quad (3)$$

$$\beta_{2i} = \gamma_{20} + u_{2i}, \quad (4)$$

$$\beta_{3i} = \gamma_{30}, \quad (5)$$

The time-to-death model additionally included age at T1 and age at death,

$$\begin{aligned}\beta_{0i} = & \gamma_{00} + \gamma_{01} (\text{cohort}_i) + \gamma_{02} (\text{gender}_i) + \gamma_{03} (\text{education}_i) + \gamma_{04} (\text{health conditions}_i) \\ & + \gamma_{05} (\text{number of observations}_i) + \gamma_{06} (\text{age at T1}_i) + \gamma_{07} (\text{age at death}_i) \\ & + u_{0i},\end{aligned}\quad (6)$$

$$\begin{aligned}\beta_{1i} = & \gamma_{10} + \gamma_{11} (\text{cohort}_i) + \gamma_{12} (\text{gender}_i) + \gamma_{13} (\text{education}_i) + \gamma_{14} (\text{health conditions}_i) \\ & + \gamma_{15} (\text{number of observations}_i) + \gamma_{16} (\text{age at T1}_i) + \gamma_{17} (\text{age at death}_i) \\ & + u_{1i}.\end{aligned}\quad (7)$$

The covariates were effect-coded (for dichotomous variables) or centered (for continuous variables) so that γ_{00} , γ_{10} , γ_{20} , and γ_{30} indicated the average trajectory across all individuals. Positive parameter estimates indicate differences favoring the later-deceased cohort, women, those with more education, better health, those who provided more data points, as well as participants with older ages at T1 and at death. Residual between-person differences u_{0i} , u_{1i} and u_{2i} were assumed to be multivariate normally distributed, correlated with each other and uncorrelated with the residual errors, e_{it} . Between-person differences in β_{3i} were not modeled because of the relatively small number of data points provided by each participant ($n = 4$). Models were estimated with SAS Proc Mixed (Littell, Miliken, Stoup, & Wolfinger, 1996) with incomplete data treated as missing at random (Little & Rubin, 1987).

Results

Differences in Cognitive Aging between Death-Year Cohorts

The left panel of Table 3 presents results from our growth model examining whether earlier- and later-deceased cohorts differed in age-related trajectories of episodic memory. Average level of episodic memory performance at 80 years ($\gamma_{00} = 49.685$) was about the same as the performance level at the first wave in 1993 ($M = 50$). The variance in average levels was reliably different from 0 after accounting for age trends and covariates ($\sigma^2_{u0} = 32.308$). In line with previous work (Finkel et al., 2007; Singer, Verhaeghen, Ghisletta, Lindenberger, & Baltes, 2003), episodic memory declined with increasing age. The linear component of episodic memory decline amounted to a little more than half standard deviation unit per decade ($\gamma_{10} = -0.613$ per year) with a quadratic component that was not significantly different from zero ($\gamma_{20} = 0.002$, $p = 0.385$). Of particular interest for our research question were the differences between earlier- and later-deceased cohorts in level and rates of decline in episodic memory. Results revealed that the later-deceased cohort showed both lower levels of episodic memory performance at age 80 years ($\gamma_{01} = -1.022$) and steeper age-related declines ($\gamma_{11} = -0.131$), compared to the earlier-deceased cohort. Panel A of Figure 2 illustrates these cohort differences in age-related changes in episodic memory.

Several covariates were also related to level and change of episodic memory. Retest effects were not reliably different from 0 at the sample level ($\gamma_{20} = 0.132$), however; there were individual differences in the amount the participants profited from increased familiarity with the testing procedure after the initial assessment, as indicated by the variance of the retest effect ($\sigma^2_{u2} = 6.603$). At the sample level, women and the more educated showed better memory performance ($\gamma_{02} = 1.817$; $\gamma_{03} = 0.705$, respectively), and in line with previous findings on longitudinal selection (Ghisletta, McArdle, & Lindenberger, 2006; Lindenberger, Singer & Baltes, 2002, McArdle, Hamagami, Elias, & Robbins, 1991), participants who provided more data points also demonstrated higher levels of cognitive functioning ($\gamma_{05} = 1.743$). Although their overall performance was higher, women and the more educated also showed slightly steeper age-related declines ($\gamma_{12} = -0.109$; $\gamma_{13} = -0.017$, respectively).

Differences in Terminal Decline between Death-Year Cohorts

The right panel of Table 3 presents results from our mortality-related growth model that examined cohort differences in terminal decline of episodic memory. At two years prior to death (the centering point), the average performance level ($\gamma_{00} = 47.217$) was about a quarter of a standard deviation lower than the overall level at the first wave in 1993 ($M = 50$). The variance in average levels was reliably different from 0 after accounting for terminal decline and covariates ($\sigma^2_{u0} = 32.838$). In line with previous reports of cognitive decrements associated with terminal decline (Bäckman & MacDonald, 2006), the linear component of episodic memory decline amounted to more than a full standard deviation per decade ($\gamma_{10} = -1.195$ per year) with some negative quadratic curvature ($\gamma_{20} = -0.059$), indicating an accumulation of lower scores proximate to death. Of primary interest for our research question were again the cohort differences in level and change of episodic memory performance over time-to-death. The mortality model revealed an even more drastic picture than the age model. The later-deceased cohort showed a less favorable terminal decline trajectory in episodic memory performance, characterized by lower levels of episodic memory performance at two years prior to death ($\gamma_{01} = -0.848$) and substantially steeper mortality-related declines ($\gamma_{11} = -0.437$) than the earlier-deceased cohort. Panel B of Figure 2 illustrates that those dying in the 2000s experienced steeper terminal declines than those dying in the 1990s.

Retest effects were reliably different from 0 at the sample level ($\gamma_{20} = 0.889$), indicating that increased familiarity with the testing procedure after the initial assessment led to improved performance. There were individual differences in the amount the participants profited from retesting ($\sigma^2_{u2} = 7.128$). Women and the more educated again showed higher levels of performance ($\gamma_{02} = 1.311$; $\gamma_{03} = 0.612$, respectively) and experienced slightly steeper mortality-related declines ($\gamma_{12} = -0.174$; $\gamma_{13} = -0.034$, respectively). Participants who contributed more data points were characterized by a higher overall episodic memory performance two years before death ($\gamma_{05} = 2.641$), again demonstrating predictive effects of cognition for longitudinal selection (Ghisletta et al., 2006; Lindenberger et al., 2002, McArdle et al., 1991). Age at T1 was positively associated ($\gamma_{06} = 0.903$), and age at death was negatively associated ($\gamma_{07} = -1.369$) with level of episodic memory two years prior to death, probably indicating both a selection effect (older participants at T1 may have come from a higher functioning segment of the population) and the expected frailty that accompanies terminal decline at later ages.

To rule out the possibility that the lack of cohort differences in favor of the later-deceased cohort was induced by study design features, we conducted two follow-up analyses. First, we noted that participants in the earlier-deceased cohort were measured in the last six years of life only, whereas observations from the later-deceased cohort go back until 15 years before death (see right side of Table 2). In an effort to further equate the groups, we excluded from our analyses all data points obtained seven or more years before death. Although this reduced the later-deceased cohort's data substantially (by 30 % or 1,574 data points), their typical terminal decline trajectory was significantly steeper than that of the earlier-deceased cohort ($\gamma_{10} = -1.102$, $p < .05$). Second, we conducted follow-up analyses based on a matched sample that paired participants both on age at T1 and age at death so that observations were aligned with respect to both at age at study entry and length of life. Because of considerable restrictions in sample size (down to $n = 243$ in each cohort) and by implication reductions in statistical power, we decided to report findings from analyses based on a sample that matched cohorts on age at death and education. However, results on the restricted samples revealed a substantively similar pattern of findings with no evidence of secular trends favoring the later-deceased cohort. In sum, when differences between cohorts emerged, they always suggested steeper declines for the cohort that died in the 2000s relative to the cohort that died in the 1990s.

Discussion

Our goal in the present study was to examine cohort differences in age- and mortality-related cognitive decline on episodic memory. Elaborating previous work on birth-year cohort differences from a different perspective, we compared two cohorts of participants in the AHEAD study who died earlier (1993-1999) or later (2000-2010) in historical time, taking care to match the cohorts on age at death and education, and control for a variety of potential confounds (e.g., gender, health, age at death). In both age-related and mortality-related change models, the later-deceased cohort showed a less favorable trajectory. In the age-related change model, the trajectory of the later-deceased was characterized by lower average memory performances at age 80 years as well as steeper age-related memory declines. In the mortality-related model, the later-deceased cohort was characterized by lower average performances at 2 years prior to death and steeper mortality-related declines. We discuss possible factors underlying these findings and suggest that secular trends previously reported for age-related processes favoring later-cohorts do not generalize to the last phase of life.

Differences in Cognitive Aging and Terminal Decline between Death-Year Cohorts

Empirical reports on cohort differences in old age have (almost) exclusively relied on comparisons of age-related change trajectories (using chronological age as a time metric) among birth-year cohorts. A complementary and highly informative perspective is to study differences in mortality-related trajectories (using time-to-death as a time metric) among birth-year cohorts. We take an additional step, opting for an alternative death-year-oriented perspective on cohort differences that highlights the importance of late-life conditions – in contrast to the birth-year opposed those that primarily operate earlier in life. We have argued that historical trends over the past several decades have altered the nature of living conditions that profoundly shape late-life (cognitive) development. To the best of our knowledge, our study is one of the first to compare the cognitive functioning of cohorts that died during different historical times. Overall, our findings revealed only minimal evidence for secular trends favoring the later-deceased cohort and instead indicated that mortality-related mechanisms and factors associated with approaching death (e.g., terminal illnesses) might have such a strong effect on cognitive functioning that they override secular advantages that were evident in young and middle adulthood and the early phases of old age.

In an attempt to control for possible confounds, we made use of propensity score matching procedures and covaried for a number of other between-person differences. For example, it is well established that length of life and quantitative educational attainment may contribute to secular trends in cognitive abilities (Alwin & McCammon, 2001; Hauser & Huang, 1997; Flynn, 1998; Greenfield, 1998; Rönnlund et al., 2005). We used propensity score matching to equate as closely as possible the sample of participants who died in the 1990s with those who died in the 2000s on those factors. Using the available statistical tools, we sought to ensure that the cohort differences reported in the present study are independent from cohort differences in length of life and education and to take one further step toward plausible causal inference (Foster, 2010, p. 1455). We also controlled for retest effects and covaried for differences in age at first assessment, gender, health, and frequency of observations, thereby additionally reducing the effect those substantive and methodological factors may have had on our results. For example, although the number of health conditions can be expected to differ between the death-year cohorts (Crimmins, & Saito, 2000), the influence of this particular set of factors was removed as best we could by means of statistical controls. Based on these considerations, we note that the phenomenon reported here is relatively robust and cannot be reduced to the various confounds we have controlled for.

Possible Mechanisms

A multitude of different factors may have contributed to the rather disconcerting pattern of differences in cognitive aging and terminal decline found between cohorts of older people who had died in the 1990s vs. 2000s. We highlight three possible mechanisms that could explain secular trends in cognitive functioning observed in the present study, while also noting that despite our attempt to move towards a quasi-natural experimental framework, our study is descriptive and strong inferences regarding the underlying mechanisms cannot be drawn.

A first possible mechanism revolves around manufactured survival. Specifically, notions of a compression of morbidity (Fries, 1980) and manufactured survival (Olshansky et al., 2002) offer diverging views on the consequences of secular increases in length of life on late-life functioning. According to Fries (1980), increased length of life would be accompanied by spending fewer years in a phase of functional impairments (e.g., as indicated by a later age at onset of chronic diseases). Fries' predictions (1980) were based on two premises: First, that the length of life is fixed and natural death may occur without disease; and, second, that onset of diseases can be postponed and physiological markers of aging may be modified. His predictions suggest that the average period of life in good health would increase, the proportion of morbid life would decrease, which leads to a decreasing need for medical care in later life. In contrast, Olshansky et al. (2002) argued that an extension of life due to technical and medical advances would instead be achieved at the cost of lower late-life functioning. In this study, we approached the question by focusing on the cognitive domain (as operationally defined by episodic memory). Our findings of less favorable cognitive trajectories for the later-deceased cohorts suggest that secular increases in "manufactured survival" may exacerbate age- and mortality-related cognitive declines among the oldest old and could be one of the mechanisms leading to less favorable profiles of cognitive aging and terminal decline for the later-deceased cohort. It is possible that later-dying cohorts are benefiting from medical technology that helps them to survive impairments or diseases that might have resulted in mortality among earlier-dying cohorts (Christensen et al., 2009). For example, the treatment of eventually terminal illnesses is getting more and more efficient and successful in extending the life of those affected by several months or even years (Allender, Scarborough, O'Flaherty, & Capewell, 2008; La Vecchia et al., 1993; Smolina, Wright, Rayner, & Goldacre, 2012). Such an extension of life, however, is accompanied by losses in crucial domains of functioning. For example, the treatment of chronic health conditions such as hypercholesterolemia or Parkinson's disease often involves medications with in part severe cognitive side effects (Ancelin et al., 2006; Muldoon et al., 2004).

A second possible mechanism revolves around the declining efficiency of cultural means to accommodate losses in biological resources that occurs in late life. More specifically, lifespan proponents have long argued that very old age and late life are often characterized by dysfunctionalities, vulnerabilities, and constraints (Baltes & Smith, 2003). Despite an increasing need for cultural resources, it is more and more difficult for cultural means to effectively counteract functional losses, impediments, and resource limitations. Our findings corroborate extant findings and add to the literature on functional losses in very old age by showing major late-life losses in the cognitive domain (Ebly, Parhad, Hogan, & Fung, 1994; Gerstorf, Ram et al., 2010; Singer et al., 2003; Smith, 2001; Smith & Baltes, 1997; Wu, Schimmele, & Chappell, 2012). Following this perspective, rapid increases in length of life (gains in quantity of life) over the last decades have been accompanied by losses in quality of life particularly among individuals in very old age and in the last phase of life. It is well possible that medical advances are still effective at extending life into later and later years, whereas current intervention and rehabilitation efforts may reach their limits when it comes to maintaining earlier levels of functioning across the years gained. In our analyses, we

have, by design, controlled for length of life between the cohorts, but we had no information available on those factors that may additionally be operating.

A third possible mechanism underlying secular trends at the end of life observed in the present study encompasses processes of population selection. It is widely recognized that one consequence of secular increases in length of life is that lower functioning segments of the population reach higher ages (Singer et al., 2003). As a result, later-born cohorts might be less positively selected than earlier-born cohorts, leading to lower average levels of functioning at a given age in the former. Such a scenario could have also contributed to our current findings because before the matching procedure more participants reached higher ages in the 2000s (as indicated by an older age at death for the later-deceased cohort, see Table 1). Even though we have matched the cohorts subsequently on age at death, it is possible that participants in the later-dying cohort with the same age at death belonged to a lower functioning segment of the population than participants in the earlier-dying cohort.

Limitations and Outlook

The current study examined death-year cohort differences in trajectories of age- and mortality-related cognitive decline with only one indicator of episodic memory, and thus is limited in that we only examined differences in a select aspect of cognitive abilities. Previous studies of birth-year cohort differences in age-related cognitive declines have demonstrated that effects differ by cognitive domain (e.g., verbal vs. numerical skills; Finkel et al., 2007; Gerstorf, Ram et al., 2011; Schaie, 2005; Zelinski & Kenison, 2007). For example, Schaie (2005) reported that later-born cohorts consistently showed better performances on tests of inductive reasoning and perceptual speed than earlier-born same-aged peers, whereas the reversed pattern was found for number skills (i.e., lower performance among later-born cohorts, particularly those born after 1924). It is possible that previously existing cohort differences in cognitive abilities are differentially susceptible to the effects of late-life conditions. Based on two-component theories of intelligence (Baltes et al., 2006; Cattell, 1971; Horn, 1982), for example, one would expect that earlier cohort differences are minimized for those abilities that are closely tied to biological processes (e.g., fluid measures), whereas earlier cohort differences may be preserved for more crystallized measures (e.g., Verbal Meaning) as the underlying acculturated differences may be less prone to the effects of late-life living conditions. Before reaching any general conclusions, the present examination of death-year cohorts should be corroborated and extended upon by using a multivariate battery of cognitive tests. In addition, it would be instructive to examine if and how late-life cohort differences emerge or are nullified in other domains of functioning such as well-being, physical and sensory function, and social participation. Another crucial limitation on the measures side concerns the health variables being available in our study. Although our models covaried for cohort differences in overall health, the cohorts also differed in specific health conditions such as diabetes prevalence and the effects of those differences may not have been eliminated in our matching and statistical control procedures. In a similar vein, death-year cohort differences may also have existed in the treatment of terminal illnesses such as cancer (e.g., chemotherapy, beta blockers), possible cognitive side effects of such treatments, in the length of terminal illness and the age at which cognitive pathologies and dementia set in, or in place of death (home vs. hospital vs. nursing home; see Gerstorf, Ram et al., 2011).

With the aim in mind to arrive at a comparable number of participants in each cohort, we defined the death-year cohorts using the change of the millennium as a somewhat arbitrary distinction between death-year cohorts. An alternative more mechanism-oriented approach would instead be to define cohorts based on specific large-scale events that profoundly shape the circumstances of individual development. For example, previous birth-cohort studies documented the psychosocial effects of growing up during the Great Depression

over the life-course (Elder, 1974) or to post-World War II changes in education opportunity (e.g., GI bill; Laub & Sampson, 2005). For late-life living conditions, historical events that influence public health such as the implementation of health care reforms are prime candidates for generating late-life cohort differences. Utilizing such events would make it possible to test, for example, whether improvements in public health also alleviate terminal declines in the cognitive and health domains. It also remains to be seen whether the secular trends reported in the current study will generalize to death cohorts still to come in the AHEAD study. Some of the most important innovations in health care and policy (e.g. the introduction of Medicare and Medicaid programs in 1965, policies aiming to reduce tobacco use since early 1960s) were introduced long after members of our subsamples were children and it may take a long time for public health improvements to manifest at the population level. It thus remains an open question whether such programs will improve late-life functioning in the coming decades. Coupling together birth-cohort and death-cohort analyses would provide for additional examination of how changes in care and policy manifest over different life spans. Finally, we modeled terminal decline (and age-related changes) using a growth model with linear and quadratic change components. However, this model only partially maps onto notions of compressed morbidity. Specifically, under the compressed morbidity model increases in life expectancy are accompanied by decreases in the length of morbid life (Fries, 1980). In the context of our inquiry, compression would manifest as a delayed onset of terminal decline in cognitive functioning and could be operationally defined more directly using a multiphase model of change that specifically includes a transition point that indicated the timing of the onset of terminal decline (e.g., second analysis in Gerstorf, Ram et al., 2008). If morbidity is indeed compressed, the timing of the onset should appear at a later age and at closer proximity to death for later-deceased cohorts. Unfortunately, the small number of observations per participant and the relatively large time intervals between observations (2 years) in the AHEAD data did not allow us to test this notion directly. End-of-life data spanning a longer time period and with more closely spaced assessments are needed.

Conclusions

The current study adds to previous work documenting secular trends in cognitive functioning favoring later birth-year cohorts by examining whether these same trends manifest across death-year cohorts. In contrast to earlier studies, we did not find evidence of secular trends favoring the later-deceased cohort in late-life cognitive functioning, but rather evidence of secular trends in the opposite direction. For cognitive aging, the later-dying cohort (those who died in the 2000s) exhibited lower performance levels at age 80 years and steeper age-related declines than the earlier-dying cohort (those who died in the 1990s). For terminal decline, the later-dying cohort also exhibited lower levels of performance 2 years prior to death and steeper mortality-related decline rates. We take these findings to suggest that the cohort-to-cohort improvements in cognitive function reported in younger samples do not generalize to the oldest old or to the last years of life. Further research is needed to understand the mechanisms and pathways underlying these secular trends and to prompt political and societal decisions that might improve the cognitive trajectories the fastest-growing segment of the population are likely to experience in the coming decades.

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References

- Allender S, Scarborough P, O'Flaherty M, Capewell S. Patterns of coronary heart disease mortality over the 20th century in England and Wales: possible plateaus in the rate of decline. *BMC Public Health*. 2008; 8:148–60. [PubMed: 18452595]
- Alwin, DF. History, cohort, and patterns of cognitive aging. In: Hofer, SM.; Alwin, DF., editors. *Handbook of cognitive aging: Interdisciplinary perspectives*. Sage; Thousand Oaks, CA: 2008. p. 9-38.
- Alwin DF, McCammon RJ. Aging, cohorts, and verbal ability. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*. 2001; 56:P151–P161.
- Ancelin ML, Artero S, Portet F, Dupuy AM, Touchon J, Ritchie K. Non-degenerative mild cognitive impairment in elderly people and use of anticholinergic drugs: longitudinal cohort study. *British Medical Journal*. 2006; 332:455–459. [PubMed: 16452102]
- Anstey KJ, Mack HA, von Sanden C. The relationship between cognition and mortality in patients with stroke, coronary heart disease or cancer. *European Psychologist*. 2006; 11:182–195.
- Bäckman L, MacDonald SWS. Death and cognition: Synthesis and outlook. *European Psychologist*. 2006; 11:224–235.
- Bäckman, L.; Small, BJ.; Wahlin, A.; Larsson, M. Cognitive functioning in very old age. In: Craik, FIM.; Salthouse, TA., editors. *The handbook of aging and cognition*. 2nd ed. Erlbaum; Mahwah, NJ: 2000. p. 499-558.
- Baltes, PB.; Cornelius, SW.; Nesselroade, JR. Cohort effects in developmental psychology. In: Nesselroade, JR.; Baltes, PB., editors. *Longitudinal research in the study of behavior and development*. Academic Press; New York, NY: 1979. p. 61-87.
- Baltes, PB.; Reuter-Lorenz, PA.; Rösler, F., editors. *Lifespan development and the brain: The perspective of biocultural co-constructivism*. Cambridge University Press; New York: 2006.
- Baltes PB, Smith J. New frontiers in the future of aging: From successful aging of the young old to the dilemmas of the fourth age. *Gerontology*. 2003; 49:123–135. [PubMed: 12574672]
- Blair C, Gamson DA, Thorne S, Baker DP. Rising mean IQ: Cognitive demand of mathematics education for young children, population exposure to formal schooling, and the neurobiology of the prefrontal cortex. *Intelligence*. 2005; 33:93–106.
- Bowles RP, Grimm KJ, McArdle JJ. A structural factor analysis of vocabulary knowledge and relations to age. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*. 2005; 60:P234–P241.
- Brynskikh A, Warren T, Zhu J. X, Kipnis J. Adaptive immunity affects learning behavior in mice. *Brain, Behavior and Immunity*. 2007; 22:861–869.
- Cattell, RB. *Abilities: Their Structure, Growth, and Action*. Houghton Mifflin; Boston: 1971.
- Christensen K, Doblhammer G, Rau R, Vaupel JW. Ageing populations: the challenges ahead. *The Lancet*. 2009; 374:1196–1208.
- Coffman DL. Estimating causal effects in mediation analysis using propensity scores. *Structural Equation Modeling*. 2011; 18:357–369. [PubMed: 22081755]
- Condran GA, Crimmins-Gardner E. Public health measure and mortality in U.S. cities in the late nineteenth century. *Human Ecology*. 1978; 6:27–54. [PubMed: 12310464]
- Crimmins EM, Beltrán-Sánchez H. Mortality and morbidity trends: is there compression of morbidity? *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*. 2011; 66:P75–P86.
- Crimmins EM, Saito Y. Change in the prevalence of diseases among older Americans: 1984–1994. *Demographic Research*. 2000; 3
- Ebly EM, Parhad IM, Hogan DB, Fung TS. Prevalence and types of dementia in the very old: Results from the Canadian Study of Health and Aging. *Neurology*. 1994; 44:1593–1600. [PubMed: 7936280]
- Elder, GH, Jr. *Children of the great depression*. University of Chicago Press; Chicago, IL: 1974.
- Finkel D, Reynolds CA, McArdle JJ, Pedersen NL. Cohort differences in trajectories of cognitive aging. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*. 2007; 62:P286–P294.

- Flynn JR. Massive IQ gains in 14 nations: What IQ tests really measure. *Psychological Bulletin*. 1987; 101:171–191.
- Flynn, JR. IQ gains over time: Toward finding the causes. In: Neisser, U., editor. *The rising curve: Long-term gains in IQ and related measures*. American Psychological Association; Washington DC: 1998. p. 25–66.
- Flynn JR. Solving the IQ Puzzle. *Scientific American Mind*. 2007; 18:24–31.
- Foster E. Inference and developmental psychology. *Developmental Psychology*. 2010; 46:1454–1480. [PubMed: 20677855]
- Freedman VA, Schoeni RF, Martin LG, Cornman J. Chronic conditions and the decline in late-life disability. *Demography*. 2007; 44:459–477. [PubMed: 17913006]
- Fries JF. Aging, natural death, and the compression of morbidity. *New England Journal of Medicine*. 1980; 303:130–135. [PubMed: 7383070]
- Gerstorf D, Ram N, Estabrook R, Schupp J, Wagner GG, Lindenberger U. Life satisfaction shows terminal decline in old age: Longitudinal evidence from the German Socio-Economic Panel Study (SOEP). *Developmental Psychology*. 2008; 44:1148–1159. [PubMed: 18605841]
- Gerstorf D, Ram N, Goebel J, Schupp J, Lindenberger U, Wagner GG. Where people live and die makes a difference: Individual and geographic disparities in well-being progression at the end of life. *Psychology and Aging*. 2010; 25:661–676. [PubMed: 20677887]
- Gerstorf D, Ram N, Hoppmann CA, Willis SL, Schaie KW. Cohort differences in cognitive aging and terminal decline in the Seattle Longitudinal Study. *Developmental Psychology*. 2011; 47:1026–1041. [PubMed: 21517155]
- Ghisletta P, McArdle JJ, Lindenberger U. Longitudinal cognition-survival relations in old and very old age: 13-Year data from the Berlin Aging Study. *European Psychologist*. 2006; 11:204–223.
- Gray SL, Lai KV, Larson EB. Drug induced cognition disorders in the elderly--incidence, prevention and management. *Drug Safety*. 1999; 21:101–122. [PubMed: 10456379]
- Greenfield, PM. The cultural evolution of IQ. In: Neisser, U., editor. *The rising curve: Long-term gains in IQ and related measures*. American Psychological Association; Washington DC: 1998. p. 81–124.
- Grissmer, DW.; Kirby, SN.; Berends, M.; Williamson, S. *Student achievement and the changing American family*. RAND Institute on Education and Training; Santa Monica, CA: 1994.
- Haley GE, Urbanski HF, Kohama SG, Messaoudi I, Raber J. Spatial memory performance associated with measures of immune function in elderly female rhesus macaques. *European Geriatric Medicine*. 2011; 2:117–121. [PubMed: 21603071]
- Hauser RM, Huang M-H. Verbal ability and socioeconomic success: A trend analysis. *Social Science Research*. 1997; 26:331–376.
- Horn, JL. The theory of fluid and crystallized intelligence in relation to concepts of cognitive psychology and aging in adulthood. In: Craik, FIM.; Trehub, S., editors. *Aging and cognitive processes*. Plenum; New York: 1982. p. 237–278.
- Jackson JJ, Thoemmes F, Jonkmann K, Lüdtko O, Trautwein U. Military training and personality trait development: Does the military make the man or does the man make the military? *Psychological Science*. (in press).
- La Vecchia C, Lucchini F, Negri E, Reggi V, Levi F. The impact of therapeutic improvements in reducing peptic ulcer mortality in Europe. *International Journal of Epidemiology*. 1993; 22:96–106. [PubMed: 8095492]
- Larson EB, Kukull EA, Buchner D, Reifler BV. Adverse drug reactions associated with global cognitive impairment in elderly persons. *Annals of Internal Medicine*. 1987; 107:169–173. [PubMed: 2886086]
- Laub, JH.; Sampson, RJ. Coming of age in wartime: how World War II and the Korean War changed lives. In: Schaie, KW.; Elder, GH., editors. *Historical influences on lives and aging*. Springer Publishing Company; New York: 2005. p. 208–228.
- Lindenberger U, Lövdén M, Schellenbach M, Li S-C, Krüger A. Psychological principles of successful aging technologies: A mini-review. *Gerontology*. 2008; 54:59–68. [PubMed: 18259095]

- Lindenberger U, Singer T, Baltes PB. Longitudinal selectivity in aging populations: Separating mortality-associated versus experimental components in the Berlin Aging Study (BASE). *Journals of Gerontology Series B: Psychological Sciences*. 2002; 57B:P474–P482.
- Littell, RC.; Miliken, GA.; Steoup, WW.; Wolfinger, RD. SAS for mixed models. SAS Institute; Cary, NC: 1996.
- Little, RJA.; Rubin, DB. Statistical analysis with missing data. John Wiley & Sons; New York: 1987.
- Martorell, R. Nutrition and the worldwide rise in IQ. In: Neisser, U., editor. *The rising curve. Long term gains in IQ and related measures*. American Psychological Association; Washington, DC: 1998. p. 183-206.
- McArdle JJ, Fisher GG, Kadlec KM. Latent variable analyses of age trends from the Health and Retirement Study, 1992-2004. *Psychology and Aging*. 2007; 22:525–545. [PubMed: 17874952]
- McArdle JJ, Hamagami F, Elias MF, Robbins M. Structural modeling of mixed longitudinal and cross-sectional data. *Experimental Aging Research*. 1991; 17:29–52. [PubMed: 1936099]
- Muldoon MF, Ryan CM, Sereika SM, Flory JD, Manuck SB. Randomized trial of the effects of simvastatin on cognitive functioning in hypercholesterolemic adults. *The American Journal of Medicine*. 2004; 117:823–829. [PubMed: 15589485]
- National Hospital Ambulatory Medical Care Survey (NHAMCS). [Retrieved February 17, 2012] Health Data Interactive. 1997-2008. from <http://205.207.175.93/HDI/TableViewer/tableView.aspx?ReportId=166>
- National Vital Statistics System (NVSS). [Retrieved February 17, 2012] Health Data Interactive. 1997-2008. from <http://205.207.175.93/HDI/TableViewer/tableView.aspx?ReportId=673>
- Ofstedal, MB.; Fisher, GG.; Herzog, AR. Documentation of cognitive functioning measures in the Health and Retirement Study (HRS/AHEAD Documentation Report DR-006). University of Michigan; Ann Arbor, MI: 2005.
- Olshansky SJ, Hayflick L, Carnes BA. Position statement on human aging. *Journals of Gerontology: Series A: Biological Sciences and Medical Sciences*. 2002; 57:M292–M297.
- Riley MW. Aging and cohort succession: Interpretations and misinterpretations. *Public Opinion Quarterly*. 1973; 37:35–49.
- Ron-Harel N, Schwartz M. Immune senescence and brain aging: can rejuvenation of immunity reverse memory loss? *Trends in Neurosciences*. 2009; 32:367–375. [PubMed: 19520437]
- Rönnlund M, Nyberg L, Bäckman L, Nilsson L-G. Stability, growth, and decline in adult life span development of declarative memory: Cross-sectional and longitudinal data from a population-based study. *Psychology and Aging*. 2005; 20:3–18. [PubMed: 15769210]
- Rosenbaum PR, Rubin DB. Constructing a control group using multivariate matched sampling methods that incorporate the propensity score. *The American Statistician*. 1985; 39:33–38.
- Ryder NB. The cohort as a concept in the study of social changes. *American Sociological Review*. 1965; 30:843–861. [PubMed: 5846306]
- Schaie KW. A general model for the study of developmental problems. *Psychological Bulletin*. 1965; 64:92–107. [PubMed: 14320080]
- Schaie, KW. Developmental influences on intelligence: The Seattle Longitudinal Study. Oxford University Press; New York, NY: 2005.
- Schaie KW, Willis SL, Pennak S. An historical framework for cohort differences in intelligence. *Research in Human Development*. 2005; 2:43–67. [PubMed: 16858496]
- Setoguchi S, Glynn RJ, Avorn J, Mittleman MA, Levin R, Winkelmayr WC. Improvements in long-term mortality after myocardial infarction and increased use of cardiovascular drugs after discharge: a 10-year trend analysis. *Journal of American College of Cardiology*. 2008; 51:1247–1254.
- Singer T, Lindenberger U, Baltes PB. Plasticity of memory for new learning in very old age: A story of major loss? *Psychology and Aging*. 2003; 18:306–317. [PubMed: 12825778]
- Singer T, Verhaeghen P, Ghisletta P, Lindenberger U, Baltes PB. The fate of cognition in very old age: Six-year longitudinal findings in the Berlin Aging Study (BASE). *Psychology and Aging*. 2003; 18:318–331. [PubMed: 12825779]

- Sliwinski MJ, Stawski RS, Hall CB, Katz M, Verghese J, Lipton R. Distinguishing preterminal and terminal cognitive decline. *European Psychologist*. 2006; 11:172–181.
- Smith J. Well-being and health from age 70 to 100 years: Findings from the Berlin Aging Study. *European Review*. 2001; 9:461–477.
- Smith J, Baltes PB. Profiles of psychological functioning in the old and oldest old. *Psychology and Aging*. 1997; 12:458–472. [PubMed: 9308093]
- Smolina K, Wright FL, Rayner M, Goldacre MJ. Determinants of the decline in mortality from acute myocardial infarction in England between 2002 and 2010: linked national database study. *British Medical Journal*. 2012; 344
- Soldo B, Hurd M, Rodgers W, Wallace R. Asset and Health Dynamics Among the Oldest Old: An overview of the AHEAD study. *Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*. 1997; 52:P1–P20.
- Stuart EA. Matching methods for causal inference: a review and a look forward. *Statistical Science*. 2010; 25:1–21. [PubMed: 20871802]
- Sundet JM, Borren I, Tambs K. The Flynn effect is partly caused by changing fertility patterns. *Intelligence*. 2008; 36:183–191.
- Thoemmes F, Kim ES. A systematic review of propensity score methods in the social sciences. *Multivariate Behavioral Research*. 2011; 46:90–118.
- Verhaeghen P, Borchelt M, Smith J. Relation between cardiovascular and metabolic disease and cognition in very old age: cross-sectional and longitudinal findings from the Berlin aging study. *Health Psychology*. 2003; 22:559–569. [PubMed: 14640852]
- Wagstaff LR, Mitton MW, Arvik BM, Doraiswamy PM. Statin-associated memory loss: analysis of 60 case reports and review of the literature. *Pharmacotherapy*. 2003; 23:871–880. [PubMed: 12885101]
- Wilson RS, Beck TL, Bienias JL, Bennett DA. Terminal cognitive decline: accelerated loss of cognition in the last years of life. *Psychosomatic Medicine*. 2007; 69:131–137. 2007. [PubMed: 17327212]
- Wilson RS, Beckett LA, Bienias JL, Evans DA, Bennett DA. Terminal decline in cognitive function. *Neurology*. 2003; 60:1782–1787. [PubMed: 12796531]
- Wilson RS, Segawa E, Buchman AS, Boyle PA, Hise LP, Bennett DA. Terminal decline in motor function. *Psychology and Aging*. 2012
- Wilson-Genderson M, Pruchno RA, Cartwright FP. Effects of caregiver burden and satisfaction on affect of older end-stage renal disease patients and their spouses. *Psychology and Aging*. 2009; 24:955–967. [PubMed: 20025409]
- Wu Z, Schimmele CM, Chappell NL. Aging and late-life depression. *Journal of Aging and Health*. 2012; 24:3–28. [PubMed: 21956098]
- Zelinski EM, Kennison RF. Not your parents' scores: Cohort reduces psychometric aging effects. *Psychology and Aging*. 2007; 22:546–557. [PubMed: 17874953]
- Ziv Y, Ron N, Butovsky O, Landa G, Sudai E, Greenberg N, Cohen H, Kipnis J, Schwartz M. Immune cells contribute to the maintenance of neurogenesis and spatial learning abilities in adulthood. *Nature Neuroscience*. 2006; 9:268–275.

Figure 1.

Frequency of observations in the Health Dynamics among the Oldest Old (AHEAD) Study for the age model (Panel A) and the time-to-death model (Panel B), separately for the two death-year cohorts. The earlier-deceased cohort encompassed 1,408 participants who contributed 2,514 data points. The later-deceased cohort encompassed 1,408 participants who contributed 5,235 data points. To control for differences between cohorts, we added the frequency of observations per participant as a covariate into our models.

Figure 2.

Illustrating differences between death-year cohorts in cognitive aging of episodic memory from age 65 to age 100 (Panel A) and in terminal decline across the last 7 years of life (Panel B). Death-year cohorts were matched on age at death and education and models additionally residualized for differences in gender, number of health conditions, retest, and frequency of observations. Results revealed no evidence for secular trends favoring the later-deceased cohort. Panel A: To the contrary, AHEAD participants who died in the 2000s (grey lines) showed a less favorable trajectory of age-related memory decline than those who died in the 1990s (black lines). Panel B: Over time to death, those dying in the 2000s (grey lines) experienced even steeper terminal declines than those dying in the 1990s (black lines).

Table 1

Descriptive Statistics for Socio-demographic Measures, Separately by Death-Year Cohort.

	Death-year cohort			
	Earlier-deceased (1993–1999)		Later-deceased (2000–2010)	
	Entire sample	Matched sample	Entire sample	Matched sample
<i>N</i>	1,775	1,408	2,541	1,408
Propensity scores ^a	0.51 (0.19) _a [0.04 – 0.95]	0.57 (0.15) _b [0.09 – 0.94]	0.65 (0.15) _c [0.04 – 0.97]	0.58 (0.15) _b [0.13 – 0.95]
Years of education	10.64 _a (3.65)	10.83 _a (3.65)	11.26 _b (3.51)	10.68 _a (3.67)
Age at individual T1	79.21 _a (6.62) [54 - 103]	81.19 _b (5.71) [65 - 99]	79.46 _a (5.82) [50 - 105]	77.30 _c (5.36) [65 - 98]
% women	56 _a	55 _a	62 _b	62 _b
Age at death	82.76 _a (6.73) [58 - 107]	84.88 _b (5.66) [67 - 103]	87.53 _c (5.84) [58 - 112]	85.26 _b (5.37) [67 - 105]
Number of health conditions at the last measurement occasion	2.29 _a (1.42)	2.25 _a (1.41)	2.69 _b (1.42)	2.71 _b (1.43)

Note. *SD* in parentheses. Ranges in square brackets. Subscripts that differ between columns are statistically significant at $p < 0.05$.

^aPropensity scores indicate the probability of membership in the later-deceased cohort based on age at death and education.

Table 2
Descriptive Statistics for Episodic Memory Performance over Age and Time-to-Death, Separately for Death-Year Cohort.

	Chronological age						Time-to-death						
	Earlier-deceased 1993-1999			Later-deceased 2000-2010			Earlier-deceased 1993-1999			Later-deceased 2000-2010			
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	
65	2	60.19	11.09	3	61.94	6.58	15	0	-	-	8	47.45	6.47
66	0	-	-	14	55.15	9.20	14	0	-	-	5	44.50	4.13
67	4	58.89	15.62	19	56.20	9.76	13	0	-	-	18	53.37	11.39
68	3	44.50	14.56	19	54.83	11.01	12	0	-	-	19	51.52	9.63
69	3	60.19	20.76	30	57.06	9.55	11	0	-	-	55	49.21	9.76
70	12	51.04	11.61	59	56.60	7.04	10	0	-	-	108	50.92	9.19
71	18	54.67	11.48	84	54.03	10.87	9	0	-	-	266	52.07	9.49
72	37	53.34	7.25	150	53.19	8.96	8	0	-	-	527	52.60	9.43
73	59	52.48	9.74	171	54.26	9.89	7	0	-	-	568	51.65	9.48
74	86	51.98	9.27	225	54.29	9.39	6	277	49.49	9.63	581	52.15	9.73
75	107	51.20	9.55	250	53.57	9.13	5	211	49.45	9.53	529	50.99	9.24
76	132	50.78	9.69	276	53.02	10.22	4	530	47.56	9.22	562	49.26	9.66
77	159	51.41	9.77	337	52.73	9.68	3	462	46.86	9.20	604	48.43	9.10
78	144	49.46	8.69	322	51.94	9.62	2	432	46.91	9.63	658	48.14	9.56
79	151	48.66	8.78	377	50.50	9.43	1	494	46.38	8.79	504	46.95	9.02
80	161	48.39	9.38	357	50.25	8.66	0	99	49.23	9.44	316	46.41	8.58
81	163	47.47	8.42	377	49.44	9.81							
82	159	47.22	8.61	304	48.82	8.85							
83	157	46.19	9.20	302	47.71	9.03							
84	139	45.50	8.37	285	47.76	8.15							
85	157	45.55	8.33	226	47.26	8.83							
86	122	45.15	8.04	234	47.29	8.44							
87	116	44.84	8.24	166	46.33	8.12							
88	89	43.06	7.74	155	45.26	7.85							
89	79	44.30	9.30	124	45.77	8.14							
90	68	43.43	8.79	94	45.42	7.58							

Chronological age						Time-to-death					
Earlier-deceased 1993-1999			Later-deceased 2000-2010			Earlier-deceased 1993-1999			Later-deceased 2000-2010		
<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>
91	43	44.81	9.62	76	43.71	7.98					
92	41	42.91	7.89	51	44.25	7.95					
93	28	43.76	8.05	49	44.61	8.22					
94	22	43.79	8.16	29	43.69	7.79					
95	19	43.40	8.00	32	42.95	7.51					
96	15	40.49	7.19	13	43.70	8.76					
97	7	40.77	8.11	11	42.60	8.36					
98	7	43.01	9.65	6	37.09	5.59					
99	4	41.23	6.88	6	44.94	4.50					
100	1	44.50	-	2	45.81	1.85					

Note. Earlier-deceased (1993 – 1999; $n = 1,408$) versus later-deceased (2000 – 2010; $n = 1,408$) death-year cohorts. T-scores standardized to 1993 memory scores of the subsample in the present study ($M = 50$, $SD = 10$).

Table 3

Growth Curve Models of Cognitive Aging and Terminal Decline: The Role of Cohort and the Covariates.

Parameter	Cognitive Aging		Terminal Decline	
	Estimate	SE	Estimate	SE
Fixed effects				
Intercept ^a , γ_{00}	49.685 *	0.157	47.217 *	0.160
Linear slope, γ_{10}	-0.613 *	0.024	-1.195 *	0.071
Quadratic slope, γ_{30}	0.002	0.002	-0.059 *	0.015
Retest, γ_{20}	0.132	0.237	0.889 *	0.306
Death-year cohort, γ_{01}	-1.022 *	0.467	-0.848	0.514
Gender, γ_{02}	1.817 *	0.280	1.311 *	0.291
Education (in years), γ_{03}	0.705 *	0.039	0.612 *	0.040
Number of health conditions, γ_{04}	0.123	0.097	0.105	0.101
Frequency of observations, γ_{05}	1.743 *	0.216	2.641 *	0.290
Age at T1, γ_{06}	–	–	0.903 *	0.119
Age at death, γ_{07}	–	–	-1.369 *	0.120
Death-year cohort $\times$ Linear Slope, γ_{11}	-0.131 ^b	0.070	-0.437 *	0.145
Gender $\times$ Linear Slope, γ_{12}	-0.109 *	0.041	-0.174 *	0.066
Education $\times$ Linear Slope, γ_{13}	-0.017 *	0.005	-0.034 *	0.009
Number of health conditions $\times$ Linear Slope, γ_{14}	0.014	0.014	-0.013	0.023
Frequency of observations $\times$ Linear Slope, γ_{15}	-0.062	0.032	0.099	0.076
Age at T1 $\times$ Linear Slope, γ_{16}	–	–	0.033	0.026
Age at death $\times$ Linear Slope, γ_{17}	–	–	-0.052	0.027
Random effects				
Variance intercept, σ^2_{u0}	32.308 *	1.517	32.838 *	1.610
Variance linear slope, σ^2_{u1}	0.054 *	0.028	0.294 *	0.086
Variance retest, σ^2_{u2}	6.603 *	3.173	7.128 *	3.775
Covariance intercept, slope, σ_{u0u1}	-0.642 *	0.141	0.514 *	0.293
Covariance intercept, retest, σ_{u0u2}	3.261 *	1.418	1.987 *	1.402
Covariance slope, retest, σ_{u1u2}	-0.050	0.218	-0.058	0.443
Residual variance, σ^2_e	39.443 *	0.984	36.295 *	1.039
Proportional reduction in unexplained variance accounted for by <i>time</i> (age or distance-to-death) and covariates	16.7 %		23.4 %	
Correlation between observed and predicted values	0.841		0.866	
– 2LL	53,822		53,627	

Note. Memory score standardized to a T metric ($M = 50$, $SD = 10$) based on cross-sectional data at study entry. Unstandardized estimates and standard errors are presented. Boldface type highlights cohort differences in cognitive aging. All covariates were effect coded/centered. Parameter estimates indicate the average trajectory and the extent of differences of a particular covariate. Positive parameters indicate differences favoring women, the later-deceased cohort, participants with higher age at death and at T1, and participants with higher education. Cohort = earlier-deceased

(1993–1999; $n = 1,408$) versus later-deceased (2000–2010; $n = 1,408$). $2LL = 2 \log$ likelihood, a relative model fit statistic. The correlation between observed and predicted values is an indicator of model fit indicating the congruence between observed values and model predictions.

^aIntercept is centered at age 80 years for the cognitive aging model and at 2 years before death for the terminal decline model.

^b
 $p = 0.06$

^{*}
 $p < .05$.