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A Comparison of the Prevalence of Dementia in the United States in 2000 and 2012

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Abstract

Importance—The aging of the US population is expected to lead to a large increase in the number of adults with dementia, but some recent studies in the US and other high-income countries suggest that the age-specific risk of dementia may have declined over the last 25 years. Clarifying current and future population trends in dementia prevalence and risk has important implications for patients, families, and government programs.

Objective—To compare the prevalence of dementia in the United States in 2000 and 2012.

Design—We used data from the Health and Retirement Study (HRS), a nationally representative population-based longitudinal survey of US adults.

Setting—Population-based prospective cohort of US adults.

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Participants—Individuals aged 65 or older from the 2000 (N = 10,546) and 2012 (N = 10,511) waves of the HRS.

Main Outcomes and Measures—Dementia was identified in each year using HRS cognitive measures and validated methods for classifying self-respondents, as well as those represented by a proxy. Logistic regression was used to identify socioeconomic and health variables associated with change in dementia prevalence between 2000 and 2012.

Results—Dementia prevalence among those aged 65 or older decreased from 11.6% in 2000 to 8.8% (8.6% with age- and sex-standardization) in 2012 (P < 0.001). More years of education was associated with a lower risk for dementia, and average years of education increased significantly (from 11.8 to 12.7 years; P < 0.001) between 2000 and 2012. The decline in dementia prevalence occurred even though there was a significant age- and sex-adjusted increase between years in the cardiovascular risk profile (e.g., prevalence of hypertension, diabetes, and obesity) among older US adults.

Conclusions—The prevalence of dementia in the United States declined significantly between 2000 and 2012. An increase in educational attainment was associated with some of the decline in dementia prevalence, but the full set of social, behavioral, and medical factors contributing to the decline is still uncertain. Continued monitoring of trends in dementia incidence and prevalence will be important for better gauging the full future societal impact of dementia as the number of older adults increases in the decades ahead.

Introduction

Dementia, a decline in memory and other cognitive functions that lead to a loss of independent function, is a common and feared geriatric syndrome that affects an estimated 4 to 5 million older adults in the United States,¹ and has a large social and economic impact on patients, families, and government programs.² Although the number of older adults with dementia in the US and around the world is expected to grow up to three-fold by 2050 due to the large increase in the size of the elderly population,³ recent studies suggest that the age-specific risk of dementia may have actually declined in some high-income countries over the past 25 years, perhaps due to increasing levels of education and better control of key cardiovascular risk factors, such as hypertension, diabetes, and hypercholesterolemia.^{4–6} For instance, the incidence of dementia among older participants in the Framingham Heart Study declined by about 20% per decade between 1977 and 2008, and the decline in risk was seen only among those with at least a high school education.⁷

If confirmed in representative populations, a decline in age-specific risk for dementia would have important implications for public health and public policy. For instance, a recent population-based study of dementia in England found a 24% decline in the expected number of cases of dementia between 1991 and 2011 (a 6.5% prevalence among older adults in 2011, compared to 8.3% in 1991, p=0.003), which translates to more than 200,000 fewer cases of dementia.⁸

There have been changes over the last two to three decades in both the prevalence and treatment of cardiovascular risk factors that also influence the risk for dementia. For instance, 23% of US adults were obese in 1990 compared to 35% in 2012^{9,10}; among adults

aged 65+, the prevalence of diabetes increased from 9% to 21%.¹⁰ However, intensity of treatment for diabetes, hypertension, and high cholesterol has increased with more patients achieving treatment goals, and a significant decline in the vascular complications of diabetes such as heart attack, stroke, and lower-extremity amputations,¹¹ suggesting that there could be a "spill-over" benefit of a decline in the vascular-related risk for dementia.^{4,7}

Rising levels of education among US adults over the past 25 years may also have contributed to decreased dementia risk. The proportion of adults aged 65 or older with a high school diploma increased from 53% in 1990 to 80% in 2010, while the proportion with a college degree increased from 11% to 23%.¹² More years of formal education is associated with a reduced risk of dementia, likely through multiple causal pathways, including a direct effect on brain development and function (i.e., the building of "cognitive reserve"), health behaviors, as well as the general health advantages of having more wealth and opportunities.^{13–15}

To further address these questions, we used the Health and Retirement Study (HRS), a large nationally representative prospective cohort study of US adults, to test whether the age-specific prevalence of dementia declined in the United States between 2000 and 2012. Since most prior studies of dementia trends have used samples from geographically-restricted regions and with limited representation of minority populations, we could determine if those studies' findings were replicated in a sample representative of the US population.

Methods

Data and Study Sample

We used data from the 2000 and 2012 waves of the HRS. The HRS is a biennial, survey of US adults that started in 1992 and collects a wide-range of data on health, cognition, family, employment, and wealth.¹⁶ The HRS follows respondents longitudinally until death, and new cohorts have been enrolled at different times since the 1992 baseline interviews in order to maintain population representativeness as the study sample has aged.¹⁶ As a result, 4,008 individuals in our analysis were included in both the 2000 and 2012 cohorts, while 6,538 were included only in 2000 and 6,503 only in 2012.

Our study sample included all HRS participants aged 65 or older, living in the community or in nursing homes in 2000 and 2012. There were 10,546 respondents in 2000 and 10,516 respondents in 2012, after excluding 165 (1.5%) and 218 (2.0%) respondents from the 2000 and 2012 samples, respectively, due to missing data for one or more covariates used in the analysis. If a respondent is unable or unwilling to participate in the survey, the HRS attempts to identify a proxy respondent (usually a spouse or adult child) to complete the survey for them. There were 1,317 (12.5% unweighted) respondents represented by a proxy in 2000 and 860 (8.2% unweighted) in 2012. The response rate for the full HRS sample was 88% in 2000 and 89% in 2012.¹⁷

Informed consent to participate in the HRS is obtained from all respondents. The HRS has been approved by the Health Sciences and Behavioral Sciences IRB at the University of Michigan.

Measurement of Cognitive Function and Cognitive Category Definitions

The HRS assesses cognitive function in self-respondents with a range of tests adapted from the Telephone Interview for Cognitive Status (TICS). Based on our prior work,¹⁸ we used a 27-point cognitive scale that included an immediate and delayed 10-noun free recall test, a serial seven subtraction test, and a backwards count from 20 test. Cut-points for normal, cognitive impairment—no dementia (CIND), and dementia were validated against the prevalence of CIND and dementia in the Aging, Demographics, and Memory Study (ADAMS), an HRS sub-study of Alzheimer's disease and dementia that used a 3–4 hour inhome neuropsychological and clinical assessment as well as expert clinician adjudication to obtain a "gold-standard" diagnosis of CIND or dementia.^{18,19} Respondents who scored from 0 to 6 on the 27-point scale were classified as having dementia, 7 to 11 as having CIND, and 12 to 27 as normal.

For respondents represented by a proxy, an 11-point scale was developed using the proxy's assessment of the respondent's memory ranging from excellent to poor (score 0–4), the proxy's assessment of whether the respondent had limitations in five instrumental activities of daily living (IADLs; managing money, taking medication, preparing hot meals, using phones, and doing groceries; score 0–5), and the survey interviewer's assessment of whether the respondent had difficulty completing the interview because of a cognitive limitation (score 0–2 indicating, none, some, and prevents completion). Using this information, respondents with high scores (6–11) were classified as having dementia, and those with midrange scores (3–5) as having CIND.¹⁸

Using the ADAMS dementia diagnosis as the gold standard, this categorization method correctly classifies 78% of HRS respondents as having dementia or not (76% of self-respondents and 84% of those represented by a proxy).¹⁸

More detail on the HRS self-report and proxy cognition measures is available at the HRS web site. $^{\rm 20}$

Independent Variables Used as Covariates

The following sociodemographic measures were included in the regression analyses as independent variables: age, self-reported race/ethnicity (white, black, Hispanic, other), sex, education (<12 years; 12 years; 13 to 15 years, and 16 years), and net worth (quartiles in year 2000 dollars). The self-reported chronic medical conditions and cardiovascular risk factors included were: stroke, diabetes, heart disease, hypertension, and body-mass index (BMI, derived from self-reported height and weight). All of these sociodemographic and health measures were selected for inclusion in the regression analyses a priori, based on prior studies suggesting that they are associated with dementia risk.

Analytic Framework

For descriptive analyses (Tables 1 and 2), the 2012 sample was age- and sex-standardized to the 2000 population using direct standardization. For multivariable analyses (Table 4), we pooled data from 2000 and 2012 and estimated logistic regression models with a dichotomous dependent variable indicating whether an individual had dementia (the

reference group included those with normal cognition or CIND). A linear trend variable that took the value of 0 in 2000 and 1 in 2012 was included in the regression models. An odds ratio (OR) less than 1 for this trend variable would indicate a decrease in the prevalence of dementia (i.e., a decrease in the overall odds of dementia among the 65+ population) between 2000 and 2012. We estimated four separate logistic models with different sets of independent variables added sequentially (e.g., trend variable only, an age- and sex-adjusted model, and then subsequent models that included sociodemographic variables and then health variables) in order to better assess which variables were associated with a change in the prevalence of dementia between 2000 and 2012. We tested for interactions between each independent variable and the year of observation.

Statistical analyses were performed using STATA (Release 13.1, Stata Corp, College Station, TX). HRS sampling weights were used to adjust for non-response and the complex sampling design of the HRS survey.

Results

Characteristics of the Study Sample

Table 1 shows the characteristics of the 2000 and 2012 study cohorts (with age- and sexstandardization to the 2000 cohort). Compared to the 2000 cohort, the 2012 cohort had a significantly larger proportion of those who were aged 85 or older, but the average age for the full cohort was similar across the two years. The 2012 cohort had significantly more years of education; individuals with fewer than 12 years of education comprised 32.6% of the sample in 2000, but only 20.6% in 2012 (P<0.001). On average, individuals in the 2012 cohort had nearly 1 more year of education, compared to those in the 2000 cohort (12.7 years vs. 11.8 years, P<0.001). There was a greater disparity in household net worth in 2012 (in constant 2000 dollars), with a greater proportion of the 2012 cohort in both the lowest and highest wealth quartiles (P=0.02).

The 2012 cohort had significantly higher rates of self-reported cardiovascular risk factors, including obesity (29.2% in 2012 vs. 18.3% in 2000, P<0.001), diabetes (24.7% vs. 16.4%, P<0.001), and hypertension (67.6% vs. 54.6%, P<0.001). The prevalence of heart disease increased from 29.1% to 31.8% between 2000 and 2012 (P<0.001), but the prevalence of stroke did not change significantly. There was a small decline between 2000 and 2012 in the proportion of individuals with 1 or more IADL limitations, but this change was not significant (P=0.14). The proportion of the sample living in a nursing home at the time of their HRS interview declined from 4.4% in 2000 to 2.8% in 2012 (P<0.001), and the weighted and standardized proportion of the HRS sample represented by a proxy respondent declined from 12.1% in 2000 to 6.6% in 2012 (P<0.001).

Trend in Prevalence and Adjusted Relative Risk of Dementia

Table 2 displays the weighted percentage of individuals in each cognitive function category in 2000 and 2012, and shows a significant decrease in the proportion of individuals aged 65+ with dementia between 2000 and 2012 (11.6% in 2000 compared to 8.8% in 2012 [P<0.001]). The prevalence of CIND also decreased significantly across the 2 cohorts from

21.2% to 18.8% (P<0.001). After age- and sex-standardizing the 2012 cohort to the 2010 cohort, the decline in dementia prevalence was slightly greater (8.6% in 2012) because of the greater proportion of those who were age 85+ in 2012. Tables 3a and 3b provide results stratified by age groups (65–74, 75–84, and 85+).

Table 4 reports the results of four different logistic regression models with the presence of dementia as the outcome variable, using pooled 2000 and 2012 data. The trend variable in the first row of the table represents the odds ratio (OR) of dementia in 2012 compared to 2000. Model 1 shows the significant decline (OR = 0.73, 95% CI: 0.67 to 0.82) in unadjusted dementia prevalence already noted in Table 2, and Model 2 shows the OR after adjusting for differences across the cohorts in age and sex (OR = 0.69, 95% CI: 0.62 to 0.77). Controlling for education, net worth, and race (Model 3) explained 9 percentage points of the decrease in age- and sex-standardized odds of dementia between 2000 and 2012 (OR = 0.78, 95% CI: 0.70 to 0.88), while the addition of cardiovascular risk factors and BMI (Model 4) accounted for 4 additional percentage points of the decline in prevalence (OR = 0.82, 95% CI: 0.73 to 0.92) In the fully adjusted model (Model 4), more years of education and higher net worth were associated with a significantly lower odds of dementia, while older age, being African-American or Hispanic, and having a history of stroke or diabetes were all associated with increased odds. Being underweight was also associated with higher odds of dementia, while being overweight or obese was associated with lower odds of dementia, compared to those at normal BMI.

When testing for an interaction effect between each independent variable and year, controlling for the main effects of all other variables, heart disease had a significantly lower OR for dementia in 2012 compared to 2000 (P<0.001). No other interactions were significant at the P<0.05 level.

Discussion

In a large nationally representative survey of older Americans we found that, among those aged 65 or older, the prevalence of dementia decreased from 11.6% to 8.8% between 2000 and 2012, representing an absolute decrease of 2.8 percentage points, and a relative decrease of about 24%. Educational attainment increased significantly, with those aged 65+ in 2012 having nearly one additional year of education compared to the 2000 cohort. After controlling for the socioeconomic factors of education, wealth, and race / ethnicity, controlling for changes in the prevalence of cardiovascular risk factors did not explain much of the additional difference in dementia risk across the two cohorts.

Our study, along with prior studies, supports the notion that "cognitive reserve" resulting from early-life and life-long education and cognitive stimulation may be a potent strategy for the primary prevention of dementia in both high- and low-income countries around the world.²¹ However, it should be noted that the relationships among education, brain biology, and cognitive function are complex and likely multi-directional; for instance, a number of recent population-based studies have shown genetic links with level of educational attainment,^{22,23} and with the risk for cognitive decline in later-life.²⁴ Higher levels of educational attainment are also associated with health behaviors (e.g., physical activity, diet,

and smoking), more cognitively-complex occupations, and better access to health care, all of which may play a role in decreasing lifetime dementia risk.

The prevalence of obesity and diabetes among those aged 65+ increased significantly between 2000 and 2012, and diabetes was associated with 39% higher odds of dementia, after controlling for all other factors. As in prior studies among older adults, we found that obesity was associated with a decreased risk of dementia, consistent with the hypothesis that, while obesity in mid-life may increase risk for later-life cognitive decline and dementia, obesity at older ages may be associated with cognitive and other health advantages.^{25–27} The trend toward a declining risk for dementia in the face of a large increase in the prevalence of diabetes suggests that improvements in treatments between 2000 and 2012 may have decreased dementia risk, along with the documented declines in the incidence of common diabetes-related complications such as heart attack, stroke, and amputations.¹¹ Our finding of a significant decline between 2000 and 2012 of the heart disease-related OR for dementia would also be consistent with improved cardiovascular treatments leading to a decline in dementia risk. To explore this hypothesis further, we used additional HRS data on selfreported treatments for diabetes (either oral medications or insulin). The proportion of adults with diabetes reporting either oral medication or insulin use increased from 86% in 2000 to 90% in 2012 (P <0.01). Further, the interaction of diabetes treatment by survey year in our regression model was statistically significant (P<0.01), suggesting that diabetes treatment in 2012 was associated with a significantly lower OR of dementia compared to 2000.

Our findings are consistent with a number of recent studies that also found declines in dementia incidence or prevalence in high-income countries around the world,^{6–8,28–31} and also suggest that the trend toward a declining prevalence of cognitive impairment or dementia in the US that we found between 1993 and 2002 using earlier waves of the HRS data¹³ has continued through 2012, even with significant increases in the prevalence of cardiovascular risk factors that may increase dementia risk. Our findings are consistent with the declining incidence of dementia found over the past four decades in the Framingham Heart Study,⁷ as well as the decline in dementia prevalence between 1991 and 2011 in the Cognitive Function and Ageing Study (CFAS) in England.⁸ Both the Framingham and CFAS studies also pointed to increases in education and better control of cardiovascular risk factors as likely contributors to declining dementia risk.^{7,8}

Our study has several limitations. Our dementia diagnosis is based on a limited set of cognitive tests, although prior validation studies show a 78% concordance for dementia diagnosis when using these tests compared to the detailed ADAMS clinical evaluation.¹⁸ The recent Framingham⁷ and CFAS⁸ studies both used more extensive cognitive testing and clinical information when making a dementia diagnosis in their studies, so likely have less diagnostic mis-classification. In addition, although we used a validated method to define diagnostic categories for both self-respondents and respondents represented by a proxy, the proportion of the HRS sample represented by a proxy declined significantly between 2000 and 2012 (from 12.5% to 8.2% unweighted), likely due in part to a change in HRS field procedures between these two waves. In 2006, the HRS purposefully increased the proportion of interviews administered face-to-face in respondents' homes, and decreased the proportion administered by phone. Since 2006, about one-half of HRS interviews at each

wave have been administered face-to-face, while prior to 2006 only about 20% were face-toface. This shift in survey mode likely encouraged an increase in self-interviews that in prior waves would have been completed by proxy, possibly leading to a change in the calibration of the self- and proxy-cognitive measures to dementia status. Another potential limitation is that changes in diagnostic thresholds and in the frequency of diagnostic testing between 2000 and 2012 may have affected the self-reported prevalence of cardiovascular risk factors, and the relationship of treatments to both cardiovascular and cognitive outcomes. Finally, the accuracy of self-report of cardiovascular risk factors may be less reliable for those with cognitive impairment or dementia.

In conclusion, using nationally representative data we found a significant decline in dementia prevalence among older US adults between 2000 and 2012, using the same cognitive measures and the same diagnostic classification strategy in both years. Increases in the level of education among the later-born cohort accounted for some of the decreased dementia risk, and there was some evidence that improvements in treatments for cardiovascular risk factors (e.g., diabetes) may also have played a role. However, the full set of social, behavioral, and medical factors contributing to the decline in dementia prevalence is still uncertain. Continued monitoring of trends in dementia incidence and prevalence will be important for better gauging the full future societal impact of dementia as the number of older adults increases in the decades ahead, as well as for clarifying potential protective and risk factors for cognitive decline.

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References

- Plassman BL, Langa KM, Fisher GG, et al. Prevalence of dementia in the United States: the aging, demographics, and memory study. Neuroepidemiology. 2007; 29(1–2):125–132. [PubMed: 17975326]
- Hurd MD, Martorell P, Delavande A, Mullen KJ, Langa KM. Monetary costs of dementia in the United States. N Engl J Med. 2013; 368(14):1326–1334. [PubMed: 23550670]
- Prince M, Bryce R, Albanese E, Wimo A, Ribeiro W, Ferri CP. The global prevalence of dementia: a systematic review and metaanalysis. Alzheimers Dement. 2013; 9(1):63–75. e62. [PubMed: 23305823]
- Larson EB, Yaffe K, Langa KM. New insights into the dementia epidemic. N Engl J Med. 2013; 369(24):2275–2277. [PubMed: 24283198]
- Gerstorf D, Hulur G, Drewelies J, et al. Secular changes in late-life cognition and well-being: Towards a long bright future with a short brisk ending? Psychol Aging. 2015; 30(2):301–310. [PubMed: 25799003]
- Wu YT, Fratiglioni L, Matthews FE, et al. Dementia in western Europe: epidemiological evidence and implications for policy making. Lancet Neurol. 2016; 15(1):116–124. [PubMed: 26300044]

- Satizabal CL, Beiser AS, Chouraki V, Chene G, Dufouil C, Seshadri S. Incidence of Dementia over Three Decades in the Framingham Heart Study. N Engl J Med. 2016; 374(6):523–532. [PubMed: 26863354]
- Matthews FE, Arthur A, Barnes LE, et al. A two-decade comparison of prevalence of dementia in individuals aged 65 years and older from three geographical areas of England: results of the Cognitive Function and Ageing Study I and II. Lancet. 2013; 382(9902):1405–1412. [PubMed: 23871492]
- 9. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014; 311(8):806–814. [PubMed: 24570244]
- Ogden, C.; Carroll, M. Prevalence of overweight, obesity, and extreme obesity among adults: United States, trends 1960–1962 through 2007–2008. Jun. 2010 http://www.cdc.gov/nchs/data/ hestat/obesity_adult_07_08/obesity_adult_07_08.pdf
- Gregg EW, Li Y, Wang J, et al. Changes in diabetes-related complications in the United States, 1990–2010. N Engl J Med. 2014; 370(16):1514–1523. [PubMed: 24738668]
- Federal Interagency Forum on Aging-Related Statistics. Older Americans 2012: Key Indicators of Well-Being. Washington, DC: U.S. Government Printing Office; Jun. 2012 Available at: http:// www.agingstats.gov/Main_Site/Data/2012_Documents/Population.aspx
- Langa KM, Larson EB, Karlawish JH, et al. Trends in the prevalence and mortality of cognitive impairment in the United States: is there evidence of a compression of cognitive morbidity? Alzheimers Dement. 2008; 4(2):134–144. [PubMed: 18631957]
- Vemuri P, Lesnick TG, Przybelski SA, et al. Association of lifetime intellectual enrichment with cognitive decline in the older population. JAMA Neurol. 2014; 71(8):1017–1024. [PubMed: 25054282]
- 15. Stern Y, Albert S, Tang MX, Tsai WY. Rate of memory decline in AD is related to education and occupation: cognitive reserve? Neurology. 1999; 53(9):1942–1947. [PubMed: 10599762]
- 16. Sonnega A, Faul JD, Ofstedal MB, Langa KM, Phillips JW, Weir DR. Cohort Profile: the Health and Retirement Study (HRS). Int J Epidemiol. 2014; 43(2):576–585. [PubMed: 24671021]
- 17. Health and Retirement Study (HRS). Sample Sizes and Response Rates. 2011. http:// hrsonline.isr.umich.edu/sitedocs/sampleresponse.pdf
- Crimmins EM, Kim JK, Langa KM, Weir DR. Assessment of cognition using surveys and neuropsychological assessment: the Health and Retirement Study and the Aging, Demographics, and Memory Study. J Gerontol B Psychol Sci Soc Sci. 2011; 66(Suppl 1):i162–171. [PubMed: 21743047]
- Langa KM, Plassman BL, Wallace RB, et al. The Aging, Demographics, and Memory Study: study design and methods. Neuroepidemiology. 2005; 25(4):181–191. [PubMed: 16103729]
- 20. Ofstedal, MB.; Fisher, GG.; Herzog, AR. Documentation of Cognitive Functioning Measures in the Health and Retirement Study. 2005. http://hrsonline.isr.umich.edu/sitedocs/userg/dr-006.pdf
- Norton S, Matthews FE, Barnes DE, Yaffe K, Brayne C. Potential for primary prevention of Alzheimer's disease: an analysis of population-based data. Lancet Neurol. 2014; 13(8):788–794. [PubMed: 25030513]
- Rietveld CA, Medland SE, Derringer J, et al. GWAS of 126,559 individuals identifies genetic variants associated with educational attainment. Science. 2013; 340(6139):1467–1471. [PubMed: 23722424]
- Rietveld CA, Esko T, Davies G, et al. Common genetic variants associated with cognitive performance identified using the proxy-phenotype method. Proc Natl Acad Sci U S A. 2014; 111(38):13790–13794. [PubMed: 25201988]
- 24. Deary IJ, Yang J, Davies G, et al. Genetic contributions to stability and change in intelligence from childhood to old age. Nature. 2012; 482(7384):212–215. [PubMed: 22258510]
- 25. Fitzpatrick AL, Kuller LH, Lopez OL, et al. Midlife and late-life obesity and the risk of dementia: cardiovascular health study. Arch Neurol. 2009; 66(3):336–342. [PubMed: 19273752]
- Hughes TF, Borenstein AR, Schofield E, Wu Y, Larson EB. Association between late-life body mass index and dementia: The Kame Project. Neurology. 2009; 72(20):1741–1746. [PubMed: 19451529]

- Tolppanen AM, Ngandu T, Kareholt I, et al. Midlife and late-life body mass index and late-life dementia: results from a prospective population-based cohort. J Alzheimers Dis. 2014; 38(1):201– 209. [PubMed: 23948937]
- Schrijvers EM, Verhaaren BF, Koudstaal PJ, Hofman A, Ikram MA, Breteler MM. Is dementia incidence declining?: Trends in dementia incidence since 1990 in the Rotterdam Study. Neurology. 2012; 78(19):1456–1463. [PubMed: 22551732]
- Qiu C, von Strauss E, Backman L, Winblad B, Fratiglioni L. Twenty-year changes in dementia occurrence suggest decreasing incidence in central Stockholm, Sweden. Neurology. 2013; 80(20): 1888–1894. [PubMed: 23596063]
- Christensen K, Thinggaard M, Oksuzyan A, et al. Physical and cognitive functioning of people older than 90 years: a comparison of two Danish cohorts born 10 years apart. Lancet. 2013; 382(9903):1507–1513. [PubMed: 23849796]
- Dodge HH, Zhu J, Lee CW, Chang CC, Ganguli M. Cohort effects in age-associated cognitive trajectories. J Gerontol A Biol Sci Med Sci. 2014; 69(6):687–694. [PubMed: 24270062]

Table 1

Characteristics of the 2000 and 2012 cohorts

	2000 (N=10,546)	2012 (10,511)
Age**		
65–74	5,566 (52.7)	4,983 (55.2)
75–84	3,668 (35.9)	3,991 (31.4)
85+	1,312 (11.4)	1,537 (13.4)
Mean \pm SD	75.0 ± 7.7	74.8 ± 7.3
Sex**		
Male	4,482 (41.6)	4,414 (43.8)
Female	6,064 (58.4)	6,097 (56.3)
Race**		
White	8,364 (84.8)	7,934 (82.0)
Black	1,293 (8.4)	1,450 (8.7)
Hispanic	702 (4.9)	901 (7.0)
Other	187 (1.9) 226 (2.3	
Education (years) **		
< 12	3,641 (32.6)	2,517 (20.6)
12	3,467 (33.7)	3,631 (34.3)
13–15	1,764 (17.2)	2,160 (21.5)
16+	1,673 (16.6)	2,203 (23.6)
Mean \pm SD **	11.8 ± 3.6	12.7 ± 2.9
Net worth (year 2000 \$s)*		
32,000	2,465 (22.6)	2,739 (24.1)
32,001-120,100	2,794 (25.4)	2,661 (24.3)
120,101-300,500	2,699 (26.4)	2,536 (24.5)
300,501	2,588 (25.6)	2,575 (27.1)
$Mean \pm SD$	295,396 ± 673,843	329,765 ± 883,353
Median [*]	121,000	114,000
ADL limitations †		
0	7,611 (72.5)	7,361 (72.9)
1–3	2,164 (20.1)	2,276 (20.4)
4–6	771 (7.3)	874 (6.7)
Mean \pm SD	.69 ± 1.5	.67 ± 1.3
IADL limitations $\stackrel{\neq}{\downarrow}$		
0	8,467 (80.4)	8,271 (81.3)
1–3	1,502 (14.1)	1,600 (14.2)

	2000 (N=10,546)	2012 (10,511)
4–5	568 (5.5)	634 (4.5)
Mean \pm SD	.48 ± 1.3	.44 ± 1.1
Cardiovascular Risk Factors		
Stroke	1,068 (10.2)	1,170 (10.0)
Diabetes **	1,807 (16.4)	2,760 (24.7)
Heart Disease **	3,063 (29.1)	3,486 (31.8)
Hypertension **	5,826 (54.6)	7,324 (67.6)
BMI**		
< 18.5	330 (3.2)	245 (2.2)
18.5–24.9	4,101 (39.5)	3,299 (30.8)
25.0-29.9	4,133 (39.1)	3,940 (37.8)
30.0	1,982 (18.3)	3,027 (29.2)
Mean \pm SD **	26.1 ± 5.2	27.7 ± 5.4
Nursing Home Resident **	405 (4.4)	434 (2.8)
Respondent Type **		
Self	9,229 (87.9)	9,651 (93.4)
Proxy	1,317 (12.1)	860 (6.6)

The reported P-value is for a chi-square or t-test for a significant difference in proportion or mean between years, after adjusting for the age and sex differences across the two cohorts.

 † ADLs indicates Activities of Daily Living (eating, transferring, toileting, dressing, bathing, and walking across a room).

^{*I*}IADLs indicates Instrumental Activities of Daily Living (preparing meals, grocery shopping, making phone calls, taking medications, managing money).

Values in parentheses are weighted percentages derived using the HRS sampling weights to adjust for the complex design of the HRS survey. Weighted percentages for the 2012 sample are age- and sex-standardized to the 2000 sample using direct standardization.

*P<0.05 for difference between 2000 and 2012.

** P<0.001 for difference between 2000 and 2012.

Table 2

Cognitive Function, Age 65+, 2000 and 2012 Cohorts

Cognitive Function	2000 (N=10,546)	2012 [*] (N=10,511)	2012 [*] (N=10,511)
		Crude Rate	Age- and Sex- Standardized Rate
Normal	6,966 (67.2)	7,114 (72.4)	7,114 (72.6)
	(65.8–68.6)	(71.1–73.6)	(71.1–73.6)
CIND	2,293 (21.2)	2,224 (18.8)	2,224 (18.8)
	(20.1–22.3)	(17.8–19.9)	(17.8–19.9)
Dementia	1,287 (11.6)	1,173 (8.8)	1,173 (8.6)
	(10.7–12.7)	(8.2–9.4)	(8.2–9.4)

Values in parentheses are weighted percentages (and 95% confidence intervals) derived using the HRS sampling weights to adjust for the complex design of the HRS survey.

Column 3 shows the age- and sex-standardized weighted percentages, after direct standardization of the 2012 cohort to the 2000 cohort.

CIND is Cognitive Impairment-No Dementia.

*P<0.001 for difference between 2000 and 2012.

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Table 3a. Cognitive l	Function, by Age, 2	2000 and 2012 Coho	rts					
	Age	65–74	Age 7	5 - 84	Age	85+	Total (A	.ge 65+)
Cognitive Function	2000 (N=5,566)	2012 (N=4,983)	2000 (N=3,668)	2012 (N=3,991)	2000 (N=1,312)	2012 (N=1,537)	2000 (N=10,546)	2012 (N=10,511)
Normal	4,320 (78.1) (76.5–79.7)	3,931 (82.8) (81.1–84.4)	2,231 (62.0) (60.1–64.0)	2,603 (67.5) (65.6–69.3)	415 (32.8) (30.3–35.4)	580 (40.8) (38.0–43.6)	6,966 (67.2) (65.8–68.6)	7,114 (72.4) (71.1–73.6)
CIND	942 (16.5) (15.2–17.8)	837 (14.0) (12.7–15.4)	924 (24.4) (23.0–25.9)	936 (22.6) (20.9–24.3)	427 (32.9) (29.5–36.5)	451 (29.9) (27.4–32.6)	2,293 (21.2) (20.1–22.3)	2,224 (18.8) (17.8–19.9)
Dementia	304 (5.4) (4.7-6.3)	215 (3.2) (2.7–3.8)	513 (13.6) (12.1–15.1)	452(9.9) (9.0 -10.9)	470 (34.4) (31.2–37.6)	506 (29.3) (26.9–31.8)	1,287 (11.6) (10.7–12.7)	$1,173\ (8.8)\\(8.2-9.4)$
Table 3b. Cognitive I	Function, by Age, 2	2000 and 2012 Coho	orts, Age- and Sex-	Standardized to 20	00 Population			
	Age	65–74	Age 7	5 - 84	Age	85+	Total (A	.ge 65+)
Cognitive Function	2000 (N=5,566)	2012 (N=4,983)	2000 (N=3,668)	2012 (N=3,991)	2000 (N=1,312)	2012 (N=1,537)	2000 (N=10,546)	2012 (N=10,511)
Normal	4,320 (78.1) (76.5–79.7)	3,931 (82.9) (81.1-84.4)	2,231 (62.0) (60.1-64.0)	2,603 (67.6) (65.6–69.3)	415 (32.8) (30.3–35.4)	580 (40.7) (38.0–43.6)	6,966 (67.2) (65.8–68.6)	7,114 (72.6) (71.2–73.7)
CIND	942 (16.5) (15.2–17.8)	837 (14.0) (12.7–15.4)	924 (24.4) (23.0–25.9)	936 (22.5) (20.9–24.3)	427 (32.9) (29.5–36.5)	451 (29.7) (27.4–32.6)	2,293 (21.2) (20.1–22.3)	2,224 (18.8) (17.8–19.9)
Dementia	304 (5.4) (4.7-6.3)	215 (3.2) (2.7–3.8)	513 (13.5) (12.1–15.1)	452(9.9) (9.0 -10.9)	470 (34.3) (31.2–37.6)	506 (29.6) (26.9–31.8)	$\begin{array}{c} 1,287\ (11.6)\\ (10.7{-}12.7)\end{array}$	$1,173\ (8.6)\\(8.1-9.3)$
Values in parentheses an	re weighted percents	ages (and 95% confi	dence intervals) deri	ved using the HRS	sampling weights to	adjust for the comp	lex design of the HR	S survey.

Values for 2012 weighted percentages in Table 3b are age- and sex-standardized to the 2000 population using direct standardization.

CIND is Cognitive Impairment-No Dementia.

Table 4

Odds Ratios for Presence of Dementia in 2000 and 2012 (N=21,057)

Variable	Model 1	Model 2	Model 3	Model 4
Trend (2012 vs. 2000)	.73 (.65 .82)	.69 (.62 .77)	.78 (.70 .88)	.82 (.73 .92)
Age (years)		1.13 (1.12–1.14)	1.13 (1.12–1.14)	1.12 (1.12–1.13)
Female sex		1.07 (.95–1.19)	.93 (.82–1.04)	.93 (.81–1.05)
Education (years)				
< 12			Ref.	Ref.
12			.44 (.39 .50)	.42 (.37 .48)
13–15			.37 (.31 .45)	.36 (.30 .44)
16			.28 (.22 .36)	.27 (.21 .35)
Net Worth (2000 \$s)				
32,000			Ref.	Ref.
32,001-120,100			.54 (.47 .64)	.57 (.48 .67)
120,101-300,500			.42 (.34 .51)	.45 (.37 .54)
300,501			.34 (.29 .40)	.36 (.31 .43)
Race				
White			Ref.	Ref.
Black			2.24 (1.92–2.61)	2.34 (1.98–2.77)
Hispanic			1.38 (1.10–1.74)	1.47 (1.16–1.87)
Other			1.41 (.80–2.48)	1.58 (.88–2.84)
Cardiovascular Risks				
Stroke				3.20 (2.70-3.79)
Diabetes				1.39 (1.19–1.62)
Hypertension				.97 (.84–1.11)
Heart disease				.84 (.74 .95)
BMI				
<18.5 (Underweight)				2.47 (1.88-3.24)
18.5–24.9 (Normal)				Ref.
25.0-29.9 (Overweight)				.70 (.61 .80)
30.0 (Obese)				.68 (.57 .80)

Values in parentheses are 95% confidence intervals..

Adjusted odds ratios derived using a logistic regression model with pooled 2000 (N=10,546) and 2012 (N=10,511) data, with dementia as the dependent variable.