

Published in final edited form as:

Alzheimers Dement. 2009 May ; 5(3): 227–233. doi:10.1016/j.jalz.2009.01.026.

Prevalence rates for dementia and Alzheimer’s disease in African Americans: 1992 vs. 2001

KS Hall, PhD¹, S Gao, PhD², O Baiyewu, MD³, KA Lane, MS², O Gureje, PhD³, J Shen, MS^{2,9}, A Ogunniyi, MBChB⁴, JR Murrell, PhD⁵, FW Unverzagt, PhD¹, J Dickens, MD^{1,7}, V Smith-Gamble, MD^{1,8}, and HC Hendrie, DSc^{1,6}

¹ Department of Psychiatry, Indiana University School of Medicine, Indianapolis, Indiana USA 46202

² Division of Biostatistics, Indiana University School of Medicine, Indianapolis, Indiana USA 46202

³ Department of Psychiatry, College of Medicine, University of Ibadan, Ibadan, Nigeria

⁴ Department of Medicine, University of Ibadan, Ibadan, Nigeria

⁵ Department of Pathology, Indiana University School of Medicine, Indianapolis, Indiana USA 46202

⁶ Regenstrief Institute, Inc., Indiana University School of Medicine, Indianapolis, Indiana USA

⁷ Madison Center, South Bend, Indiana USA

⁸ Richard L. Roudebush VA Medical Center, Indianapolis, Indiana USA

⁹ Pharmaceutical Product Development, Inc., Wilmington, North Carolina USA

Abstract

Background—This study compares age-specific and overall prevalence rates for dementia and Alzheimer’s disease (AD) in two non overlapping population-based cohorts of elderly African Americans in Indianapolis in 2001 and 1992.

Methods—Two-stage design, first stage is Community Screening Interview for Dementia (CSI-D). CSI-D scores grouped into good, intermediate and poor performance for selection into clinical assessment. Diagnosis was made using standard criteria in consensus diagnosis conference; clinicians

Corresponding Author: Kathleen S. Hall, Ph.D., Department of Psychiatry, Indiana University School of Medicine, 1111 West Tenth Street, PB A319, Indianapolis, IN 46202, Phone (317)274-1249, Fax (317)274-4139, E-mail: khall@iupui.edu.

Author Contributions: Dr. Hall had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Hall, Hendrie, Gao, Baiyewu, Gureje, Ogunniyi, Murrell, Unverzagt.

Acquisition of data: Hall, Baiyewu, Dickens, Gureje, Smith-Gamble, Ogunniyi, Unverzagt, Hendrie.

Analysis and interpretation of data: Gao, Lane, Shen, Hall, Hendrie, Unverzagt, Ogunniyi, Gureje, Baiyewu, Murrell.

Drafting of the manuscript: Hall, Hendrie, Gao, Lane.

Critical revision of the manuscript for important intellectual content: Hall, Hendrie, Gao, Lane, Unverzagt, Baiyewu, Gureje, Ogunniyi, Murrell, Dickens, Smith-Gamble, Shen.

Statistical Analysis: Gao, Shen, Lane.

Obtained funding: Hendrie, Hall.

Administrative technical or material support: Hall, Baiyewu, Dickens, Gureje, Smith-Gamble, Ogunniyi, Hendrie, Unverzagt.

Supervision: Hall, Hendrie, Gao, Unverzagt, Baiyewu, Gureje, Ogunniyi.

Final Approval: Hall, Hendrie, Gao, Lane, Baiyewu, Gureje, Shen, Ogunniyi, Murrell, Unverzagt, Dickens, Smith-Gamble.

Financial Disclosure: The authors report no conflicts of interest.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

blind to performance group. In 1992, interviewers went to randomly sampled addresses enrolling self identified African Americans age ≥ 65 ; of 2,582 eligible, 2,212 enrolled, (9.6% refused, 4.7% too sick). In 2001, Medicare roles were used for African Americans age ≥ 70 years; of 4,260 eligible, 1,892 (44%) enrolled, 1,999 (47%) refused, the remainder had other reasons.

Results—The overall age adjusted prevalence rate for dementia for age ≥ 70 years in 2001 was 7.45% (95 confidence interval [CI] 4.27–10.64) and for the 1992 cohort was 6.75% (95% CI 5.77–7.74). The overall age adjusted prevalence rate for age ≥ 70 years, for AD for the 2001 cohort was 6.77% (95% CI 3.65–9.90), and for the 1992 cohort was 5.47% (95% CI 4.51–6.42). Rates for dementia and AD were not significantly different in the two cohorts (dementia, $p=0.3534$, AD, $p=0.2649$).

Conclusions—We find no differences in prevalence rates of dementia and AD between 1992 and 2001 in spite of significant medical history and medical treatment differences in these population-based cohorts of African American elderly.

Keywords

Prevalence rates; Alzheimer's disease; African Americans; 1992 vs. 2001; population-based

1. Introduction

Dementia and Alzheimer's disease (AD) represent a significant public health challenge for our society and one that is only likely to increase as our population ages. A cluster of demographic, lifestyle, and medical factors have been identified which appear to alter the risk of developing dementia and AD(1,2). Prominent amongst these are cardiovascular risk factors including hypertension and diabetes(3). These are of particular interest because they are potentially treatable. Indeed, the importance of recognizing and treating these disorders has been generally accepted by the medical community. With the advent of new therapies like statins, more widespread and earlier treatment has been attributed, at least in part, to the decline in mortality for heart disease and stroke which has occurred over the past two decades(4). However, while the likelihood of treatment is increased, so also, is the prevalence of these disorders. In African Americans over the age of 65, hypertension climbed from 72.9% to 83.1% between 1994 and 2002, and during that same time diabetes rose from 26.4% to 35.5%(5). The effect of these trends on rates of dementia and AD are however unclear.

There have been few studies that have tracked cognitive outcomes over time, including cognitive decline, dementia, and AD in the USA. Two of these have reported that prevalence rates for cognitive impairment have in fact decreased. A study of a representative sample of U.S. elderly from 1982 to 1999 reported an average decline in prevalence of severe cognitive impairment from 5.7% to 2.9%(6). Another nationally representative study reported a reduction in prevalence of cognitive impairment from 12.2% in 1993 to 8.7% in 2002(7). However, a study conducted in Rochester, Minnesota reported prevalence rates for dementia increased from 1980 to 1985(8). The Indianapolis-Ibadan comparative epidemiological study of dementias compares rates and risk factors for dementing disorders in population-based samples of African Americans in Indianapolis and Yoruba in Ibadan, Nigeria. This study, which was initiated in 1992, provided us with a unique opportunity to compare prevalence rates over a decade in community dwelling elderly African Americans. In 2001, the population-based sample which was originally recruited in 1992 was enriched to replace original participants who had died or were otherwise lost to follow up. The purpose of this paper is to report on a comparison of the prevalence rates for dementia and AD for the 1992 and 2001 cohorts as well as their demographic and medical profiles.

2. Methods

2.1. Study Participants

The Indianapolis-Ibadan Dementia Project assembled samples of community dwelling African Americans living in Indianapolis, Indiana, in 1992. The details of the construction of the sample were previously reported(9). Interviewers went to randomly sampled addresses, and invited eligible residents to have an interview. Criteria for eligibility were: self identified as African American, age ≥ 65 years, with residence at a sampled address. At that time 2,212 individuals were enrolled, 249 (9.6%) refused, and 121 (4.7%) were too sick. In 1992, the original cohort had 1,500 individuals age ≥ 70 years. In 2001 new participants were recruited using Medicare rolls. After fulfilling the compliance procedures of Medicare, the study was given names and addresses, but no medical information, for African Americans age ≥ 70 years living in Indianapolis. The study area was expanded from 29 census tracts to the entire city of Indianapolis (396 square miles) in order to enroll new participants. The expansion of the study area made door to door recruitment impossible, so the initial contacts were two consecutive letters followed by a telephone call. There were a total of 7,583 names on the list. Interviewers were able to contact by telephone or home visit 4,260, of whom 1,892 (44%) were enrolled, 1,999 (47%) refused, and 369 (9%) were too ill. Determination of race was by self report. Nursing home residents were not included in these samples. Informed consent was obtained from study participants prior to each interview. The study was approved by the Institutional Review Board at Indiana University School of Medicine.

2.2 Research Design

The study follows a two-stage study design. In the first stage all study participants have an in-home interview using the Community Screening Interview for Dementia (CSI-D). The second stage is a full diagnostic clinical assessment in a subgroup of participants selected from the larger cohort. Scores on the CSI-D are divided into performance groups (good, intermediate, and poor). The scores for the groups were initially based on our pilot study and confirmed in subsequent waves of the study(10). All of the individuals in the poor performance group were invited to have the clinical assessment in 1992 and 2001. In 1992, subjects were randomly sampled from the intermediate group until 50% had clinical assessment, and from the good performance group, 5% were sampled (weighted for 75% age ≥ 75 years). In 2001, because the cohort was five years older, with the youngest participants age 70 years, we increased the sampling proportion in the intermediate performance group to 75% for optimum sensitivity, and we reduced the proportion for the good performance group to 2% (weighted for 75% age ≥ 75). For those in the good or intermediate groups who refuse or are not able to participate, a replacement is sampled from the same performance group.

2.3 Screening Phase

The CSI-D was developed by our group for comparative epidemiological studies of age-associated dementias in different populations. The CSI-D has good sensitivity and specificity for discriminating dementia from no dementia in community dwelling populations, and inter-rater reliability as well as a two-week test retest reliability are good(10). The cognitive assessment has items to test the following domains: language, attention and calculation, memory, orientation, praxis, and comprehension and motor response. The CSI-D also includes a standardized interview with a close relative about cognitive function and activities of daily living. The CSI-D performance groups are determined by combining the cognitive score and the informant interview score in a discriminant score(10). For cases in which there is not an informant, cognitive scores alone are used to determine performance group.

2.4 Other Assessments

The CSI-D also collects information about medical history from the subject and the informant. Current medications are recorded by inspecting containers. There is a risk factor inventory that includes use of tobacco and alcohol, as well as social involvement measures. Some simple neurological tests are also given.

2.5 Clinical Assessment

Clinical assessments are conducted in a home visit by a physician and psychometrician. The assessment includes the following: (1) Consortium to Establish a Registry for Alzheimer's Disease (CERAD) neuropsychological test battery using normative values by education level, developed by our group for elderly African Americans(11); (2) standardized health examination and assessment of function; (3) semi-structured informant interview based upon the Cambridge Examination for Mental Disorders of the Elderly (CAMDEX) adapted for American populations(12); (4) request for medical records including neuroimaging.

2.6 Blood Samples for DNA

Blood samples were collected in 10 ml (EDTA) Vacutainer tubes. The samples were centrifuged and red blood cells, buffy coat, and plasma were separated. DNA was extracted from the buffy coat using standard protocols. HhaI digestion of amplified products was used to determine *APOE* genotype(13). Informed consent was obtained prior to blood collection.

2.7 Diagnosis

All participants are diagnosed into one of three mutually exclusive major cognitive diagnostic categories: dementia, normal, or cognitively impaired but no dementia (CIND). Diagnosis is made in a consensus diagnostic conference of clinicians reviewing the CERAD neuropsychological test battery, the physician's assessment, the informant interview, and available medical records. Clinicians are kept blind regarding CSI-D scores and screening performance group. For the diagnosis of dementia criteria of both the DSM-III-R(14) and ICD-10(15) must be met. The diagnosis of probable and possible Alzheimer's disease is made using the criteria proposed by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS/ADRDA)(16). We use ICD-10 criteria for Vascular Dementia and other secondary dementias (e.g., Alcohol Dementia, Parkinson's dementia etc). Unspecified dementia is diagnosed when the diagnostic criteria for dementia are satisfied but the subject cannot be assigned to a specific dementing disorder. Severity of dementia was recorded using the Clinical Dementia Rating (CDR) system(17).

2.8 Statistical Analysis

T-tests and chi-square tests were used to compare the cohorts on continuous and categorical variables, respectively. In addition, they were also used to compare CSI-D poor performance group subjects with and without clinical assessment from each cohort. Chi-square tests were used to examine the association between history of depression and other comorbid conditions. Prevalence rates of dementia are estimated for the following age groups: 70 – 74, 75 – 79, 80 – 84, and ≥ 85 years. The age specific prevalence rates of dementia were estimated using smoothing via logistic regression in which the probability of dementia was modeled as a function of age, gender, and CSI-D performance group and predicted probability from the model was used in prevalence estimates(18). Weighted logistic regression was used to account for different probabilities of selection into the sample for clinical assessment in different performance groups. Standard errors for age-specific prevalence rates were approximated using variance-covariance estimated from the weighted logistic model. The 95% confidence intervals for the prevalence rates were constructed on the basis of asymptotic normality. For

deriving overall standardized rates, 1990 U.S. census data for African Americans in Marion County were used which estimated 38% were between age 70–74, 30% between 75–79, 17% between 80–84, and 15% were 85 years or older. We compared the 1992 prevalence rates to those of 2001 using weighted logistic models with an indicator variable for cohorts. Dementia severity rating scales using the CDR were compared for the two cohorts using Fisher's exact test.

3. Results

Table 1 shows the demographic characteristics, medical history, medication use, and *APOE* e4 status for the 1992 cohort compared to the 2001 cohort. The 1992 cohort is slightly older. The gender distribution is about the same for both cohorts with 65% women. The 2001 cohort has a higher level of education. Fewer of the 2001 cohort lived in rural areas from birth to age 19 compared to the 1992 group. More of the 2001 cohort reported ever having used alcohol regularly and the 1992 cohort had more who reported ever having used tobacco regularly. Using either self report or report by the informant, the 2001 cohort reported higher rates of several medical conditions including cancer, hypertension, diabetes, stroke and history of depression. History of depression was significantly associated with increased rates of cancer, heart attack, hypertension, diabetes and stroke ($p < 0.05$). Use of antihypertensive and diabetic medications was also higher in the 2001 cohort. Statin medications were used by almost a quarter (24.11%) of the 2001 cohort, compared to only 1.64% in 1992. The *APOE* e4 allele is associated with risk for AD in this population(19) and occurs in about the same frequency in the two cohorts. Table 2 shows numbers for each of the cohorts by performance groups on the CSI-D by age group, showing diagnosis of dementia and Alzheimer's disease within the groups. The intermediate group of the 2001 cohort had more cases of dementia but the proportions of demented subjects in the intermediate groups were not significantly different ($p = 0.1177$) between the cohorts. Mean cognitive scores were not significantly different ($p = 0.5443$) nor were the mean informant scores ($p = 0.7083$) in the intermediate groups. For both cohorts, the poor performance group had the most cases of dementia. Age specific prevalence rates for dementia for each cohort are shown in Table 3.

The prevalence rate estimates in the older age group for the 2001 cohort are slightly higher but the confidence intervals are overlapping. Table 4 shows age specific prevalence rates for possible or probable Alzheimer's disease for each cohort. Results of the weighted logistic models comparing prevalence rates for dementia and AD for the two cohorts showed no significant differences, [(dementia, $p = 0.3534$)(AD, $p = 0.2649$)]. Other subtypes of dementia for the two cohorts included vascular dementia, with 10 in the 1992 cohort, and 15 in the 2001 cohort, Parkinson's with one in 1992 and one in 2001, other neurological disorders with two in 1992 and two in 2001, alcohol related dementia with two in 1992 and none in 2001, dementia with depression with one in 1992 but none in 2001, and one non specific dementia in 2001. Table 5 shows the CDR ratings comparing the 1992 and 2001 cohorts, one subject in the 1992 cohort had insufficient information for a CDR score. Using the Fisher's exact test, differences between the two cohorts were not significant ($p = 0.0589$).

For the 2001 cohort, comparisons were made for those who enrolled and those who did not using information from the Medicare rolls. Those who were not enrolled were significantly older, mean age = 79.06, SD = 7.42, compared to those who did enroll mean age = 76.81, SD=5.58; $p < 0.0001$. A higher proportion of women enrolled (64.75%) compared to the not enrolled group (59.63%)($p = 0.0006$). For the clinical assessment phase the study participants in the CSI-D poor performance group who were not clinically assessed in 1992 (N=88) did not differ significantly from those who were assessed in gender, age, and cognitive and informant scores. For the 2001 cohort, there were 47 in the poor performance group who were not assessed, and 124 who did have the assessment. There were no significant differences in gender,

age or informant score between the assessed and not assessed. The cognitive scores were slightly lower for those who did not have the assessment, mean 24.4 with SD 5.54, compared to those who were assessed, mean 26.36 with SD 3.66 ($p=0.018$).

4. Discussion

Comparing rates for African Americans living in Indianapolis in 1992 and 2001 we found no differences in prevalence rates of dementia, or AD. These prevalence rates, as is also the case for incidence rates, are somewhat lower than other reported studies of African Americans but not significantly so(20). The Rochester Epidemiology Project compared prevalence rates for Rochester, Minnesota in 1975, 1980 and 1985. There was no change between 1975 and 1980, but there was an increase in rates in 1985. They noted that actual changes in incidence rates could account for this but also suggested that the terms dementia and Alzheimer's disease had become better understood by physicians during these time periods and thus, more readily identified(8). Their study was carried out through the medical records linkage system of the Rochester Epidemiology Project. A report comparing disability prevalence due to severe cognitive impairment in Medicare data for 1982 and 1999 found a decline in mixed dementia but not in AD alone(6). The methodologies in these reports differed from those of our study. In our study the diagnoses of dementia were based upon direct clinical assessments in population-based samples.

The individuals who agreed to participate in 2001 have higher education than those of the 1992 cohort, which many studies, including ours, report reduces risk. It has been suggested that levels of literacy are better indicators of education than number of years of school(21), however this study did not administer tests of levels of literacy. For those with fewer years of education, we previously reported six years of education or less, combined with rural childhood residence increased risk for Alzheimer's disease(22). Fewer of the 2001 cohort reported childhood rural residence.

Our 2001 cohort reports higher rates of hypertension, diabetes, and stroke, conditions associated with risk for dementia and AD, but they also report higher rates of treatment. These differences are consistent with national trends for African Americans over this time period (23). Self reported history of depression, which is also associated with increased risk for dementia, was higher in the 2001 cohort than in the 1992 cohort. This may reflect a trend for elderly Americans (both African Americans and Whites) to report more visits to mental health professionals over this time period(24–26).

We do not have reliable data on rates of hypercholesterolemia but the use of statins has dramatically increased over this time (24.15% in 2001, and 1.13% in 1992). Thus, as described before(7), there are two conflicting trends with regard to cardiovascular risk factors, incidence rates have gone up but treatment rates have also increased. The net effects of these trends are difficult to predict particularly in diseases like AD which is likely to have a long gestational period. The *APOE* e4 allele is associated with risk for AD in this population and occurs in about the same frequency in the two cohorts and would not be expected to differentially impact prevalence rates.

We looked at severity ratings of the diagnosed cases of dementia and found no significant differences. There was an increase in the number of nursing homes in Marion County over the duration of the study, however the percentage of African Americans residing in nursing homes remained constant at 6% at both time periods(27,28). In spite of the higher frequency of the risk factors, and the current treatments, we did not find differences in rates of dementia and AD. It may be that the increases in treatment rates of cardiovascular risk factors, and possibly depression may eventually influence rates of dementia and AD, but perhaps it will take longer

exposure for the effects to be measured. It has been suggested for example that treatment for hypertension may only show an effect on incidence of AD if it is initiated in middle age(29).

Limitations of the study include the change in recruitment strategies between 1992 and 2001 which resulted in higher refusal rates in 2001. The effect of the increase in refusal rates on prevalence estimates is uncertain. The refusers were significantly older than those who enrolled, which could result in missing cases, leading to under estimation of rates. There was a higher proportion of women in the enrolled group, however female gender was not associated with higher risk(30). Rates of medical illnesses were based upon self report or informant report rather than by direct examination. This is a prevalence study, not an incidence study. Other factors may influence prevalence rates, such as length of survival with disease, and nursing home placement. If individuals with dementia were surviving longer in the community this would lead to increases in rates, while higher rates of institutionalization in nursing homes would lead to an under estimation. In this population-based study, however, the percentages of African Americans in nursing homes were the same in 1992 and 2001. Differential mortality for demented participants would lead to a decrease in prevalence estimates but there is no evidence for changes in mortality risk in our cohorts(31).

5. Conclusion

We find no differences in prevalence rates of dementia and AD between 1992 and 2001 in spite of significant demographic, medical history and medical treatment differences in these population-based cohorts of African American elderly. We are currently completing a new wave of screening and diagnoses which, when completed, will allow us to compare incidence rates between the two cohorts.

Acknowledgments

National Institute on Aging RO1 AG 009956-17. The authors thank the Indianapolis study participants who have contributed their time and support to this project over the years.

Funding Support: This study was supported by a grant from the National Institute on Aging RO1AG009956.

Role of the Sponsors: The funding source for this study played no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, or approval of the manuscript.

We thank the elderly Indianapolis participants for their continued involvement with this study. We thank Millicent Pettaway, R.N. for nursing supervision and data collection, and Jenna York for manuscript preparation.

References

1. Fratiglioni L, Paillard-Borg S, Winblad B. An active and socially integrated lifestyle in late life might protect against dementia. *Lancet Neurol* 2004;3:343–353. [PubMed: 15157849]
2. Jedrzejewski, MK.; Lee, VM-Y.; Trojanowski John, Q. Lowering the risk of Alzheimer's disease: Evidence-based practices emerge from new research; *Alzheimer's and Dementia*. 2005. p. 152-160.<http://www.sciencedirect.com/science/article/B7W6D-4HTKDPN-W/1/eeeb446a1bb563ccdf4556a3b0722fa6>
3. Grodstein, F. Cardiovascular risk factors and cognitive function; *Alzheimer's and Dementia*. 2007. p. S16-S22.<http://www.sciencedirect.com/science/article/B7W6D-4NGKPNH-9/1/7f1bb8bb5463f78e9051758b671ee375>
4. Centers for Disease Control and Prevention. National Center for Health Statistics. Trends in Health and Aging, Mortality, Deaths by Underlying and Multiple Cause, Age, Sex, and Race. [Accessed June 6, 2008]. Table MMR104a. Available at: <http://209.217.72.34/aging/TableViewer/tableView.aspx?ReportId=410>

5. Centers for Disease Control and Prevention. National Center for Health Statistics. Trends in Health and Aging, Chronic Conditions Tables, Diabetes. [Accessed June 3, 2008]. Table NHAD04a. Available at: <http://209.217.72.34/aging/TableViewer/tableView.aspx?ReportId=418>
6. Manton KC, Gu XL, Ukraintseva SV. Declining prevalence of dementia in the U.S. elderly population. *Adv Gerontol* 2005;16:30–37. [PubMed: 16075674]
7. 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? *Alzheimer's & Dementia*. 2008(In press)
8. Beard CM, Kokmen E, O'Brien PC, Kurland LT. The prevalence of dementia is changing over time in Rochester, Minnesota. *Neurology* 1995;45:75–79. [PubMed: 7824140]
9. Hendrie HC, Osuntokun BO, Hall KS, et al. Prevalence of Alzheimer's disease and dementia in two communities: Nigerian Africans and African Americans. *Am J Psychiatry* 1995;152:1485–1492. [PubMed: 7573588]
10. Hall KS, Ogunniyi AO, Hendrie HC, et al. A cross-cultural community based study of dementias: Methods and performance of the survey instrument Indianapolis, USA, and Ibadan, Nigeria. *Int J Meth Psychiatr Res* 1996;6:129–142.
11. Unverzagt FW, Hall KS, Torke AM, et al. Effects of age, education, and gender on CERAD neuropsychological test performance in an African American sample. *Clinical Neuropsychologist* 1996;10:180–190.
12. Hendrie HC, Hall KS, Brittain HM, et al. The CAMDEX: a standardized instrument for the diagnosis of mental disorder in the elderly: a replication with a US sample. *J Am Geriatr Soc* 1988;36:402–408. [PubMed: 3361042]
13. Hixson JE, Vernier DT. Restriction isotyping of human apolipoprotein E by gene amplification and cleavage with HhaI. *J Lipid Res* 1990;31:545–548. [PubMed: 2341813]
14. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. Vol. 3. Washington, DC: American Psychiatric Association; 1987.
15. American Psychiatric Association Press. *ICD-10. The International Statistical Classification of Diseases and Related Health Problems: 1 and 2*. 1992. Report No.: V. 3
16. McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology* 1984;34:939–944. [PubMed: 6610841]
17. CDR Dementia Rating. [Accessed July 1, 2006]. Available at: <http://alzheimer.wustl.edu/cdr/AboutCDR/aboutcdr.htm>
18. Beckett LA, Scherr PA, Evans DA. Population prevalence estimates from complex samples. *J Clin Epidemiol* 1992;45:393–402. [PubMed: 1569435]
19. Murrell JR, Price B, Lane KA, et al. Association of apolipoprotein E genotype and Alzheimer disease in African Americans. *Arch Neurol* 2006;63:431–434. [PubMed: 16533971]
20. Hendrie HC, Murrell J, Gao S, Unverzagt FW, Ogunniyi A, Hall KS. International studies in dementia with particular emphasis on populations of African origin. *Alzheimer Dis Assoc Disord* 2006;20:S42–S46. [PubMed: 16917194]
21. Manly JJ, Schupf N, Tang MX, Stern Y. Cognitive decline and literacy among ethnically diverse elders. *J Geriatr Psychiatry Neurol* 2005;18:213–217. [PubMed: 16306242]
22. Hall KS, Gao S, Unverzagt FW, Hendrie HC. Low education and childhood rural residence: risk for Alzheimer's disease in African Americans. *Neurology* 2000;54:95–99. [PubMed: 10636132]
23. Centers for Disease Control and Prevention. National Center for Health Statistics. Trends in Health and Aging, Use and Costs of Prescription Medication Tables, Visits to Office-Based Physicians: Distribution of Drug Mentions According to Therapeutic Classification, by Patient's Age, Sex, and Race. [Accessed June 3, 2008]. Table NAD04c. Available at: <http://209.217.72.34/aging/TableViewer/tableView.aspx?ReportId=376>
24. Centers for Disease Control and Prevention. National Center for Health Statistics. Trends in Health and Aging, Indicators of Mental Health by Age, Sex, and Race/Ethnicity. Visited a mental health professional: All races. [Accessed November 24, 2008]. Available at: <http://205.207.175.93/aging/TableViewer/tableView.aspx>

25. Centers for Disease Control and Prevention. National Center for Health Statistics. Trends in Health and Aging, Indicators of Mental Health by Age, Sex, and Race/Ethnicity. Visited a mental health professional: Non-Hispanic Black. [Accessed November 24, 2008]. Available at: <http://205.207.175.93/aging/TableView/tableView.aspx>
26. Centers for Disease Control and Prevention. National Center for Health Statistics. Trends in Health and Aging, Indicators of Mental Health by Age, Sex, and Race/Ethnicity. Visited a mental health professional: Non-Hispanic White. [Accessed November 24, 2008]. Available at: <http://205.207.175.93/aging/TableView/tableView.aspx>
27. Marion County Health Department II. Annual Reports by Nursing Homes. 1989
28. Census 2000. Population by group quarters who are Black or African American alone. [Accessed June 6, 2008]. Table PCT17B. Available at: http://factfinder.census.gov/servlet/DTSUBJECTSHOWTABLES?_ts=231335257608
29. Qiu C, Winblad B, Fratiglioni L. The age-dependent relation of blood pressure to cognitive function and dementia. *Lancet Neurol* 2005;4:487–499. [PubMed: 16033691]
30. Hall K, Gureje O, Gao S, et al. Risk factors and Alzheimer's disease: a comparative study of two communities. *Aust N Z J Psychiatry* 1998;32:698–706. [PubMed: 9805594]
31. Lane KA, Gao S, Hui SL, Murrell JR, Hall KS, Hendrie HC. Apolipoprotein E and mortality in African-Americans and Yoruba. *J Alzheimers Dis* 2003;5:383–390. [PubMed: 14646029]

Table 1

Comparisons of demographics, medical history, and APOE genotype between the 1992 and 2001 cohorts

Variables	1992 (n=1500)	2001 (n=1892)	p-value
Mean age, SD	77.39 (5.98)	76.81 (5.58)	0.0037
Mean years of education, SD	9.30 (3.18)	11.32 (2.74)	<0.0001
Female, %	65.33	64.75	0.7218
Rural childhood residency, %	34.23	24.81	<0.0001
Ever used alcohol regularly, %	30.62	36.07	0.0012
Ever smoked, %	59.54	54.58	0.0039
Self or Informant-reported history of			
Cancer, %	11.94	16.69	0.0001
Parkinson's disease, %	1.13	0.95	0.6055
Heart attack, %	15.55	13.99	0.2019
Hypertension, %	64.67	75.24	<0.0001
Diabetes, %	24.40	29.30	0.0015
Stroke, %	12.28	15.83	0.0034
Head injury, %	8.68	8.87	0.8433
Depression, %	6.87	11.05	<0.0001
Medication Usage			
Antihypertensive	44.20	76.00	<0.0001
Antidiabetic	14.40	21.55	<0.0001
Statins	1.64	24.11	<0.0001
Genetic testing	(n=456)	(n=1133)	
Has ApoE 4 allele	39.91	36.19	0.1647

Table 2

Numbers of subjects in each performance group

Age Group	Good Performance		Intermediate Performance		Poor Performance	
	Total	Dementia (AD)	Total	Dementia (AD)	Total	Dementia (AD)
<u>1992</u>						
70–74	499	0 (0)	39	0 (0)	54	6 (3)
75–79	328	0 (0)	35	2 (1)	54	10 (6)
80–85	195	1 (1)	35	3 (2)	59	22 (20)
85+	127	0 (0)	21	0 (0)	54	18 (15)
Total	1149	1 (1)	130	5 (3)	221	56 (44)
<u>2001</u>						
70–74	699	0 (0)	42	3 (2)	37	9 (5)
75–79	503	0 (0)	39	3 (3)	50	9 (7)
80–84	248	0 (0)	19	2 (2)	41	13 (11)
85+	156	1 (1)	15	3 (3)	41	14 (14)
Total	1606	1 (1)	115	11 (10)	171	45 (37)

Table 3

Age specific prevalence rates and overall standardized rates for dementia

Age group	1992		2001	
	% with dementia	95% Confidence Interval	% with dementia	95% Confidence Interval
70–74	2.50	1.64–3.37	1.90	0.86–2.94
75–79	4.86	3.51–6.21	4.68	2.16–7.19
80–85	9.68	7.28–12.07	10.26	3.64–16.87
85+	17.48	12.75–22.20	23.17	4.72–41.62
Overall standardized rate*	6.75	5.77–7.74	7.45	4.27–10.64

* for subjects age ≥ 70 using 1990 US census data for African Americans in Marion County, Indiana.

Table 4
Age specific prevalence rates and overall standardized rates for AD

Age group	1992		2001	
	% with AD	95% Confidence Interval	% with AD	95% Confidence Interval
70–74	1.68	0.93–2.42	1.34	0.44–2.24
75–79	3.53	2.27–4.79	3.97	1.56–6.39
80–85	7.80	5.52–10.09	9.36	2.35–16.37
85+	15.79	11.03–20.56	22.48	4.56–40.40
Overall standardized rate*	5.47	4.51–6.42	6.77	3.65–9.90

* for subjects age ≥ 70 using 1990 US census data for African Americans in Marion County, Indiana.

Table 5

CDR ratings

Dementia Scale Ratings	1992 (n=61)[*]	2001 (n=57)
With Rating of 1 or less	41 (67.2%)	38 (66.7%)
With Rating of 2	7 (11.5%)	14 (24.6%)
With Rating of 3 or more	13 (21.3%)	5 (8.8%)

* One demented subject is missing CDR rating.