

Age-specific and Sex-specific Prevalence and Incidence of Mild Cognitive Impairment, Dementia, and Alzheimer Dementia in Blacks and Whites

A Report From the Einstein Aging Study

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Abstract: As the population ages, the need to characterize rates of cognitive impairment and dementia within demographic groups defined by age, sex, and race becomes increasingly important. There are limited data available on the prevalence and incidence of amnesic mild cognitive impairment (aMCI) and nonamnesic mild cognitive impairment (naMCI) from population-based studies. The Einstein Aging Study, a systematically recruited community-based cohort of 1944 adults aged 70 or older (1168 dementia free at baseline; mean age, 78.8 y; average follow-up, 3.9 y), provides the opportunity to examine the prevalence and incidence rates for dementia, Alzheimer dementia (AD), aMCI, and naMCI by demographic characteristics. Dementia prevalence was 6.5% (4.9% AD). Overall dementia incidence was 2.9/100 person-years (2.3/100 person-years for AD). Dementia and AD rates increased with age but did not differ by sex. Prevalence of aMCI was 11.6%, and naMCI prevalence was 9.9%. aMCI incidence was 3.8 and naMCI incidence was 3.9/100 person-years. Rates of aMCI increased significantly with age in men and in blacks; sex, education, and race were not significant risk factors. In contrast, naMCI incidence did not increase with age; however, blacks were at higher risk compared with whites, even when controlling for sex and education. Results highlight the public health significance of preclinical cognitive disease.

Key Words: dementia, Alzheimer dementia, amnesic mild cognitive impairment, nonamnesic mild cognitive impairment, cohort study
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In 2005, it was estimated that over 24 million people worldwide had dementia. Dementia rates were higher in developed countries (North America and Western Europe) and lower in developing regions (Latin America, China, and the Western Pacific).¹ The US Census Bureau predicts that the population of adults above the age of 65 will double to constitute nearly 20% of the US population in the next 25 years.² Age-associated illnesses, particularly mild cognitive impairment (MCI) and dementia, are projected to have profound consequences for older adults, caregivers, the health care delivery system, and society. Detailed knowledge of age-specific, sex-specific, and race-specific rates of onset of MCI and dementia, as well as of their subtypes, is required to target interventions and develop preventive strategies.

Prior work has demonstrated that rates of dementia increase exponentially with age.^{3,4} The influence of race is less well established. Some studies report that the incidence and prevalence of dementia are higher in blacks than in whites,^{5–7} whereas others suggest that differences in dementia rates by race may be attributable to differences in education, socioeconomic status, health, or cultural factors.^{8,9} Additional studies are necessary to characterize rates of onset for dementia and cognitive impairment in ethnically diverse community-based studies to address these issues.

There has been increased interest in defining and diagnosing the stages of cognitive impairment that precede dementia, such as amnesic mild cognitive impairment (aMCI) and nonamnesic mild cognitive impairment (naMCI).¹⁰ However, few population-based longitudinal studies have reported the incidence and prevalence of these conditions. In addition, there is a paucity of information regarding age-specific, sex-specific, and race-specific rates of aMCI and naMCI. Studies that seek to enrich our understanding of these preclinical states will support health planning and facilitate the development of innovative preventive and treatment efforts that take age, sex, and race into account.

The Einstein Aging Study (EAS) is a longitudinal study of cognitive aging and dementia; the sample includes systematically recruited older adults drawn from an urban, multiethnic, community-dwelling population in Bronx County, NY. Participants receive comprehensive annual medical and neuropsychological evaluations. Using data from this cohort, we report total and age-specific prevalence and incidence rates for dementia, Alzheimer dementia (AD), aMCI, and naMCI overall and categorized by sex and race.

METHODS

Study Population/Eligibility

The EAS cohort has used systematic recruiting methods to reduce the selection biases that arise from clinic-based samples and to capture the racial diversity within the Bronx community. Since 1993, a total of 1944 participants have been enrolled. Between 1993 and 2004, information from the Health Care Financing Administration/Centers for Medicaid and Medicare Services rosters for Medicare-eligible persons aged 70 or above was used to develop sampling frames of community-residing participants in Bronx County. Since 2004, the New York City Board of Elections-registered voter lists for the Bronx have been used because of changes in policies for release of the Health Care Financing Administration/Centers for Medicaid and Medicare Services rosters. Individuals were mailed introductory letters regarding the study and were then contacted over telephone to complete a brief screening interview.¹¹ Potential participants who met preliminary eligibility criteria on the telephone were invited for further screening at the EAS clinical research center to determine final eligibility.

Eligible participants were at least 70 years old, Bronx residents, noninstitutionalized, and English speaking. Exclusion criteria included visual or auditory impairments that preclude neuropsychological testing, active psychiatric symptomatology that interfered with the ability to complete assessments, and nonambulatory status. Written informed consent was obtained at the initial clinic visit. The local institutional review board approved the study protocol.

EAS Assessment Battery

In-person evaluations were completed at baseline and at subsequent 12-month intervals.

Demographic and Health Status

Demographic information included self-reported race/ethnicity as defined by the US Census Bureau in 1994, education, previous occupations, income, marital status, and religion. Data on medical history, medications, family history, and health behaviors were obtained. Functional status was assessed using the clinical history form (C1 A: cognitive impairment/ dementia form) which is included in the Clinical and Neuropsychology Assessment of the Consortium to Establish a Registry for Alzheimer's Disease (CERAD),¹² a cognitive/functional impairment instrument, and the Instrumental Activities of Daily Living (IADL) Scale, a subscale of the Lawton Brody IADL Scale.¹³ The score on the IADL was based on 5 domains of function that were common to both elderly men and women. Scores for each domain were dichotomized as impaired versus nonimpaired, and then the domain scores were summed. If the participant agreed, an informant completed the CERAD clinical history form,¹² a cognitive/functional impairment instrument, and the Informant Questionnaire on Cognitive Decline in the Elderly¹⁴ forms.

Psychosocial Status

The 15-item Geriatric Depression Scale (GDS) was used to screen for depression.¹⁵ GDS ranged from 0 to 15 with scores of 6 or above indicating clinical depression. Anxiety was assessed using The Beck Anxiety Inventory.¹⁶ These instruments have high reliability and validity in community-based samples.¹⁷

Neurological Examination

The standard neurological physical examination was adapted from the Unified Parkinson's Disease Rating Scale.¹⁸ The evaluation assessed the participant's memory for significant recent events pertaining to news and personal events. The coherence and focus of responses, repetitiveness, and language were determined. When possible, informants were interviewed to ascertain whether they had noted any cognitive changes in the participant and to assess the accuracy of the participant's responses. The neurologist also assessed each participant for abnormal behaviors, fluctuation in cognition, and history of sleep disturbance and visual/auditory hallucinations. The neurologist assigned a Hachinski Ischemic Score,¹⁹ a Clinical Dementia Rating,²⁰ and provided a clinical impression of the presence or absence of dementia.

Neuropsychological Testing

Global cognitive status was ascertained by the Blessed Information–Memory–Concentration Test.²¹ Memory was measured using the Free and Cued Selective Reminding Test²² and the Logical Memory I²³ subtest from the Wechsler Memory Scale-Revised. Attention was measured using the Trail Making Test part A²⁴ and the Digit Span subtest of the Wechsler Adult Intelligence Scale-III.²⁵ Executive function was measured using the Trail Making Test part B²⁴ and the Letter Fluency “Forum Automation Solution” task.²⁶ Visuospatial construction was measured using the Block Design subtest and the Digit Symbol subtest, both from Wechsler Adult Intelligence Scale-III.²⁵ Language was measured using the Category Fluency task (animals, vegetables, and fruits)²⁷ and the Boston Naming Test.²⁸

Physical Measures

Anthropometrics, blood pressure, grip strength, and peak flow were measured according to standard protocols, and a fasting blood sample was obtained.

EAS Outcomes

Dementia and AD

A diagnosis of dementia was based on standardized clinical criteria from the Diagnostic and Statistical Manual, Fourth Edition,²⁹ and required impairment in memory plus at least 1 additional cognitive domain, accompanied by evidence of functional decline. Diagnoses were assigned at consensus case conferences, which included a comprehensive review of cognitive test results, relevant neurological signs and symptoms, and functional status. Memory impairment was defined as scores in the impaired range on any of the memory tests in the neuropsychological battery (Free and Cued Selective Reminding Test ≤ 24 ³⁰ or 1.5 SD below the age-adjusted mean on Logical Memory). Functional decline was determined at case conference based on information from self-report or informant report, impairment score on the IADL Lawton Brody Scale,¹³ clinical evaluation, and informant questionnaires.

AD was diagnosed in participants with dementia meeting clinical criteria for probable or possible disease established by the National Institute of Neurological and Communication Disorders and Stroke and the Alzheimer Disease and Related Disorders Association.³¹ Here, AD refers to AD alone or in combination with other dementia disorders. Incident dementia and AD were diagnosed in

persons free of dementia at baseline who met the criteria at follow-up.

A subset of individuals who participated in the clinical studies of the EAS came to autopsy, providing an important quality control for diagnostic accuracy. A clinical diagnosis of dementia had a positive predictive value of 96% for significant pathology upon autopsy. A clinical diagnosis of possible or probable AD had a positive predictive value of 79% for the presence of National Institute on Aging-Reagan intermediate or high likelihood Alzheimer-type pathology on the basis of an autopsy sample of 175.

MCI

Participants were classified as having aMCI if the memory domain was impaired or naMCI if there was impairment in 1 or more domains other than memory as defined below. For incidence analyses, participants were classified according to the subtype of MCI that occurred first. Persons eligible for incident MCI were therefore free of dementia and MCI at baseline.

i. aMCI was diagnosed according to updated criteria¹⁰ and required objective memory impairment as stated above, subjective memory impairment indicated by responses to self-reports or informant reports (self: CERAD¹²; informant: CERAD¹² or IQ CODE¹⁴), absence of functional decline (based on self-report or informant report, or absence of impairment on any of the domains measured in the IADL Lawton Brody Scale¹³), and that they be not classified as clinically demented. The aMCI group included both multiple and single-domain aMCI, as anyone who met the aMCI memory impairment criterion was included in the aMCI group regardless of whether they had cognitive impairments in other domains.

ii. naMCI was diagnosed in nondemented participants without functional impairment who did not meet memory criterion for aMCI but had impairment (1.5SD below the age-adjusted mean) in at least 1 nonmemory cognitive domain of attention, executive function, visuospatial ability, or language.

Data Analysis

Prevalence rates for dementia, AD, aMCI, and naMCI were calculated according to status at baseline assessment. Incidence rates were calculated as estimates of cases/100 person-years of follow-up in persons free of outcomes at baseline overall, by age, by age within sex, and race. Race-specific analyses were presented for those who identified themselves as non-Hispanic white (NHW) or non-Hispanic black (NHB). As these 2 groups constituted 94.8% of the EAS population, analyses within other racial/ethnic subgroups were not possible because of low sample size and inadequate numbers of events. Person-years of follow-up were calculated as the time between the baseline clinic visit and final follow-up examination, incident event, or death, whichever occurred earliest. For age-specific incidence, both cases and person-time were allocated to the appropriate age range. Confidence intervals (CI) were calculated using overdispersed Poisson regression models. Trend tests were conducted for each of the sex-specific and race-specific subgroups using quasilielihood regression models that included interaction terms. Cox proportional hazards models with chronological age as the time scale were used to estimate the association of incident dementia, AD, aMCI, and naMCI as a function of education, sex, and race. The proportional hazards assumption was tested for these models using sums of weighted residuals.

RESULTS

Baseline Demographics

The total EAS cohort included 1944 individuals. History of myocardial infarcts was reported by 10% of participants; 9% had experienced a stroke earlier, 57% had hypertension, and 17% had prevalent diabetes. The GDS identified 10.8% with clinically meaningful depression. Prevalence of these comorbidities was similar to rates for persons above the age of 65 in the US population.³² At baseline, 7.5% of the participants were current smokers and 46% were prior smokers.

At baseline, 126 of the 1944 participants (6.8%) were classified as having dementia (prevalent dementia) and 95

TABLE 1. Baseline Characteristics by Status at Enrollment and Follow-up

	Cohort at Baseline*	Never Developed MCI or Dementia	Normal at Baseline, MCI, No Dementia	Normal at Baseline, MCI Not Observed, Incident Dementia	Normal at Baseline, Incident MCI, Incident Dementia	MCI at Baseline, No Dementia	MCI at Baseline, Incident Dementia
Sample size	1168	660	220	33	39	158	58
Age at baseline—mean (SD)	78.8 (5.42)	78.2 (4.98)	79.0 (6.00)	81.1 (4.96)	80.3 (5.80)	79.4 (5.82)	81.7 (5.31)
Males (%)	39.3	39.2	39.0	27.3	38.5	43.0	37.9
Education in years—mean (SD)	13.5 (3.50)	13.9 (3.49)	13.1 (3.27)	12.0 (3.24)	13.4 (3.39)	12.5 (3.43)	13.2 (4.00)
Non-Hispanic white (%)	70.0	75.0	64.5	63.6	71.8	59.5	65.5
BIMC test—mean (SD)	2.45 (2.34)	1.76 (1.88)	2.64 (2.05)	4.06 (2.89)	2.90 (2.35)	3.54 (2.58)	5.14 (2.94)

*Participants free of prevalent dementia at baseline who had at least 2 annual evaluations.

BIMC indicates Blessed Information–Memory–Concentration test; MCI, mild cognitive impairment (includes both amnesic and nonamnesic MCI).

(4.9%) were subtyped as AD. Of the 1818 remaining individuals, 211 (11.6%) had prevalent aMCI, and 179 (9.9%) had prevalent naMCI. Sixty-four per cent (1168 participants) had at least 1 follow-up evaluation. Incidence rates were estimated for the 1168 participants who were free of dementia at baseline and had undergone a minimum of 2 clinic evaluations (Table 1). The mean age of this cohort at baseline was 78.8 years; 39.3% were males and 70.0% were NHW. On the basis of an average of 3.9 years of follow-up (range, 1 to 16 y), we identified 130 incident dementia cases, 127 incident aMCI cases, and 132 incident naMCI cases. Those who developed incident dementia or incident MCI were slightly older, less educated, performed less well on a test of global cognitive status, and were more likely to be black or female. Of the 130 incident dementia cases, 58 had prevalent MCI at baseline and 39 developed incident MCI and progressed to dementia (Table 1).

Incidence of Dementia and AD

Table 2 shows the number of cases and the sex-specific, age-specific, and race-specific incidence rates for all-cause dementia and specifically for AD. The overall incidence rate

of dementia for this population was 2.89 (2.33 to 3.58) per 100 person-years. As age increases, the rates of dementia increase overall for both men and women (trend test: men, $P = 0.003$; women, $P = 0.00003$) and for whites and blacks (trend test: whites, $P = 0.00007$; blacks, $P = 0.0003$). The incidence of dementia approximately doubled with each 5-year age interval, ranging from 0.66/100 person-years at ages 70 to 74 years to 11.30/100 person-years for those 90 years or more (Fig. 1). Age-specific rates for blacks were not significantly higher than for whites based on the interaction term in the quasiliikelihood regression model.

The overall incidence for AD was 2.26 (1.76 to 2.91) per 100 person-years. As age increased, the rate of AD increased for both men and women (trend test: men, $P = 0.0016$; women, $P = 0.00017$) and for whites and blacks (trend test: whites, $P = 0.0001$; blacks, $P = 0.001$). Men had nonsignificantly lower rates of AD in the younger age groups, and there were no differences in AD incidence by race.

Incidence of aMCI and naMCI

The overall incidence rate for aMCI was 3.79 (3.03 to 4.73) per 100 person-years and for naMCI it was 3.94 (3.17

TABLE 2. Incidence of All-cause Dementia and AD in the EAS Cohort: Person-years at Risk, Number of Cases, and Rates by Sex, Age, and Race (95% CI in Parentheses)

Sex or Race By Age Group	Incident Dementia			Incident Alzheimer Dementia		
	Person-years	No. Cases	Rate/100 Person-years	Person-years	No. Cases	Rate/100 Person-years
Total cohort						
70-74	628.21	4	0.64 (0.17-2.39)	628.21	4	0.64 (0.17-2.39)
75-79	1368.23	19	1.39 (0.78-2.48)	1368.23	14	1.02 (0.50-2.09)
80-84	1444.74	40	2.79 (1.52-5.03)	1444.74	31	2.15 (1.29-3.56)
85-89	859.23	44	5.12 (3.01-8.70)	859.23	33	3.84 (1.95-7.58)
90+	203.57	23	11.30 (5.55-22.99)	203.57	20	9.82 (4.61-20.92)
Total	4504.00	130	2.89 (2.33-3.58)	4504.00	102	2.26 (1.76-2.91)
Males						
70-74	249.00	1	0.40 (0.03-6.27)	249.00	1	0.40 (0.03-6.27)
75-79	551.27	4	0.73 (0.12-4.51)	551.27	2	0.36 (0.01-9.44)
80-84	570.46	16	2.80 (0.91-8.65)	570.46	12	2.10 (1.00-4.41)
85-89	345.02	14	4.06 (1.38-11.96)	345.02	10	2.90 (0.67-12.48)
90+	79.97	11	13.76 (4.21-44.93)	79.97	9	11.25 (3.12-40.53)
Total	1795.72	46	2.56 (1.77-3.71)	1795.72	34	1.89 (1.22-2.94)
Females						
70-74	379.21	3	0.79 (0.17-3.61)	379.21	3	0.79 (0.17-3.61)
75-79	816.96	15	1.84 (1.06-3.19)	816.96	12	1.47 (0.78-2.76)
80-84	874.29	24	2.75 (1.43-5.28)	874.29	19	2.17 (1.10-4.30)
85-89	514.22	30	5.83 (3.23-10.55)	514.22	23	4.47 (2.13-9.40)
90+	123.61	12	9.71 (4.18-22.56)	123.61	11	8.90 (3.60-22.00)
Total	2708.27	84	3.10 (2.38-4.04)	2708.27	68	2.50 (1.85-3.41)
Whites*						
70-74	373.70	2	0.53 (0.09-3.19)	373.70	2	0.54 (0.09-3.19)
75-79	900.63	11	1.22 (0.54-2.78)	900.63	10	1.11 (0.45-2.72)
80-84	1052.36	26	2.47 (1.07-5.71)	1052.36	19	1.81 (0.94-3.48)
85-89	658.56	30	4.55 (2.47-8.40)	658.56	21	3.19 (1.39-7.31)
90+	161.12	18	11.17 (4.81-25.93)	161.12	16	9.93 (4.16-23.73)
Total	3146.37	87	2.77 (2.13-3.60)	3146.37	68	2.16 (1.58-2.96)
Blacks*						
70-74	201.65	1	0.50 (0.03-7.42)	201.65	1	0.50 (0.03-7.42)
75-79	399.68	7	1.75 (0.75-4.09)	399.68	4	1.00 (0.34-2.97)
80-84	352.38	12	3.41 (1.56-7.46)	352.38	10	2.84 (1.14-7.08)
85-89	175.36	13	7.41 (2.53-21.74)	175.36	11	6.27 (1.80-21.88)
90+	40.49	5	12.35 (3.41-44.74)	40.49	4	9.88 (2.12-46.08)
Total	1169.57	38	3.25 (2.17-4.87)	762.67	30	3.54 (2.26-5.56)

*Sum of whites and blacks do not add to the total sample because those who designated race as "other" were not included (N = 51). AD indicates Alzheimer dementia; CI, confidence interval; EAS, Einstein Aging Study.

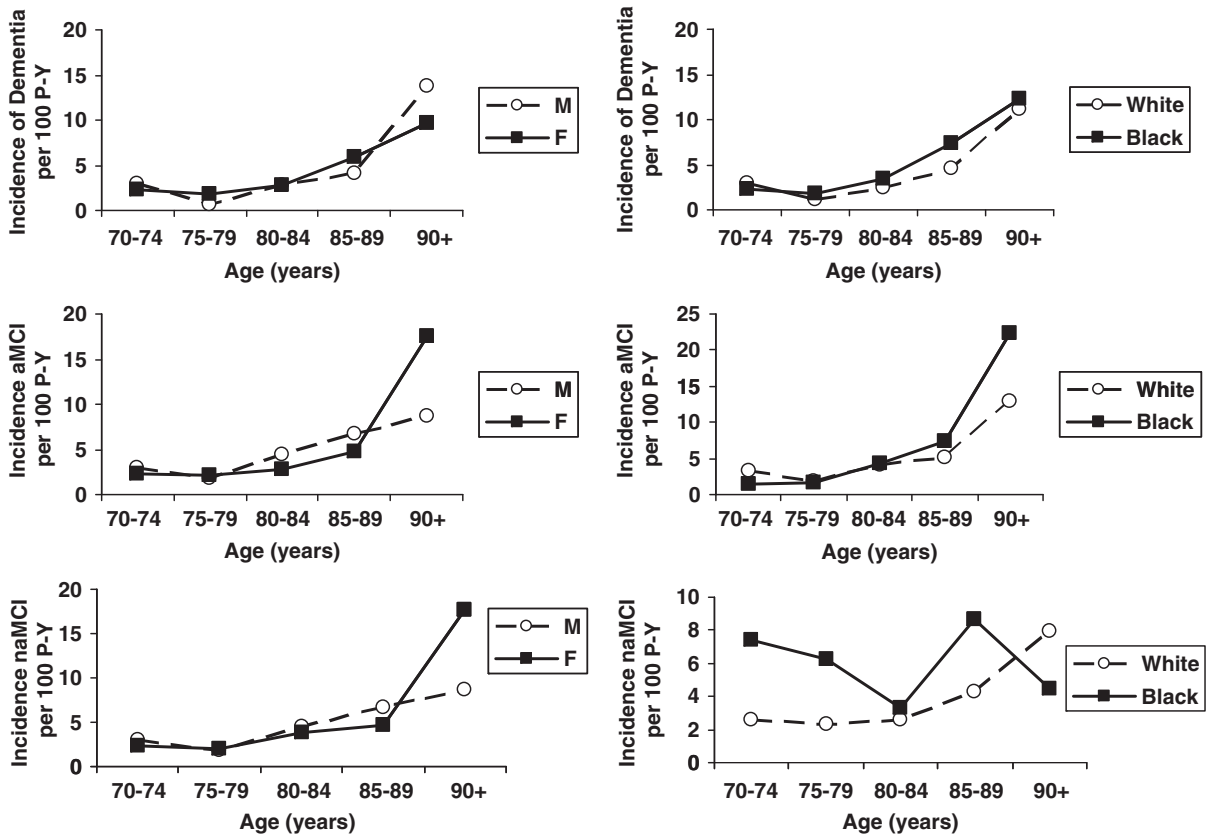


FIGURE 1. Age-specific incidence of dementia, AD, aMCI, and naMCI by sex and race. AD indicates Alzheimer dementia; aMCI, amnesic mild cognitive impairment; naMCI, nonamnesic cognitive impairment; P-Y, person-years of follow-up.

to 4.89) per 100 person-years. Table 3 shows the sex-specific, age-specific, and race-specific incidence rates for aMCI and naMCI. The rates of aMCI increased with age in men (trend test, $P = 0.039$) and in blacks (trend test, $P = 0.002$). Rates increased with age in women and in whites, but the trend was not significant. The rate of aMCI increased for individuals aged 90 or above in all subgroups of sex and race (Wald test: men, $P = 0.059$; women $P = 0.0085$; whites, $P = 0.018$; blacks, $P = 0.0048$). In contrast, the naMCI rates did not increase sharply with age for any subgroup (Fig. 1).

Demographic Risk Factors

Table 4 shows the results of the Cox proportional hazard models analyzing the effect of demographic variables on the risk of dementia, aMCI, and naMCI. A similar model was used for AD (not shown). Race, education, and sex were not significant risk factors for dementia, AD, or aMCI. Blacks were twice as likely as whites (hazard ratio, 2.04; CI, 1.39-3.01) to develop naMCI; education (hazard ratio, 0.95; CI, 0.90-0.997) was protective for naMCI.

DISCUSSION

We documented overall prevalence of dementia, AD, aMCI, and naMCI in a community sample of older adults in the Bronx. In addition, we assessed the age-specific, sex-specific, and race-specific incidence rates in this community. The EAS sample was diverse; it included 25% NHB, and

individuals with a broad range of educational achievement (0 to 25y). Age ranged from 70 to 101 years. Our results highlight the importance of preclinical cognitive disease in the population. Over 20% of the EAS sample was classified as having either prevalent aMCI or naMCI. Elderly black individuals seem to be at increased risk for naMCI.

A number of factors may explain the variability in rates across studies. Differences may be partly due to varying definitions of dementia, AD, aMCI, and naMCI or due to the application of these definitions. Geographical differences in rates may be attributable to variations in survival rates and to the prevalence of putative and protective factors. These factors may be environmental, genetic, or both.

Prevalence/Incidence of Dementia and AD

In the EAS, the prevalence of dementia was 6.5% and prevalence of AD was 4.9%. These rates are consistent with those reported in North American populations of comparable age.³³

The overall incidence rate of dementia for the EAS cohort was 2.89/100 person-years. When taking the age distributions into account, the EAS rate is generally consistent with those reported by other population-based studies.^{9,34-36} Launer et al³⁷ pooled results from 4 European countries included in the European Studies of Dementia network to provide overall and age-specific dementia rates. The overall rate of dementia was lower than EAS (1.84/100 person-years), most likely because of the younger age distribution in these samples. However, the

TABLE 3. Incidence of aMCI and naMCI in the EAS Cohort: Person-years at Risk, Number of Cases, and Rates by Sex, Age, and Race (95% CI in Parentheses)

Race, Sex, Age Groups	Incident aMCI			Incident naMCI		
	Person-years	No. Cases	Rate/100 Person-years	Person-years	No. Cases	Rate/100 Person-years
Total cohort						
70-74	499.08	13	2.60 (1.37-4.94)	499.08	23	4.61 (2.91-7.30)
75-79	1057.96	21	1.98 (0.32-12.50)	1057.96	40	3.78 (2.09-6.85)
80-84	1084.41	44	4.06 (2.42-6.80)	1084.41	31	2.86 (1.45-5.62)
85-89	563.34	31	5.50 (1.84-16.44)	563.34	28	4.97 (2.67-9.24)
90+	125.42	18	14.35 (3.14-65.58)	125.42	10	7.97 (4.00-15.88)
Total	3354.33	127	3.79 (3.03-4.73)	3354.33	132	3.94 (3.17-4.89)
Males						
70-74	198.40	6	3.02 (1.16-7.90)	198.40	9	4.54 (2.35-8.76)
75-79	424.56	8	1.88 (0.70-5.07)	424.56	18	4.24 (2.37-7.59)
80-84	405.69	18	4.44 (2.02-9.72)	405.69	13	3.20 (1.20-8.53)
85-89	225.24	15	6.66 (2.56-17.33)	225.24	8	3.55 (1.37-9.22)
90+	45.89	4	8.72 (4.17-18.23)	45.89	2	4.36 (1.22-15.62)
Total	1308.93	51	3.90 (2.70-5.61)	1308.93	50	3.82 (2.69-5.43)
Females						
70-74	300.67	7	2.33 (0.99-5.46)	300.67	14	4.66 (2.48-8.74)
75-79	633.40	13	2.05 (0.11-37.45)	633.40	22	3.47 (1.32-9.15)
80-84	678.73	26	3.83 (1.93-7.59)	678.73	18	2.65 (1.05-6.70)
85-89	338.10	16	4.73 (0.70-31.90)	338.10	20	5.92 (2.73-12.82)
90+	79.52	14	17.61 (2.67-116.26)	79.52	8	10.06 (4.61-21.95)
Total	2045.40	76	3.72 (2.81-4.91)	2045.41	82	4.01 (3.05-5.28)
Whites*						
70-74	312.16	10	3.20 (1.53-6.73)	312.16	8	2.56 (1.31-5.02)
75-79	730.40	14	1.92 (0.13-29.35)	730.40	17	2.33 (0.68-7.99)
80-84	845.40	34	4.02 (2.29-7.07)	845.40	22	2.60 (1.36-5.00)
85-89	463.02	24	5.18 (1.27-21.09)	463.02	20	4.32 (2.05-9.09)
90+	101.12	13	12.86 (1.63-101.42)	101.12	8	7.91 (3.69-16.94)
Total	2462.32	95	3.86 (2.96-5.03)	2462.32	75	3.05 (2.31-4.01)
Blacks*						
70-74	148.28	2	1.35 (0.29-6.37)	148.28	11	7.42 (3.67-15.01)
75-79	286.81	5	1.74 (0.73-4.16)	286.81	18	6.28 (3.61-10.92)
80-84	213.18	9	4.22 (1.12-15.87)	213.18	7	3.28 (0.50-21.71)
85-89	80.63	6	7.44 (2.56-21.63)	80.63	7	8.68 (2.69-27.97)
90+	22.34	5	22.38 (7.71-64.99)	22.34	1	4.48 (0.27-74.54)
Total	762.67	27	3.54 (2.26-5.56)	762.67	44	5.77 (3.90-8.54)

*Sum of whites and blacks do not add up to the total sample, as those who designated race as "other" were not included (N = 51).

aMCI indicates amnesic mild cognitive impairment; naMCI, nonamnesic mild cognitive impairment; CI, confidence interval; EAS, Einstein Aging Study.

age-specific rates for the incidence of dementia were very similar. Similarly, Kukull et al³⁴ reported an overall dementia rate of 2.03/100 person-years for members of the Group Health Cooperative of Puget Sound. Again, the lower dementia rate is likely because of the younger age of the cohort; however, when age-specific rates were compared, they were similar. Incidence in the 90+ age group was reported in a cohort study of residents of Laguna Woods, CA.³⁶ The rate of dementia in those between 90 and 94 years of age was 12.7% per year, comparable to our estimate of 11.3% per year in the EAS 90+ group (mean age, 92.8 y).

AD alone or in association with other dementia disorders comprised 77% of all dementia cases within the EAS. The overall AD incidence rate was 2.26/100 person-years in EAS. For persons up to age 85, the age-specific rates for AD were similar to those reported in other studies.¹

Comparisons for the 90+ age group are difficult, as the specific AD cases above age 90 are not uniformly reported. However, EAS rates are higher than the European Studies of Dementia network and Puget Sound studies in that age stratum.

Dementia and AD: Age Trends

In the EAS, the rates of dementia increased sharply with age. Similar findings in a meta-analysis presented by Jorm and Jolley³⁵ showed that among 23 studies age-specific incidence rates increased exponentially up to the age of 90. Corrada et al³⁶ reported similar results for age distributions beginning with 90 to 94 years (12.7% per year) and ending with rates for a 100+ age group (40.7% per year). AD rates also increased exponentially with age in the EAS, similar to prior reports.^{1,34,37}

Dementia and AD: Sex-specific Rates

Dementia rates were similar in men and women; both groups showed an increase in rates with age. The results from Jorm and Jolley,³⁵ Corrada et al,³⁶ and Fitzpatrick et al⁹ agree with these results, with the exception of those for the oldest-old age group. Prior reports have shown that in the 90+ age group, women tend to have a higher incidence of dementia, in particular AD.^{9,34,35,37} In contrast, EAS AD rates for men were nonsignificantly lower than those for women before the age of 90.

TABLE 4. Hazard Ratios for Dementia, aMCI, and naMCI in the EAS Cohort*

Variable	Hazard Ratio	95% CI	P
Dementia			
Sex (male)	0.97	0.92-1.02	0.18
Education (per y)	0.89	0.61-1.29	0.53
Race (black)	1.31	0.88-1.94	0.18
aMCI			
Sex (male)	1.11	0.77-1.62	0.57
Education (per y)	1.01	0.96-1.06	0.84
Race (black)	0.93	0.60-1.45	0.74
naMCI			
Sex (male)	1.07	0.73-1.56	0.73
Education (per y)	0.95	0.90-0.997	0.04
Race (black)	2.04	1.39-3.01	0.0003

*Cox models included sex, education, and race, with non-Hispanic whites and women as the reference groups. "Years of education" was included as a continuous predictor. All models used chronological age as the time scale.

aMCI indicates amnesic mild cognitive impairment; naMCI, non-amnesic mild cognitive impairment; CI, confidence interval; EAS, Einstein Aging Study.

Dementia and AD: Race-specific Rates

A few studies have compared dementia rates by race within the same geographic area, and results have been inconsistent across studies. In the EAS population-based sample, we observed no differences in rates of total dementia among whites and blacks. The Cox models showed that race was not a significant risk factor for dementia after adjustment for sex and education. The model for AD showed similar nonsignificant results.

Blacks have been reported to have higher incidence of AD compared with NHWs in a cohort of community-based residents of northern Manhattan.^{6,7} Gurland et al⁶ reported higher prevalence and incidence of all-cause dementia for African Americans compared with non-Latino whites in the North Manhattan Aging Project, although the excess incidence among African Americans was observed only below the age of 85. Tang and colleagues⁷ has reported higher incidence of AD for blacks than for NHWs in a community-based sample from northern Manhattan. These differences were not attributable to differences in education, nor to differences in frequency of cardiovascular comorbidities. In contrast, other studies have not observed race differences in dementia,⁸ nor have they found that differences are diminished after adjustment for age and education.⁹ Fillenbaum et al⁸ reported no differences in incidence rates for all-cause dementia between blacks and whites in the Piedmont area of North Carolina. Fitzpatrick⁹ estimated incidence rates for total dementia in the Cardiovascular Health Study and found racial differences, with higher rates for blacks. However, the difference was attenuated when adjusted for age and education and with further adjustment for differences in case ascertainment. Similarly, analyses by dementia subtype demonstrated higher incidence of AD among African Americans, but the difference was only of borderline statistical significance and was not evident after adjustment for differences in case ascertainment.

Prevalence/Incidence of aMCI and naMCI

The overall prevalence of MCI (aMCI and naMCI combined) in the EAS was 21.5% at baseline assessment.

This rate is similar to rates reported by other community-based cohorts in the United States.^{38,39} We found little difference in rates of prevalent MCI between men and women (21.0% in women, 22.2% in men) and lower rates in whites than in blacks (19.1% vs. 27.3%). These results were consistent with Manly's findings.³⁹ Our observed difference in MCI by race was entirely due to a higher prevalence of naMCI in blacks (16.30% vs. 6.86% in whites). Petersen et al³⁸ reported prevalence rates of 11.1% for aMCI and 4.9% for naMCI in a cohort from the Mayo Clinic. The EAS prevalent aMCI rates are similar but prevalent naMCI rates were higher in the EAS; similar diagnostic criteria were used in both studies. This difference may be attributable to differences in racial/ethnic or general health characteristics of the study populations. In particular, the Mayo Clinic population is predominately white.

In our study, the incidence of aMCI was 3.8 and for naMCI it was 3.9/100 person-years. In the northern Manhattan study, incidence rate for aMCI was 2.3 and that for MCI without memory impairment was 2.8/100 person-years.³⁹ The higher rates in the EAS may be because of its older age distribution.

aMCI and naMCI: Age-specific, Sex-specific, and Race-specific Incidence Rates

There is a paucity of data regarding age-specific, sex-specific, and race-specific incidence rates of aMCI and naMCI. In the EAS, rates of aMCI increased after the age of 80 for men and women and for whites and blacks. In the Cox proportional hazards model, sex, education, and race were not significant risk factors for incident aMCI. In contrast, there was no marked increase in naMCI with increasing age or between men and women in the EAS cohort. However, naMCI incidence rates were higher in blacks compared with whites. When controlling for sex and education in the Cox model, we observed a 2-fold increased risk for naMCI among blacks compared with whites. This observation is consistent with prior studies showing higher rates of cerebrovascular disease and more prevalent cardiovascular risk factors among African Americans compared with whites⁴⁰ and with recent reports suggesting that naMCI often has a vascular etiology.^{33,41} Roberts et al⁴¹ have reported that cardiovascular disease is more strongly associated with naMCI than with aMCI. Similarly, Reitz et al³³ have reported that the association between hypertension and total MCI is driven by an association with naMCI.

Strengths and Limitations

The operational definition of MCI and its subtypes has evolved over time and continues to vary considerably among studies. Some definitions are primarily based on clinical impression, whereas others rely on neuropsychological cutoff scores and still others combine approaches. These neuropsychological tests also vary in sensitivity and specificity for identification of impairment. Classification of cognitive status in the EAS was based on a comprehensive neuropsychological test battery supplemented by a complete neurological examination. Diagnosis of dementia was based on standard criteria applied at a consensus case conference. The definitions of aMCI and naMCI reported here follow widely adopted diagnostic guidelines,¹⁰ whereas dementia and AD were also defined according to standard criteria.^{20,29,31} Given the many sources of unreliability in ascertaining these outcomes, the consistency of EAS results with available data is reassuring.

The prevalence of dementia and AD may be underestimated in the EAS, because individuals with severe dementia are less likely to participate in a community-based study and those who are institutionalized were not included. Another limitation of this study was that persons lost to follow-up may not develop cognitive impairment at the same rate as do those who remain in the sample, a phenomenon known as informative censoring. When comparing baseline characteristics of the 1168 EAS participants with at least 1 follow-up clinic visit with those of participants with only baseline evaluations, there were no significant age, sex, or race differences. However, the mean years of education was significantly lower ($P < 0.0001$) and the Blessed Information–Memory–Concentration test was significantly higher ($P < 0.0001$) for the group not completing a follow-up visit. As those lost to follow-up tended to have reduced global cognitive performance and lower education, the incidence rates we reported may be underestimates. Another limitation is the relatively short follow-up interval. For those with more than 1 annual visit, the mean follow-up time was 3.9 years (range, 1 to 16 y). In any cohort, censoring before death or dementia diagnosis results in under-ascertainment of cases. Finally, when subtypes of dementia and MCI were examined, demographic comparisons were limited by small sample size.

This study had several notable strengths. First, the cohort was systematically recruited, and comparisons with data from the US Census indicate that the EAS cohort was similar to the elderly population of Bronx County, NY, in distributions of age, sex, and education. Another strength is the racial and educational diversity of this cohort. Clinic-based studies often include only those with more severe cognitive impairment compared with a community-based sample.

In summary, this study contributes valuable information regarding the prevalence and incidence of cognitive impairment in community-dwelling individuals. In particular, few prior studies have reported the incidence of aMCI and naMCI in an ethnically diverse community-based sample. As the population ages, both MCI and dementia will present an increasing burden on the health care system as well as on families and caretakers. The high prevalence of preclinical dementia documented here underscores the need to develop interventions that will delay or prevent the onset of dementia.

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