Incidence of Dementia and Cognitive Impairment, Not Dementia in the United States

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Objective: Estimates of incident dementia, and cognitive impairment, not dementia (CIND) (or the related mild cognitive impairment) are important for public health and clinical care policy. In this paper, we report US national incidence rates for dementia and CIND.

Methods: Participants in the Aging, Demographic, and Memory Study (ADAMS) were evaluated for cognitive impairment using a comprehensive in-home assessment. A total of 456 individuals aged 72 years and older, who were not demented at baseline, were followed longitudinally from August 2001 to December 2009. An expert consensus panel assigned a diagnosis of normal cognition, CIND, or dementia and its subtypes. Using a population-weighted sample, we estimated the incidence of dementia, Alzheimer disease (AD), vascular dementia (VaD), and CIND by age. We also estimated the incidence of progression from CIND to dementia.

Results: The incidence of dementia was 33.3 (standard error [SE], 4.2) per 1,000 person-years and 22.9 (SE, 2.9) per 1,000 person-years for AD. The incidence of CIND was 60.4 (SE, 7.2) cases per 1,000 person-years. An estimated 120.3 (SE, 16.9) individuals per 1,000 person-years progressed from CIND to dementia. Over a 5.9-year period, about 3.4 million individuals aged 72 and older in the United States developed incident dementia, of whom approximately 2.3 million developed AD, and about 637,000 developed VaD. Over this same period, almost 4.8 million individuals developed incident CIND.

Interpretation: The incidence of CIND is greater than the incidence of dementia, and those with CIND are at high risk of progressing to dementia, making CIND a potentially valuable target for treatments aimed at slowing cognitive decline.

The rapid growth in the number of older adults in the United States puts an increasing number of individuals at risk for cognitive impairment and dementia. As a result of this demographic shift in the population, the prevalence of Alzheimer disease (AD) is estimated to grow to nearly 9 million individuals in North America by 2050.[1] Previous estimates of the incidence of dementia and AD in the United States for individuals aged 65 years and older primarily have been extrapolated from data on local or regional samples. These estimates range from 20 to 43 cases per 1,000 person-years for dementia[2–6] and from 14 to 27 cases per 1,000 person-years for AD.[2–3,5,7,8]

Fewer estimates are available for the incidence of milder impairment that does not reach the severity of

dementia, often termed mild cognitive impairment (MCI) or cognitive impairment, not dementia (CIND). The criteria for CIND and MCI have grown more similar over time,[9] with the 1 remaining difference being that CIND requires either complaint of a problem or impaired test performance,[10] and MCI requires both complaint of a problem and impaired test performance.[11] This difference in criteria for CIND and MCI does not appear to account for the broad variability in estimated incidence rates for mild impairment,[12] which range from 22 to 51 cases per 1,000 person-years.[13–15] Although incidence estimates for MCI and CIND vary, there is general agreement that individuals with cognitive impairment are at higher risk of progressing to dementia compared to older individuals with normal cognition.

Estimates of the incidence of CIND and dementia are essential to provide benchmarks against which to assess trends in incidence rates over time and to assess the effect of preventative measures devoid of survival effects that can bias prevalence estimates. Both the large numbers of individuals affected with CIND or dementia and the conditions' broad impact on the individual's health, family resources, and healthcare mean that even small benefits from future interventions may have a significant impact on disease burden in the United States.

The Aging, Demographics, and Memory Study (ADAMS) uses a national probability sample that includes individuals from all regions of the United States and provides a unique resource to estimate incidence rates for dementia, AD, and CIND. As a longitudinal study, it is also able to assess rates of progression from CIND to dementia and better identify the full implications of cognitive impairment for public health and clinical care policy.

Subjects and Methods

Sample

The ADAMS sample was drawn from the larger Health and Retirement Study (HRS), an ongoing national probability cohort study of individuals born prior to 1960, designed to investigate the health, social, and economic implications of the aging of the American population.[16–18] The HRS began in 1992, and the current sample includes approximately 22,000 individuals.

The ADAMS began with a stratified random subsample of 1,770 individuals from the approximately 7,000 HRS respondents aged ≥70 years old who completed the 2000 or 2002 wave of HRS. Participants in the full ADAMS sample lived in 42 states distributed throughout all census regions of the United States. To achieve a sufficient number of ADAMS respondents across the full range of cognitive ability, the sample was stratified based on cognitive screening scores on the HRS. Respondents were each classified into 1 of 5 cognitive strata, ranging from low to high cognition based on their HRS cogni-

tive screening scores. For respondents who completed the HRS interview themselves, the score from an abbreviated version of the modified Telephone Interview for Cognitive Status[19] was used to assign them to a stratum. For respondents unable to complete the interview themselves, a proxy for the respondent completed the Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE),[20] which assessed changes in the respondent's cognitive and functional ability. These respondents were classified based on the IQCODE scores. The highest 3 cognitive strata were further stratified by age (70–79 years vs ≥80 years) and sex, to ensure adequate numbers in each subgroup. Additional details of the ADAMS sample design are described elsewhere.[21,22]

The Figure summarizes the number of participants at each phase of the study; additional details on participation rates have been reported previously.[22] The initial ADAMS assessments (Wave A) were conducted from 2001 to 2003 and occurred on average 13.3 (standard deviation [SD], 6.9) months after the HRS interview. Three waves of follow-up assessments have been conducted on this sample. For the first follow-up wave (Wave B), a subset of ADAMS participants (see Fig) was selected to undergo an assessment approximately 16 to 18 months after the initial assessment if: (1) they received an initial diagnosis of cognitive impairment without dementia; or (2) they received an initial diagnosis of normal or demented, but the consensus diagnostic panel thought the initial assessment findings were ambiguous and longitudinal follow-up would help clarify the diagnosis. A second follow-up (Wave C) was done beginning in 2006 among the living participants not previously diagnosed with dementia in the ADAMS. The Wave C assessment took place on average 3.69 (range, 1.25–6.00) years after the participant's prior ADAMS assessment. Finally, beginning in 2008, the third follow-up (Wave D) was conducted an average of 1.8 years (range, 1.25–6.50) after the most recent assessment. The assessment protocols for the initial and follow-up assessments were similar, with only minor modifications in the assessment measures. The sample for the present analyses includes 456 individuals, spanning 36 states, who participated in the first wave (Wave A) of ADAMS data collection and who were not diagnosed with dementia at that wave.

All ADAMS data are publicly available and can be obtained from the HRS website.[23] The institutional review boards at Duke University Medical Center and the University of Michigan approved all study procedures, and study participants or their surrogates provided informed consent.

Dementia Assessment and Diagnosis

All participants were assessed for cognitive impairment in-person in their residence by a nurse and neuropsychology technician. The full details of these assessment and diagnostic procedures have been previously described.[22] Briefly, an informant (usually a spouse or adult child) was identified by the respondent as someone who was knowledgeable about the respondent's daily activities and health issues. The following information about the respondent was collected from the informant: (1) a detailed chronological history of cognitive and functional
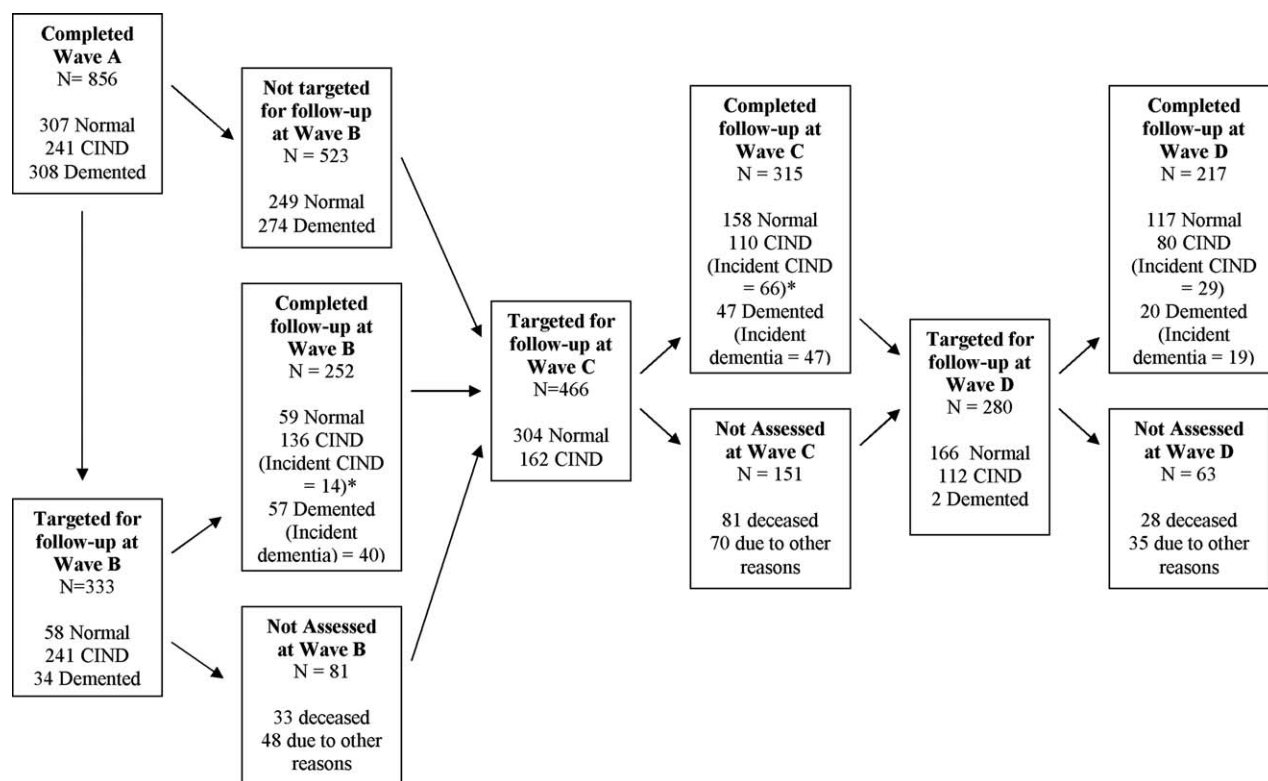
FIGURE: Flowchart of number of participants at each phase of the study. *Includes 16 incident cognitive impairment, not dementia (CIND) cases excluded from incidence analysis because of nonresponse (part of the cases not assessed at Wave C and/or Wave D "due to other reasons").

symptoms, (2) medical history, (3) current medications, (4) current neuropsychiatric symptoms, (5) measures of severity of cognitive and functional impairment, and (6) family history of memory problems. During the assessment, each respondent completed: (1) a battery of neuropsychological measures, (2) a self-report depression measure, (3) a standardized neurological examination, (4) a blood pressure measure, (5) collection of buccal DNA sample for *APOE* genotyping, and (6) a 7-minute videotaped segment covering portions of the cognitive status and neurological examinations. The neuropsychological battery has been described elsewhere,[22] and included measures of orientation, verbal and visual immediate and delayed memory, language, attention, executive function, praxis, and reading ability. Medical record releases were also sought to obtain relevant prior neuroimaging and laboratory results from participants' physicians.

Final diagnoses were assigned by a consensus expert panel made up of neuropsychologists, a cognitive neuroscientist, neurologists, geropsychiatrists, and internists. The consensus panel reviewed all information collected during the in-home assessment and assigned a diagnosis in 2 stages, first without the medical records and then with the medical records. As part of the consensus process, the panel compared performance on the neuropsychological tests from all assessments, but the consensus panel was blind to the cognitive diagnosis assigned at the previous assessments. All individuals involved in the clinical assessments or diagnosis of ADAMS participants were blind to the participants' HRS cognitive screening scores.

Diagnoses fell within the 3 general categories: normal cognitive function, CIND, and dementia. The dementia diagnosis was anchored by the Diagnostic and Statistical Manual of Mental Disorders (DSM)-III-R[24] and DSM-IV[25] criteria, but the final diagnosis was based on the clinical judgment of the consensus panel. Currently accepted diagnostic criteria were used for AD[26] and vascular dementia (VaD), as adapted for research studies.[27] These assessment and diagnostic procedures for AD have been validated against neuropathological diagnoses.[28]

The criteria for CIND are less well-established than the criteria for dementia. In the ADAMS, the diagnosis of CIND was determined by clinical judgment, but was anchored in the following guidelines: cognitively not normal but not demented; self and/or informant report of problems with cognition or daily activities; or performance on neuropsychological measures that was both below expectation relative to education, reading level, and occupational attainment, and at least 1.5 SDs below published norms on at least 1 test. These CIND criteria are similar to those currently used for MCI,[11] except that the latter require both self and/or informant complaint and impairment on neuropsychological measures.

To reflect that dementia and CIND are often the consequence of >1 pathological process, when appropriate we assigned a primary and secondary diagnosis denoting these multiple etiologies. Dementia onset was estimated as the age at which the individual met criteria for dementia based on a systematic review of the chronological history of cognitive and functional changes.

Analyses

Population sample weights were constructed to take into account the probabilities of selection in the stratified longitudinal sample design and to adjust for differential participation in ADAMS at each assessment wave. Details of the development of these weights are described in a technical report elsewhere.[21] We compared sample characteristics by final cognitive status using the Wald chi-square test for categorical measures and the *t* test for continuous measures. Using the ADAMS sample weights, we estimated the national incidence of CIND and dementia (as well as the dementia subtypes AD and VaD) as the number of individuals affected per 1,000 person-years by 8- or 10-year age at follow-up categories.

To examine possible predictors of incident cognitive impairment reported in other studies, we used logistic regression to estimate the likelihood of dementia, AD, and CIND first as a function of age and then as a function of age and each of the following variables individually: education, gender, race, and *APOE* genotype. We then created models with all of the variables included. The results for each of the variables in the full models were similar to those for the reduced models, so we report only the full models in the Results section. For the estimation of incidence rates, person-years were calculated for each case as the number of years from the baseline assessment until the first of any of the following events: (1) the event of interest (dementia onset, for example), (2) death, or (3) completion of the final follow-up assessment (Wave D).

All analyses were conducted in SAS version 9.2 (SAS Institute, Cary, NC) using procedures that account for the influence of the weighting and other complex sample design features.

Results

The Figure shows the number of individuals in each outcome category for each phase of the study. Table 1 provides sample characteristics of individuals based on cognitive status at follow-up. Individuals who did not participate at Wave C or Wave D (n = 92) due to illness, refusal, or being unable to be located did not differ from participants on baseline age, years of education, *APOE* e4 allele status, or mean Mini-Mental State Examination score; but they did differ on ethnicity, in that Hispanics made up 11.6% of nonparticipants but only 4.1% of participants. Participants were followed an average of 4.89 (SD, 1.76) years. The mean estimated age of onset for incident dementia was 83.72 years (SD, 5.49), and the estimated mean age at first diagnosis for those with incident CIND was 82.83 years (SD, 5.35).

Table 2 shows rates of incident CIND, AD, and dementia by age at follow-up. The incidence of dementia increased progressively with age, reaching 82 cases per 1,000 person-years for individuals aged >90 years. AD incidence also increased with age and accounted for >70% of the dementia cases overall. The incidence of VaD (n = 16) overall was 6.2 (95% confidence interval [CI], 1.7–10.7) cases per 1,000 person-years. VaD incidence increased from 2.1 (95% CI, 0.0–4.9) cases per 1,000 person-years for those aged 72 to 79 years to 11.4 (95% CI, 1.9–20.9) for those aged 80 to 89 years; however, there were no additional cases of vascular dementia for those aged 90 and older. Overall, we found that an estimated 3.4 million individuals aged 72 and older in the United States developed incident dementia over a follow-up period of 5.9 years, of whom approximately 2.3 million developed AD and 636,825 developed VaD.

Table 2 also shows that the incidence of CIND progressively increased with age, and at each age stratum the incidence of CIND was higher than the incidence of dementia. When limiting the analyses just to those with CIND, the incidence rate at which individuals with CIND progressed to dementia was 120.3 cases per 1,000 person-years. In contrast, the incidence rate of conversion to dementia among those with normal cognition was 17.1 cases per 1,000 person-years, a rate 7× lower than among the CIND group. Even so, it is important to note that among the 214 individuals with CIND at baseline who were followed longitudinally, 110 retained their CIND designation throughout the period that they were followed, which suggests that progression to dementia may not be inevitable among individuals in this group. Twenty-five individuals (11.7%) with CIND at baseline reverted to normal cognition at a subsequent follow-up assessment, but 15 of these 25 then later reverted back to CIND, and 5 individuals progressed to dementia after further longitudinal follow-up. Over an average follow-up period of 4.8 years, an estimated 4.8 million individuals aged 72 and older in the United States developed incident CIND. Based on these national estimates, individuals who progress from CIND to dementia account for 75% of all incident cases of dementia.

Table 3 shows the results of the full logistic models predicting dementia, AD, and CIND. Older age and fewer years of education tended to be associated with a higher risk of AD and dementia among all participants; the odds ratios for the 3 cognitive outcomes were similar, but only some of the values reached standard significance levels. The presence of at least 1 *APOE* e4 allele was associated with increased risk of incident dementia, AD, and CIND. There was no significant association between race and the incidence of CIND, dementia or AD among all participants, whereas being male was associated with decreased risk of AD.

When limiting the group to those with CIND, males had a lower risk of progressing to dementia than

TABLE 1: Characteristics of the ADAMS Sample, by Diagnostic Category at Follow-up Assessment

Characteristic	Final Cognitive Status				Group Significant Differences, p < 0.05
	(1) Cognitively Normal	(2) CIND at Baseline and Follow-up	(3) Incident Dementia	(4) Incident CIND	
Total participants, No.	158	110	106	82	
Mean baseline age (SD), yr	77.67 (4.90)	81.80 (6.57)	80.20 (5.47)	79.07 (56.4)	1-2, 1-3, 2-4
Men, No. (%)	96 (37.8)	78 (50.4)	41 (32.7)	45 (49.8)	1-2, 2-3, 3-4
Mean education (SD), yr	12.95 (2.66)	10.78 (3.57)	10.80 (4.20)	11.54 (3.27)	1-2, 1-3, 1-4
Mean baseline MMSE (SD)	28.22 (1.99)	24.20 (3.53)	25.68 (3.81)	26.97 (2.76)	1-2, 1-3, 1-4, 2-3, 2-4, 3-4
Mean last MMSE (SD)	27.46 (2.19)	23.58 (4.13)	19.45 (4.99)[a]	24.77 (2.67)[a]	1-2, 1-3, 1-4, 2-3, 3-4
Any *APOE* e4 allele, No. (%)	39 (19.4)	35 (17.4)	26 (32.7)	25 (28.1)	None
Race/ethnicity, No. (%)					
Non-Hispanic white	174 (92.8)	95 (85.3)	67 (83.0)	59 (86)	1-2, 1-3, 1-4
Non-Hispanic black	19 (4.4)	30 (7.6)	25 (10.8)	18 (11.6)	1-3, 1-4
Hispanic	20 (2.9)	22 (7.1)	14 (6.1)	5 (2.4)	None

Incident CIND excludes 11 cases that progressed to dementia. These cases are included in the Incident Dementia column. Wave C nonparticipants were significantly different from all participants combined. Numbers are unweighted; means and percentages are weighted.

[a]MMSE score at time of diagnosis of CIND or dementia.

ADAMS = Aging, Demographics, and Memory Study; CIND = cognitive impairment, not dementia; SD = standard deviation; MMSE = Mini-Mental State Examination.

TABLE 2: Incidence Rates for AD, Dementia, and CIND

Incident Condition	Age Group, yr	ADAMS Sample Incident Cases, Unweighted	Estimate of Population Incident Cases	Population Person-Years	Estimate of Incidence/1,000 Person-Years (SE)	Incidence/1,000 Person-Years, 95% CI
Dementia	72–79	22	938,949	49,574,616	18.9 (4.3)	10.1–27.8
	80–89	58	1,965,362	46,528,062	42.2 (7.9)	26.0–58.5
	90+	26	503,009	6,127,925	82.1 (20.5)	39.9–124.3
	Total	106	3,407,320	102,230,604	33.3 (4.2)	24.7–42.0
AD	72–79	16	821,592	49,574,616	16.6 (4.2)	8.0–25.1
	80–89	39	1,127,323	46,528,062	24.2 (4.5)	15.0–33.4
	90+	22	392,057	6,127,925	64.0 (15.7)	31.8–96.2
	Total	77	2,340,972	102,230,604	22.9 (2.9)	17.0–28.8
CIND	72–79	38	1,653,503	42,034,445	39.3 (8.2)	22.5–56.2
	80–89	45	2,538,220	34,701,193	73.1 (11.8)	48.8–97.5
	90+	10	630,925	3,057,444	206.4 (56.1)	91.1–321.6
	Total	93	4,822,649	79,793,083	60.4 (7.2)	45.6–75.3
Dementia (among CIND)	72–79	19	741,835	7,253,899	102.3 (21.8)	57.5–147.0
	80–89	50	1,474,533	10,988,270	123.8 (26.5)	679.3–178.4
	90+	21	341,135	2,737,281	124.6 (35.6)	51.4–197.8
	Total	90	2,557,503	21,259,407	120.3 (16.9)	85.6–155.0

Total sample size for incident dementia and AD analyses = 456; total sample size for incident CIND analysis = 266; total sample size for incident dementia among CIND group = 268; total sample size for incident dementia from normal group = 242. Number of incident CIND is 11 more than in Table 1 because Table 2 numbers for incident CIND include the 11 individuals who developed incident CIND and then progressed to incident dementia.
AD = Alzheimer disease; CIND = cognitive impairment, not dementia; SE = standard error; CI = confidence interval.

females (odds ratio [OR], 0.59; 95% CI, 0.36–0.97) (see Table 3). The association between *APOE* e4 carriers and progression to dementia was significant (OR, 2.76; 95% CI, 1.27–5.97). Age, education, and race were not associated with progression from CIND to dementia.

Discussion

We estimate that about 3.4 million new cases of dementia occur during 6 years of follow-up for a US cohort that is aged 72+ years at baseline; approximately 2.3 million of these individuals have AD. Almost 4.8 million individuals are estimated to develop incident CIND during the same time period, which is approximately 40% more than the number of incident dementia cases of AD in this age group in the United States. These are the first estimates of the incidence of AD, dementia, and CIND

in a national probability US sample. Direct comparisons to other studies of dementia and AD incidence estimates are difficult due to differences in the age structure of the samples and the reporting of different age strata among the various studies. However, estimates of the incidence of dementia and AD from ADAMS are broadly within the midrange of estimates reported by other studies in the United States and Canada.[2–7,29] We note that the other studies used age 65 years as the lower limit; ADAMS incidence rates would likely have been lower if its age range were lowered from 72 to 65 years. Brookmeyer and colleagues[1] developed an equation for age-specific AD incidence rates based on a review of the worldwide literature. Applying this equation to US 2004 life tables,[30] the estimated incidence of AD for ages 72 to 79 years would be 8.5 cases per 1,000 person-years, 25.6 cases per 1,000 person-years for ages 80 to 89 years, and

TABLE 3: Regression Models Predicting AD, Dementia, and CIND

Characteristic	Dementia, OR (95%)	AD, OR (95%)	CIND, OR (95%)	Dementia from CIND, OR (95%)
Baseline age	1.05 (1.00–1.12)	1.04 (0.99–1.11)	1.06 (1.00–1.14)[a]	1.01 (0.95–1.07)
Years of schooling	0.89 (0.82–0.96)[a]	0.89 (0.81–0.98)[a]	0.86 (0.77–0.97)[a]	0.99 (0.91–1.08)
Gender				
Male	0.69 (0.39–1.21)	0.37 (0.15–0.94)[a]	1.64 (0.84–3.19)	0.59 (0.36–0.97)[a]
Female (ref)	1.00	1.00	1.00	1.00
Race				
Black	0.98 (0.50–1.95)	1.22 (0.58–2.57)	1.67 (0.78–3.57)	1.45 (0.59–3.56)
Nonblack (ref)	1.00	1.00	1.00	1.00
Any *APOE* E4 alleles				
Yes	2.20 (1.09–4.44)[a]	2.93 (1.68–5.13)[c]	2.65[b] (1.27–5.56)	2.76 (1.27–5.97)[a]
No	1.00	1.00	1.00	1.00

[a] $p \leq 0.05$.
[b] $p \leq 0.001$.
[c] $p \leq 0.01$.
AD = Alzheimer disease; CIND = cognitive impairment, not dementia; OR = odds ratio; CI = confidence interval; ref = reference.

76.7 cases per 1,000 person-years for those aged 90 years and older.[1] Although these estimated incidence rates are remarkably close to those found in ADAMS for individuals aged 80 years and older, the AD incidence rates from ADAMS for ages 72 to 79 years are higher than those estimated by the Brookmeyer equation. Notably, however, this is the age group in which the literature review[1] found the most heterogeneity among estimates worldwide and in which the US estimates were generally among the highest worldwide.

Few studies worldwide and even fewer US studies have estimated the incidence of mild impairment, labeled as either CIND or MCI. Differences in case ascertainment and diagnostic criteria make it difficult to compare studies, but our estimate of 60 cases per 1,000 person-years is slightly higher than 1 other multiethnic US study.[14] Although most studies report an increased risk of dementia among those with various forms of mild cognitive impairment, the estimates of rates of progression have varied widely. One study compared rates of progression for 16 different diagnostic definitions of mild impairment and found that the rates of progression to dementia over 2 years ranged from 0.3% to 29%.[31] Generally the definitions that allowed for impairment in both memory and nonmemory domains, similar to that used in the present study, resulted in the highest rates of conversion to dementia. We found that individuals who progressed from CIND accounted for 75% of the incident dementia cases. It is likely that for many of the other 25% of incident dementia cases, we may have been able to detect CIND prior to dementia with shorter follow-up intervals. These findings highlight that a period of mild impairment prior to dementia is likely detectable in the majority of individuals. A point that is often overlooked is that many individuals remain mildly impaired for years and may not progress to dementia. Over 50% of individuals with CIND in the present study did not progress to dementia during the time period that they were followed. From this group, we may be able to identify factors that delay progression to dementia. It has been estimated that delaying the onset of AD by 1 year in those at risk would result in about 1 million fewer cases by 2050,[1] with the largest decrease in the number of severe AD cases. So interventions, whether behavioral or pharmaceutical, that can even modestly delay progression from CIND to dementia may significantly decrease the burden of dementia on patients, families, and the health system.

One of the challenges of studying the outcomes of mild impairment has been the variable rates of diagnostic stability over time, regardless of the diagnostic nomenclature used. In our sample, 12% of those with CIND reverted to normal cognition at 1 of the follow-up assessments. This rate of reversion to normal is virtually identical to that in another study[32] that has used the same assessment and diagnostic procedures used in ADAMS.

In a study[31] that examined 2-year follow-up outcomes using 16 different algorithm classifications for MCI in a population-based sample, the rates of reversion to normal varied from 18% to 45% across the range of diagnoses indicating pathological decline; but in other studies using algorithm diagnoses, the reversion rate was as high as 93%.[33] These studies suggest that longitudinal outcomes may differ based on the diagnostic criteria applied and whether an algorithm versus a consensus conference approach is used.[9]

Limitations of ADAMS include possible response bias due to selective nonparticipation and attrition. This is a potential risk for all longitudinal studies, especially epidemiological studies of older individuals where comorbidities and mortality are common. To minimize response bias, we used archived information from prior interviews to develop response propensity models and associated weighting adjustments. Due to the relatively small sample size in some age groups, the estimates of incidence lack some precision. Also, due to the relatively small numbers in any given geographic region, we could not assess regional differences in outcomes. The higher rate of attrition for the Hispanic group may limit the generalizability of the results for that ethnic group. Diagnostic errors may have occurred due to inaccuracies in both the diagnostic criteria and in the assignment of the diagnosis by the consensus panel. The diagnostic criteria particularly for cognitive impairment without dementia are in the developmental stages and require further validation.[11,34] We attempted to minimize variability in the assignment of the clinical diagnosis within this study by using assessment teams based at a single site who used methods established in previous epidemiological studies and by using 1 common expert case review panel.

Our findings show that combined dementia and CIND affect a very large segment of the elderly population. Those with CIND are at particularly high risk of progressing to dementia; however, at least within a 6-year period, not all individuals with CIND progress to dementia. Our estimates of national incidence rates for CIND and dementia may serve as a benchmark against which to measure the effectiveness of potential future dementia prevention interventions.

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Potential Conflicts of Interest

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