ELSEVIER

Alzheimer's & Dementia 4 (2008) 134–144

Alzheimer's & Dementia

Trends in the prevalence and mortality of cognitive impairment in the United States: Is there evidence of a compression of cognitive morbidity?

Kenneth M. Langa[a,b,c,*], Eric B. Larson[d], Jason H. Karlawish[e], David M. Cutler[f], Mohammed U. Kabeto[a], Scott Y. Kim[g], Allison B. Rosen[a,b]

[a]*Division of General Medicine, Department of Medicine, University of Michigan, Ann Arbor, MI, USA*
[b]*Veterans Affairs Center for Practice Management and Outcomes Research, Ann Arbor, MI, USA*
[c]*Institute for Social Research, University of Michigan, Ann Arbor, MI, USA*
[d]*Center for Health Studies, Group Health Cooperative of Puget Sound, Seattle, WA, USA*
[e]*Department of Medicine, Division of Geriatric Medicine, University of Pennsylvania, Philadelphia, PA, USA*
[f]*Department of Economics, Harvard University, and the National Bureau of Economic Research, Cambridge, MA, USA*
[g]*Department of Psychiatry, Bioethics Program, and Center for Behavioral and Decision Sciences in Medicine, University of Michigan, Ann Arbor, MI, USA*

Abstract

Background: Recent medical, demographic, and social trends might have had an important impact on the cognitive health of older adults. To assess the impact of these multiple trends, we compared the prevalence and 2-year mortality of cognitive impairment (CI) consistent with dementia in the United States in 1993 to 1995 and 2002 to 2004.

Methods: We used data from the Health and Retirement Study (HRS), a nationally representative population-based longitudinal survey of U.S. adults. Individuals aged 70 years or older from the 1993 (N = 7,406) and 2002 (N = 7,104) waves of the HRS were included. CI was determined by using a 35-point cognitive scale for self-respondents and assessments of memory and judgment for respondents represented by a proxy. Mortality was ascertained with HRS data verified by the National Death Index.

Results: In 1993, 12.2% of those aged 70 or older had CI compared with 8.7% in 2002 ($P < .001$). CI was associated with a significantly higher risk of 2-year mortality in both years. The risk of death for those with moderate/severe CI was greater in 2002 compared with 1993 (unadjusted hazard ratio, 4.12 in 2002 vs 3.36 in 1993; $P = .08$; age- and sex-adjusted hazard ratio, 3.11 in 2002 vs 2.53 in 1993; $P = .09$). Education was protective against CI, but among those with CI, more education was associated with higher 2-year mortality.

Conclusions: These findings support the hypothesis of a compression of cognitive morbidity between 1993 and 2004, with fewer older Americans reaching a threshold of significant CI and a more rapid decline to death among those who did. Societal investment in building and maintaining cognitive reserve through formal education in childhood and continued cognitive stimulation during work and leisure in adulthood might help limit the burden of dementia among the growing number of older adults worldwide.

Keywords: Dementia; Epidemiology; Cardiovascular disease; Education

1. Introduction

Dementia, a decline in memory and other cognitive functions that leads to a loss of independent function [1], is a common geriatric syndrome that exacts considerable impact

*Corresponding author. Tel.: 734-615-8341; Fax: 734-936-8944.
E-mail address: klanga@umich.edu

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doi:10.1016/j.jalz.2008.01.001

on individuals, families, and government programs. There has been progress during the last 25 years in identifying medical, lifestyle, and demographic factors that might affect either the risk of developing dementia or its rate of progression. In addition, social and cultural events have raised awareness of cognitive health and that dementia is a terminal illness. What is not known is the collective impact of these trends on America's overall cognitive health.

Because the number of older Americans, especially the oldest-old (those aged 85 years or older), has grown in recent decades, several key developments likely have had an important impact on "brain health." New medications and other therapies for cardiovascular and cerebrovascular disease introduced since the early 1990s (eg, wider use of antihypertensive and 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor [statin] medications) might have contributed to a reduction in myocardial infarction, stroke, and vascular dementia during the past 15 years [2,3]. However, an increased prevalence of diabetes during this time period (from about 12.8% of older adults in 1992 to 15.1% in 2002 [4]) might have led to an accompanying increase in the prevalence of dementia, given the growing evidence of an association between diabetes and cognitive decline [5,6].

In addition to these trends in control of cardiovascular and cerebrovascular risk factors, there have been major changes in the management of persons with dementia during the past 15 years. Cholinesterase inhibitors (ChIs) were introduced during the mid-1990s for the treatment of Alzheimer's disease. Short-term studies reported improvements in cognitive test scores among those with dementia taking ChIs compared with those taking placebo [7–10], but whether there is a delay to important clinical outcomes, such as severe disability, nursing home placement, or death, is still debated [11–16].

Rising levels of education among older adults during the past 15 years might have influenced the prevalence and outcomes of dementia. The proportion of adults age 65 or older with a high school diploma increased from 53% in 1990 to 72% in 2003, whereas the proportion with a college degree increased from 11% to 17% during this same time period [17]. More years of formal education is associated with a reduced risk of dementia [18–20], likely through multiple causal pathways including a direct effect on brain development and function (ie, the building of "cognitive reserve" [21]), better health behaviors, and the general health advantages of having more wealth and social opportunities [3,22–24]. However, higher levels of education are also associated with a more rapid decline in cognitive function after the onset of dementia [25,26], which might translate to increased mortality [22,27,28] among those with dementia.

Wealth of older adults has also increased significantly, with median household net worth for those age 65 or older increasing (in constant 2001 dollars) from $108,000 in 1989 to $180,000 in 2001 [29]. Similar to education, greater wealth is associated with lower levels of disability throughout the life course, likely through multiple causal pathways [30,31], and might have contributed to declining levels of dementia during the past 15 years.

To investigate the impact of these multiple trends on dementia prevalence and mortality, we used a large nationally representative study of older Americans to identify individuals with cognitive impairment (CI) consistent with dementia in 1993 and 2002 and then followed each cohort to determine 2-year mortality for those with and without impairment.

2. Methods

2.1. Data and study population

We used data from the 1993 and 2002 waves of the Health and Retirement Study (HRS) [32]. In selecting our study samples from the HRS, our main analytic goal was to identify two similar nationally representative cohorts of older individuals (age 70 or older) in 1993 and 2002, characterize their cognitive function by using the same cognitive tests in each year, and then follow each cohort for 2 years to determine mortality for individuals with (1) normal cognitive function and (2) CI consistent with dementia. The CI category was further subdivided into mild CI and moderate/severe CI (described more fully below).

The HRS is a biennial, longitudinal, nationally representative survey of U.S. adults, but new cohorts have been entered into the study at different times [33]. As a result, 3,419 individuals in our analysis were included in both the 1993 and 2002 cohorts, whereas 4,024 were included only in 1993 and 4,205 only in 2002. The standard errors for all parameter estimates and the P values for statistical tests comparing results across the 1993 and 2002 cohorts were adjusted for this overlap in samples [34].

The 1993 wave of the HRS was limited to individuals who were living in the community (ie, not residing in nursing homes). Of the 7,443 HRS respondents who were 70 or older at the time of their 1993 interview, 793 (10.7%) had died by the 1995 interview, and 24 (0.3%) had unknown status. The 2002 wave of the HRS included both community-dwelling and institutionalized adults. Of the 7,624 respondents who were 70 or older at the time of the 2002 interview, 473 (6.2%) were in a nursing home at the time of interview. These respondents were excluded from the analysis to ensure comparable community-dwelling cohorts in both years. Of the remaining 7,151 respondents, 722 (10.1%) died by the 2004 interview, and 33 (0.5%) had unknown status. We excluded the small number of respondents with unknown vital status at the 2-year follow-up. The overall response rate for the HRS survey was 80.4% in 1993 and 86.9% in 2002 [33].

The Social Sciences Institutional Review Board at the University of Michigan approved the HRS, and the Medical

School Institutional Review Board approved the use of HRS data for the current study.

2.2. Measurement of cognitive function and cognitive category definitions

The HRS assesses cognitive function with a 35-point scale that includes an immediate and delayed 10-noun free recall test to measure memory; a serial seven subtraction test to measure working memory; a counting backwards test to measure speed of mental processing; an object naming test to measure knowledge and language; and recall of the date, the president, and the vice-president to measure orientation [35,36]. For self-respondents, the presence and severity of CI were defined by using this 35-point cognitive scale. A score of 11 or above was defined as normal cognitive function, and a score of 10 or below was defined as CI. The CI category was further subdivided into mild CI for those with a score of 8 to 10 and moderate/severe CI for those scoring from 0 to 7.

The 35-point cognitive scale was not administered to respondents represented by a proxy (about 10% of the HRS sample in each cohort), but each proxy was asked: "How would you rate [the respondent's] memory at the present time?", and "How would you rate [the respondent] in making judgments and decisions?" If a respondent's memory was assessed as excellent, very good, or good, they were considered to have normal cognitive function, whereas those with fair or poor memory were considered to have CI. Proxy assessments of judgment were used to further stratify those with CI into mild CI (judgment assessed as excellent, very good, or good) and moderate/severe CI (judgment assessment as fair or poor).

Our definitions and cut points for these categories were based on our prior studies with the HRS data [37], as well as the methods used for the Aging, Demographics, and Memory Study (ADAMS), a supplemental study of dementia in the HRS [38]. The validity of these categories is supported by the clear trends in functional limitations with which they are associated. We assessed the mean number of limitations in instrumental activities of daily living (IADLs) (preparing meals, grocery shopping, making phone calls, taking medications, managing money) and found that those in the normal, mild CI, and moderate/severe CI categories had an average of 0.4, 1.1, and 2.5 IADL limitations, respectively ($P < .001$). Similar trends were found for both self-respondents and proxy-respondents when we analyzed these groups separately. More details on the HRS self-report and proxy cognitive measures are available at the HRS website [39].

2.3. Independent variables used as covariates

The following sociodemographic measures were included in the analyses as independent variables: age (70 to 79, 80 to 89, ≥90 years), race (white, black, other), gender, education (<12 years, 12 years, 13 to 15 years, and >16 years), potential caregiver network (a spouse and/or living children), and net worth (tertiles, 1993 dollars). The self-reported chronic medical conditions included were stroke, diabetes, heart disease, hypertension, lung disease, cancer, psychiatric problems, smoking status, and obesity (self-reported height and weight resulting in a body mass index [BMI] >30 kg/m^2).

2.4. Determining mortality and date of death

Vital status and date of death were ascertained by using the HRS tracker and exit files and were verified by using the National Death Index [33]. For the 1993 cohort, the number of days between the 1993 interview and the date of death was calculated for all respondents who died before the 1995 interview. For those who were alive at the time of the 1995 interview, we calculated the number of days between the 1993 and 1995 interview dates. The same method was used for the 2002 cohort.

2.5. Analytic framework

2.5.1. Trend in the prevalence and predictors of CI 1993 and 2002

We pooled data from 1993 and 2002 and estimated logistic regression models with a dichotomous dependent variable indicating whether an individual had CI (mild, moderate, or severe). A linear trend variable that took the value of 0 in 1993 and 1 in 2002 was included in the logistic regression models. An odds ratio (OR) less than 1 for this trend variable would indicate a decrease in the prevalence of CI between 1993 and 2002 [40]. We estimated seven separate logistic regression models with different sets of independent variables (eg, demographic variables, education, cardiovascular risks, and other chronic conditions) to determine which variables were most significantly associated with change in the prevalence of CI between 1993 and 2002.

2.5.2. Two-year mortality in 1993 and 2002

To examine changes in mortality from 1993 to 2002, we estimated separate Cox proportional hazards models for 1993 and 2002 to determine the unadjusted and adjusted hazard ratios (HRs) of baseline cognitive category for subsequent 2-year mortality. For each year, we estimated a model containing only the cognitive categories as an independent variable (unadjusted HR of cognitive category for 2-year mortality) and a second model that added age and gender (age- and gender-adjusted HR). We then estimated a fully adjusted model that included all covariates, as well as interactions for cognitive category and education level. Statistical significance of changes in the HRs between years was assessed by using bootstrapping methods to estimate standard errors.

We repeated all of the prevalence and mortality regression analyses after inclusion of an indicator variable for self-respondent or proxy-respondent. This did not signifi-

cantly change the coefficient estimates or across-year trends, so we reported the results from the original analyses that did not include the self-respondent or proxy-respondent indicator variable.

Statistical analyses were performed with STATA (Release 8.0; Stata Corp, College Station, TX) and SUDAAN (Release 9.0; Research Triangle Institute, Research Triangle Park, NC). All results were adjusted for the complex sampling design of the HRS survey.

3. Results

3.1. Characteristics of the study population

Table 1 shows the characteristics of the 1993 and 2002 study cohorts. Compared with the 1993 cohort, the 2002 cohort was slightly older (mean, 77.8 vs 77.5 years), had significantly more years of education, and had higher net worth (in constant 1993 dollars). Individuals with less than a high school diploma (12 years of education) comprised 42% of the sample in 1993 but only 31% in 2002. On average, individuals in the 2002 cohort had almost 1 more year of education compared with those in the 1993 cohort (11.8 vs 11.0 years). The 1993 and 2002 cohorts had the same likelihood of being married, but those in the 2002 cohort were more likely to have a living child.

Those in the 2002 cohort had significantly fewer IADL limitations but higher rates of cardiovascular risk factors and cardiovascular disease, including diabetes, hypertension, obesity, and heart disease. The proportion of the HRS sample represented by a proxy respondent was the same (10%) in both the 1993 and 2002 cohorts.

3.2. Trend in prevalence and adjusted odds of CI

Table 2 shows the unadjusted proportion of individuals in each cognitive function category in 1993 and 2002 and displays a significant decrease in the proportion of individuals who had CI consistent with dementia between 1993 and 2002 (12.2% had CI in 1993 compared with 8.7% in 2002; $P < .001$).

Table 3 reports the results of seven different logistic regression models with the presence of CI (mild, moderate, or severe) as the outcome variable, with pooled 1993 and 2002 data. The trend variable in the first row of the table represents the odds of CI in 2002 compared with 1993. Model 1 shows the statistically significant decline (OR, 0.68) in unadjusted CI prevalence already noted in Table 2. When adjusting for age and sex differences between the two cohorts (Model 2), the trend toward decreased CI prevalence was slightly larger (the trend OR drops from 0.68 to 0.65), as a result of the older age of the 2002 cohort and the strong association of older age with increased odds of CI. Higher levels of education (Model 3) and net worth (Model 4) were associated with significantly lower odds of CI, and the higher levels of education and net worth in the 2002 cohort accounted for about 43% (15 percentage points) of the decrease in CI prevalence between the years (ie, the trend OR increased from 0.65 to 0.80, with adjustment for education and net worth). In the fully adjusted model (Model 7), stroke was associated with increased odds of CI and hypertension with lower odds of CI, but the other cardiovascular risks did not show a significant association with CI. After adjustment for education and net worth, additional independent variables (Models 5 through 7) did not explain more of the decrease in CI prevalence between 1993 and 2002.

3.3. Two-year mortality: 1993 to 1995 and 2002 to 2004

Table 4 shows the unadjusted 2-year mortality for individuals in each cognitive function category in 1993 and 2002. In both years, cognitive function was clearly related to risk of death, with significantly higher mortality among those with worse cognitive function.

Table 5 shows the unadjusted and adjusted HRs for 2-year mortality (with normal cognitive function as the reference group). CI was associated with increased 2-year mortality in both 1993 and 2002. Mortality among those with moderate/severe CI in 2002 was higher than in 1993 (unadjusted HR, 4.12 vs 3.36; $P = .08$; age-and sex-adjusted HR, 3.11 vs 2.53; $P = .09$). To determine whether more years of education were associated with an increased risk of death among those with CI, we tested for a significant interaction between cognitive function category and education in the fully adjusted model. More years of education were generally associated with an increased risk of death among those with CI (ORs >1 for the CI $\times$ education interaction). The magnitude of this interaction was larger in the 2002 cohort compared with the 1993 cohort, suggesting an increasing risk of mortality for those with CI and more years of education in 2002 compared with 1993.

4. Discussion

In a large nationally representative survey of older Americans we found that between 1993 and 2002, the prevalence of CI consistent with dementia decreased from 12.2% to 8.7%, representing an absolute decrease of 3.5 percentage points and a relative decrease of nearly 30%. In addition, we found an increased risk of death among those with moderate or severe CI, and this increased mortality was most evident among those with CI who had higher levels of education.

The decline in the prevalence of CI suggests that, overall, the combined impact of recent trends in medical, lifestyle, demographic, and social factors has been positive for the cognitive health of older Americans. Although the prevalence of some cardiovascular risks that are also associated with a higher risk of dementia [15] increased significantly, other factors showed trends that favored a reduced prevalence of CI. Most importantly, individuals who were 70 or

Table 1
Characteristics of the 1993 and 2002 study cohorts

Variable	1993 (N = 7,406)	2002 (N = 7,104)	*P* value*
Age (y)			.08
70 to 79	4,860 (67.0)	4,494 (64.6)	
80 to 89	2,248 (29.1)	2,258 (31.2)	
≥90	298 (3.9)	352 (4.3)	
Mean ± SE	77.5 ± 0.1	77.8 ± 0.9	.02
Race			.004
White	6,237 (90.2)	6,096 (89.4)	
Black	1,014 (8.0)	793 (7.7)	
Other	154 (1.8)	207 (2.9)	
Gender			.1
Male	2,886 (40.0)	2,990 (40.8)	
Female	4,520 (60.0)	4,114 (59.2)	
Education (y)			<.001
<12	3,337 (42.0)	2,350 (31.2)	
12	2,151 (30.6)	2,370 (34.1)	
13 to 15	1,055 (14.8)	1,194 (17.2)	
≥16	863 (12.7)	1,190 (17.4)	
Mean ± SE	11.0 ± .09	11.8 ± .10	<.001
Net worth (1993 $)			<.001
≤43,500	2,668 (32.5)	1,968 (26.8)	
43,500 to 167,100	2,667 (36.8)	2,202 (30.7)	
>167,100	2,071 (30.6)	2,934 (42.5)	
Mean ± SE	179,000 ± 8,400	284,000 ± 10,900	<.001
Potential caregiver network			
Spouse present	3,625 (50.4)	3,745 (50.1)	.9
Living child	6,433 (87.1)	6,484 (90.4)	<.001
No. of ADLs† impaired			.7
0	5,160 (70.8)	4,989 (70.9)	
1 to 3	1,751 (23.0)	1,676 (23.3)	
4 to 6	495 (6.2)	439 (5.8)	
Mean ± SE	0.67 ± 0.02	0.66 ± 0.02	.7
No. of IADLs‡ impaired			<.001
0	5,112 (70.3)	5,631 (80.0)	
1 to 3	1,955 (25.5)	1,141 (15.9)	
4 to 5	339 (4.2)	329 (4.2)	
Mean ± SE	0.56 ± 0.02	0.44 ± 0.02	<.001
Chronic conditions			
Stroke	785 (10.5)	774 (10.7)	.7
Diabetes	987 (12.4)	1,310 (17.8)	<.001
Heart disease	2,339 (32.0)	2,448 (34.1)	.005
Hypertension	3,694 (49.1)	4,228 (59.6)	<.001
Lung disease	842 (11.9)	768 (11.0)	.1
Cancer	1,014 (14.0)	1,287 (18.4)	<.001
Psychiatric problem	805 (10.8)	942 (13.2)	<.001
BMI (kg/m^2)			<.001
<18.5	295 (4.0)	243 (3.4)	
18.5 to 24.9	3,320 (46.6)	2,858 (41.1)	
25.0 to 29.9	2,637 (35.7)	2,653 (37.7)	
≥30.0	1,050 (13.7)	1,261 (17.8)	
Smoking status			<.001
Never	3,508 (47.8)	3,128 (44.6)	
Former	3,113 (42.5)	3,381 (47.6)	
Current	729 (9.7)	541 (7.7)	
Respondent type			.7
Self	6,621 (89.7)	6,295 (89.9)	
Proxy	785 (10.3)	809 (10.1)	

NOTE. Values in parentheses are weighted percentages derived by using the HRS respondent population weights to adjust for the complex sampling design of the HRS survey.

Abbreviation: SE, standard error.

* *P* value for χ^2 or *t* test for a significant difference in proportion or mean between years.

† Includes eating, transferring, toileting, dressing, bathing, and walking across a room.

‡ Includes preparing meals, grocery shopping, making phone calls, taking medications, managing money.

Table 2
Cognitive function at baseline, 1993 and 2002 cohorts

Cognitive function	1993 (N = 7,393)	2002* (N = 7,083)
Normal	6,354 (87.8)	6,413 (91.3)
Mild CI	440 (5.2)	257 (3.5)
Moderate/severe CI	599 (7.0)	413 (5.2)

NOTE. Values in parentheses are weighted percentages derived by using the HRS respondent population weights to adjust for the complex sampling design of the HRS survey. Cognitive function data were missing for 13 respondents in 1993 and 21 respondents in 2002. These respondents were excluded from this analysis.

* $P < .001$ for differences in weighted percentages across study years.

older in 2002 had significantly higher levels of education, on average, than those who were 70 or older in 1993. Our trend analyses suggested that increasing levels of education and net worth among older Americans explained about 40% of the observed relative decrease in CI prevalence between 1993 and 2002.

Higher levels of education are likely associated with greater cognitive reserve, in that brains of the more educated are able to sustain greater damage (eg, Alzheimer's disease pathology or ischemia) before reaching the threshold of clinically significant CI [21,26]. However, at the time this threshold is finally crossed, brain pathology is more advanced in those with more education, resulting in a more rapid cognitive decline [22,26] and greater risk of mortality [22,27]. Our findings support the cognitive reserve hypothesis in that we found a significant protective effect of education on CI risk in both the 1993 and 2002 cohorts and increased risk of 2-year mortality among those with CI who had higher levels of education in both the 1993 and 2002 cohorts. Our findings of a declining prevalence of CI between 1993 and 2002 and the strong association of education with decreased risk for CI are consistent with similar trends found between 1982 and 1999 in a recent study with data from the National Long Term Care Survey [3], and our findings extend those of Freedman et al [23,41], who also found a decline between 1993 and 1998 in severe CI with HRS data.

Potential mechanisms leading from more education to better cognitive function and reserve include a direct positive effect of schooling on brain development [3,24,26], greater mental stimulation throughout the life course as a result of more cognitively demanding occupations [42,43] and leisure time activities [20,24,44], and more "brain healthy" lifestyles such as better control of cardiovascular and cerebrovascular risk factors, as well as better access to health care interventions that might help preserve cognitive function [30,31].

Our finding that the increasing prevalence of cardiovascular risks was not accompanied by an increasing prevalence of CI suggested that these risks were treated more successfully in 2002 compared with 1993. For instance, a recent analysis of Medicare Current Beneficiary Survey (MCBS) data showed that the use of statin medications to treat high cholesterol increased from 4% to 22% of older individuals with heart disease between 1993 and 2002, whereas use of any antihypertensive medication increased from 46% to 62% [45]. This more intensive treatment was accompanied by significantly better blood pressure control and improved cholesterol profiles among those 65 to 84 years old, as measured in the National Health and Nutrition Examination Survey (NHANES). In sum, both more intensive and successful treatment of cardiovascular risks in 2002 compared with 1993 might have had a "spill-over" benefit for population cognitive health. The association of self-reported hypertension with lower risk of dementia in our study is consistent with a possible protective effect of antihypertensive medications [46,47]; however, we were unable to test this hypothesis directly.

There have been significant changes in the treatment of Alzheimer's disease, the most common cause for dementia, during the time period of our study. Since 1993, ChI medications have been approved for treatment of mild to moderate Alzheimer's disease. The use of these medications has increased rapidly since their introduction; about 25% of patients with Alzheimer's disease were using a ChI during the late 1990s in one population-based study [48], and prescriptions have increased steadily since then [49]. Because these medications are used mainly only after diagnosis of dementia, and because their impact on cognitive function is modest, it is highly unlikely that they are an important explanation for the decreased prevalence of CI between 1993 and 2002 that we found in our study.

One prior study with HRS data to study trends in cognitive function did not show the same results as our study. Rodgers et al [50] found no significant decline in the proportion of those with CI, after adjusting for a number of survey design issues, including whether respondents had taken the HRS cognitive test at a prior wave. The exclusion from that study of proxy respondents, a significant proportion of whom have CI, might be one source for the difference in findings. However, Freedman et al [23] found a significant decline in severe CI in the community-dwelling sample (both self-respondents and proxy respondents) between 1993 and 1998, and these findings were robust to various assumptions regarding loss to follow-up, trends in the size and composition of the nursing home population, and the handling of item nonresponse on the HRS cognitive scale [41]. Our study adds to this prior work by tracking important changes in the mortality associated with CI during this time period and by using more recent HRS data.

To further examine the potential impact of differential loss to follow-up on our results, we determined the baseline cognitive status of those who were lost to follow-up between the 1993 and 2002 waves of the study (ie, those who were not known to be dead and who did not provide either a self-interview or proxy interview in 2002). Of the 7,406

Table 3
ORs for presence of CI in 1993 and 2002 (N = 14,476)

Variable	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Trend (2002 vs 1993)	0.68 (0.60–0.77)	0.65 (0.58–0.73)	0.76 (0.67–0.86)	0.80 (0.70–0.91)	0.77 (0.67–0.87)	0.74 (0.64–0.86)	0.72 (0.62–0.83)
Age (y)							
70 to 79		Reference	Reference	Reference	Reference	Reference	Reference
80 to 89		2.38 (2.08–2.73)	2.21 (1.93–2.52)	2.11 (1.84–2.41)	2.30 (1.98–2.67)	2.27 (1.98–2.61)	2.34 (2.07–2.72)
≥90		6.82 (5.67–8.21)	5.56 (4.57–6.75)	5.00 (4.11–6.08)	5.86 (4.71–7.29)	5.63 (4.51–7.04)	6.01 (4.84–7.46)
Female gender		0.83 (0.72–0.95)	0.83 (0.72–0.96)	0.72 (0.62–0.82)	0.77 (0.66–0.89)	0.79 (0.66–0.94)	0.76 (0.64–0.90)
Education (y)							
<12			Reference	Reference	Reference	Reference	Reference
12			0.32 (0.28–0.38)	0.40 (0.35–0.47)	0.44 (0.38–0.51)	0.44 (0.38–0.51)	0.45 (0.39–0.52)
13 to 15			0.24 (0.19–0.29)	0.32 (0.26–0.40)	0.35 (0.28–0.43)	0.35 (0.28–0.44)	0.36 (0.29–0.45)
≥16			0.19 (0.15–0.24)	0.29 (0.22–0.37)	0.30 (0.23–0.39)	0.30 (0.23–0.39)	0.30 (0.23–0.39)
Net worth (1993 $)							
≤43,500				Reference	Reference	Reference	Reference
43,500 to 167,100				0.50 (0.43–0.57)	0.55 (0.47–0.63)	0.58 (0.50–0.66)	0.59 (0.51–0.68)
>167,100				0.34 (0.28–0.41)	0.38 (0.33–0.46)	0.42 (0.35–0.51)	0.44 (0.36–0.53)
Race							
White					Reference	Reference	Reference
Black					2.38 (1.97–2.86)	2.52 (2.11–3.02)	2.61 (2.19–3.12)
Other					2.35 (1.70–3.26)	2.60 (1.88–3.61)	2.60 (1.85–3.67)
Caregiver network							
Spouse present					1.17 (1.01–1.36)	1.19 (1.03–1.38)	1.19 (1.03–1.37)
Living child					1.02 (0.82–1.28)	1.01 (0.81–1.25)	1.02 (0.83–1.26)
Cardiovascular risks							
Stroke						2.86 (2.49–3.29)	2.86 (2.48–3.29)
Diabetes						1.08 (0.92–1.26)	1.07 (0.91–1.25)
Hypertension						0.84 (0.73–0.96)	0.82 (0.71–0.94)
Obesity						0.90 (0.73–1.11)	0.90 (0.73–1.11)
Heart disease						0.94 (0.81–1.10)	0.92 (0.79–1.07)
Smoking status							
Never						Reference	Reference
Former						0.99 (0.83–1.18)	0.98 (0.83–1.13)
Current						1.14 (0.85–1.54)	1.12 (0.83–1.52)
Other chronic conditions							
Lung disease							0.97 (0.80–1.17)
Cancer							0.92 (0.77–1.12)
Psychiatric problem							1.86 (1.62–2.14)

NOTE. CI includes those with mild, moderate, or severe CI. 95% confidence intervals are in parentheses. Adjusted ORs derived by using a logistic regression model with pooled 1993 (N = 7,393) and 2002 (N = 7,083) data, with CI (mild, moderate, or severe) as the dependent variable. Values greater than 1 indicate increased odds of CI.

Table 4
Unadjusted 2-year mortality, by cognitive function category, 1993 and 2002 cohorts

Cognitive function	1993 (N = 7,393)	2002 (N = 7,098)
	% Dead at 2-year follow-up	
Normal	8.6 (7.6–9.6)	8.2 (7.5–8.9)
Mild CI	18.5 (14.8–22.1)	16.8 (12.4–21.1)
Moderate/severe CI	25.9 (21.5–30.3)	29.8 (24.9–34.6)

NOTE. Values are weighted percentages derived by using the HRS respondent population weights to adjust for the complex sampling design of the HRS survey. 95% confidence intervals are in parentheses.

individuals included in the baseline 1993 survey, 453 (6.1%) were lost to follow-up in 2002. Among this group, 6.3% of those who had normal cognitive function in 1993 were lost, 4.6% of those with mild CI in 1993 were lost, and 3.4% of those with moderate/severe CI were lost. These results suggested that differential loss to follow-up was not an important reason for the lower prevalence of CI found in 2002, because the overall loss to follow-up rate was low (93.9% of individuals were accounted for), and those with poorer cognitive function at baseline were actually somewhat less likely to be lost to follow-up by the 2002 wave.

This study has a number of potential limitations that should be considered when interpreting the results. The HRS cognitive measures provided an assessment of cognitive function, but they did not allow the determination of a clinical diagnosis of dementia. However, we used cognitive categories and cutoff scores that have shown good correlation with dementia in prior studies; specifically limitations in ADLs and IADLs [37], extent of informal caregiving [37], and the likelihood of nursing home admission [51]. Another limitation related to the cognitive measures is that different instruments were used for self-respondents and proxy respondents. The currently available HRS data did not allow us to calibrate and validate the instruments against one another. However, a recently completed dementia substudy of the HRS—the ADAMS [38]—administered both the self and proxy instruments for each respondent, so future analyses of these data will allow a calibration and validation of the instruments.

We excluded 473 nursing home residents from the 2002 sample to increase the comparability with the 1993 sample, which only included individuals living in the community. A significant change in the pattern of institutionalization between 1993 and 2002, specifically if those with CI were much more likely to be institutionalized in 2002 compared with 1993, could have contributed to the decrease in CI prevalence that we found. As future waves of HRS data become available to follow individuals from the community into nursing homes during longer time periods, the contribution of possible shifts in institutionalization patterns to changes in CI prevalence in the community will be more easily determined.

There are also data limitations regarding the self-report of comorbid health conditions and the treatments for those conditions. Diagnostic criteria for health conditions (eg, hypertension and diabetes), as well as the threshold to undertake diagnostic work-ups for health conditions, change over time. These diagnostic changes likely account for some of the difference in self-reported prevalence of the chronic conditions included in our analysis. Finally, we did not have data on the use of cardiovascular and dementia medications, so we could not directly assess how the increasing use of these agents during the time period of our study might have affected overall brain health.

The strengths of this analysis include its large nationally representative samples of U.S. adults who used the same cognitive tests in both years. In addition, the HRS measured cognitive function directly and in a consistent way and also used a proxy informant to provide an assessment of memory and judgment for those respondents unable to participate. These features overcome the shortcomings of using dementia diagnoses obtained from administrative data and excluding those who are significantly impaired because data are not gathered from a proxy. The representative community sample of the HRS included a wide range of educational attainment, allowing a better assessment of the relationship of education to CI and mortality than most clinical samples in which individuals with low levels of education are often under-represented. We also had nearly complete 2-year mortality follow-up of the more than 7,000 individuals in each cohort. Hence, our mortality analyses are unlikely biased by nonrandom attrition from the cohorts.

In summary, our findings engender optimism regarding trends in the overall cognitive health and quality of life of older Americans and support the hypothesis of a possible compression of cognitive morbidity between 1993 and 2004, with fewer older Americans reaching a threshold of significant CI and a more rapid decline to death among those who did. Given the complexity of making valid comparisons of population cognitive health across different birth cohorts, replication of these findings with future waves of the HRS, other longitudinal studies in the United States, and longitudinal studies in other countries is necessary to further test the compression of cognitive morbidity hypothesis. The growth in the elderly population in the coming decades increases the public health importance of better understanding trends in cognitive health and whether a societal investment in building and maintaining cognitive reserve through formal education in childhood, as well as continued cognitive stimulation during work and leisure in adulthood, might help limit the future burden of dementia, especially among the oldest-old.

Acknowledgments

The National Institute on Aging (NIA) provided funding for the Health and Retirement Study (U01 AG09740), data

Table 5
Unadjusted and adjusted HRs for 2-year mortality, 1993 and 2002 cohorts

	1993			2002		
	Unadjusted	Age- and sex-adjusted	Fully adjusted*	Unadjusted	Age- and sex-adjusted	Fully adjusted*
Cognitive function						
Normal	Reference	Reference	Reference	Reference	Reference	Reference
Mild CI	2.24 (1.85–2.72)	1.90 (1.56–2.30)	1.57 (1.23–2.00)	2.16 (1.59–2.94)	1.82 (1.34–2.47)	1.03 (0.65–1.65)
Moderate/severe CI	3.36 (2.74–4.13)	2.53 (2.04–3.14)	1.84 (1.41–2.40)	4.12† (3.22–5.26)	3.11‡ (2.34–4.12)	1.88 (1.33–2.65)
Education (y)						
<12			Reference			Reference
12			0.86 (0.70–1.04)			0.82 (0.66–1.02)
13–15			0.85 (0.68–1.06)			0.64 (0.51–0.81)
≥16			0.84 (0.59–1.21)			0.88 (0.66–1.19)
Cognitive function × education						
Mild CI × 12			1.44 (0.70–2.95)			3.65 (1.74–7.67)
Mild CI × 13–15			0.63 (0.06–6.59)			2.87 (1.05–7.90)
Mild CI × ≥16			2.25 (0.95–5.34)			0.40 (0.04– 3.73)
Mod/Sev CI × 12			1.85 (1.05–3.28)			1.71 (0.85–3.44)
Mod/Sev CI × 13–15			2.69 (1.20–6.00)			2.93 (1.06–8.07)
Mod/Sev CI × ≥16			1.14 (0.32–4.05)			2.44 (0.78–7.62)

NOTE. 95% confidence intervals are in parentheses.

Abbreviation: Mod/Sev, Moderate/severe.

* Includes cognitive function, age, race, gender, net worth, education, potential caregiver network, chronic conditions, smoking status, and cognitive function × education level interaction terms.

† $P = .08$ for comparison of unadjusted HRs in 2002 vs 1993 (4.12 vs 3.36).

‡ $P = .09$ for comparison of age- and sex-adjusted HRs in 2002 vs 1993 (3.11 vs 2.53).

from which were used for this analysis. The Health and Retirement Study is performed at the Survey Research Center, Institute for Social Research, University of Michigan. Additional support was provided by grants from the NIA (R01 AG015911, R01 AG019805, and R01 AG027010) and the Harvard University Interfaculty Program for Health Systems Improvement. Dr Langa was supported by a Career Development Award from the National Institute on Aging (K08 AG019180) and a Paul Beeson Physician Faculty Scholars in Aging Research award. Dr Karlawish was supported by a Greenwall Faculty Scholar in Ethics and NIH grants P30 AG10124, R01 AG020627, R01 MH071634. Dr Larson was supported by NIA grant U01 AG06178.

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