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Prevalence of Narcolepsy in King Count, Washington, USA

WT Longstreth Jr, MD, MPH[1,2,3], **Thanh G N Ton, PhD**[1,2,3], **Thomas Koepsell, MD, MPH**[1,3,4,5], **Vivian H Gersuk, PhD**[6], **Audrey Hendrickson, MPH**[1,2], and **Sarah Velde, PhD**[1,2]

[1] Neuroepidemiology Group, University of Washington, Seattle, WA

[2] Department of Neurology, School of Medicine, University of Washington, Seattle, WA

[3] Department of Epidemiology, School of Public Health and Community Medicine, University of Washington, Seattle, WA

[4] Department of Health Services, School of Public Health and Community Medicine, University of Washington, Seattle, WA

[5] Department of Medicine, School of Medicine, University of Washington, Seattle, WA

[6] The Benaroya Research Institute at Virginia Mason, Seattle, WA

Abstract

Background—Relatively few epidemiologic studies have focused on narcolepsy, a disabling sleep disorder with strong association with HLA-DQB1*0602.

Methods—We sought to estimate prevalence of narcolepsy using multiple overlapping techniques to identify residents of King County, Washington who were 18 years or older with physician-diagnosed narcolepsy. Patients were entered into a registry and recruited into an epidemiologic study entailing interview and buccal scrapings to determine HLA-DQB1*0602 status. Missing values were imputed to allow prevalence to be estimated based on all 425 patients entered into the registry between 2001 and 2005, whether they were recruited into the epidemiologic study (n=279) or not (n=146).

Results—As of 2001 July 01, estimated prevalence per 100,000 of physician-diagnosed narcolepsy with cataplexy was 21.8 (95% confidence interval (CI): 18.8 to 24.8), similar to prior studies. The median age of onset was 14 (interquartile range: 10 to 18). For narcolepsy with HLA-DQB1*0602, prevalence was 15.3 (95% CI: 12.8 to 17.9). Estimated prevalence was higher in women than men and in African-Americans than other racial groups.

Conclusions—These differences could reflect problems in identification and recruitment or may provide etiologic clues about narcolepsy. This study illustrates the challenges in performing population-based studies of narcolepsy.

Keywords

narcolepsy; cataplexy; HLA-DQB1*0602; epidemiologic studies; prevalence; incidence

Correspondence to WT Longstreth, Jr, MD, Department of Neurology, Box 359775, Harborview Medical Center, 325 Ninth Avenue, Seattle, WA 98040-2420 (voice: 206 731-3251; facsimile: 206 731-8787; wl@u.washington.edu).

INTRODUCTION

Narcolepsy is a disabling sleep disorder characterized by excessive daytime sleepiness and episodic loss of muscle tone triggered by strong emotions (cataplexy). Narcolepsy likely results from selective destruction of hypocretin-producing cells in the lateral hypothalamus of genetically susceptible people, defined by their carrying human leucocyte antigen (HLA-) DQB1*0602.[1] We sought to identify all residents of King County, Washington with narcolepsy in order to estimate prevalence of disease, to seek etiologic clues, and to characterize clinical features in a population-based rather than clinic-based series of patients. King County, Washington was an attractive study setting because similar population-based studies of neurologic conditions have been done in King County,[2–4] it has a high density of medical care providers, and it includes a large number of sleep disorder centers. We reasoned that patients with narcolepsy would have available many sources of medical care for sleep disorders, including from experts in sleep medicine. Here we estimate the prevalence of narcolepsy in this population by various disease definitions, gender and race. To allow comparison to another study that reported incidence,[5] we also tried to estimate approximate incidence using recalled year of onset, realizing that study design and data available were not ideal for this purpose.

METHODS

Setting

Our goal was to identify as many residents of King County, Washington as possible who were 18 years or older with a physician diagnosis of narcolepsy. King County is the most populous county in the state, having about 1.7 million people by the 2000 Census in 2,134 square miles (5,527 km^2). Its largest city is Seattle, with about a third of the County's population. Most King County residents seek medical care within the county, which includes 10 sleep disorder centers.

Case ascertainment

We tried to identify all eligible patients over the course of study recruitment from 2001 July 01 to 2005 June 30 and determine whether they had the onset of their disease before the prevalence date, which we set at 2001 July 01. We used multiple overlapping methods, some of which were directed at clinicians and some at patients. We met with directors of sleep disorders centers to explain the study and seek their help. During the course of recruitment, we sent monthly letters to clinicians working in sleep disorder centers and to all neurologists in King County asking that they let us know about any patient whom they diagnosed with narcolepsy. We also had a single mailing to the County's family medicine physicians and to psychiatrists. Finally we had a mailing to the community clinics in King County where patients without financial resources often receive their care.

With respect to direct appeals to patients, study fliers were posted in waiting rooms of sleep disorder centers. Presentations about the study were made at the region's support groups and at the Narcolepsy Network's national annual meeting held in King County in 2004. During recruitment, a monthly newsletter was sent to all who had participated in the study. Spreading news about the study by word of mouth was encouraged. Pharmacists in King County agreed to include an information sheet about the study with all prescriptions for certain medications commonly used to treat patients with narcolepsy. Advertisements and public service announcements were placed in several newspapers, on County buses, on several radio stations, and on television. All of these sources of information included a contact telephone number and address for the study's web site. The University of Washington also maintained a web site for research volunteers where information about the study was available.

People who contacted study personnel concerned that they might have a sleep disorder, or specifically narcolepsy, were encouraged to discuss their concerns with their regular health care provider and were provided information about the County's sleep disorder centers. If a physician subsequently diagnosed them with narcolepsy, they became eligible for the study.

Data sources

Patients with physician-diagnosed narcolepsy identified by any of these means were entered into a registry and invited to participate in an epidemiologic study of narcolepsy. Patients identified by clinicians were invited through their clinician, while patients contacting study personnel were invited directly. The Institutional Review Board at the University of Washington approved the study.

For those not recruited into the epidemiologic study, information was limited. For those who agreed to participate and who provided written informed consent, a trained professional interviewer asked the patient specific questions about clinical manifestations, onset of disease, and diagnosis. Also in those who agreed, medical records were requested and abstracted by the study neurologist. Information about cataplexy came from interviews and review of medical records. The interview included self-reported cataplexy as well as several screening questions. An affirmative response to experiencing muscle weakness when telling or hearing a joke, or when laughing, was considered a positive screen for cataplexy.[6] For this study, we defined cataplexy as present if indicated by medical record review, self-reported cataplexy, or a positive screen.

Because most patients with narcolepsy, especially narcolepsy with cataplexy, have been found in previous studies to carry HLA-DQB1*0602,[1] we were keenly interested in patients' HLA type. Thus, in those who consented, the interviewer collected buccal specimens, which were used to obtain genomic DNA. Genotyping of HLA-DQB1 entailed quantitative DNA amplification and fluorescence detection with sequence-specific probes, as described previously for HLA-DRB1 alleles.[7] Briefly, primers and probes were designed within exon 2 of the HLA-DQB1 locus based on the sequence alignments of the IMGT/HLA Sequence Database (http://www.ebi.ac.uk/imgt/hla/align.html) to distinguish major DQB polymorphisms at codons 25–28 and 47–48, which identify allele DQB1*0602 and distinguish several additional DQB1 alleles. Primers and probes for HLA-DRA were used to control for template quality and quantity, as previously described.[7] Approximately 30 nanograms of genomic DNA in TaqMan® Universal Master Mix (Applied Biosystems, Inc.) in a final volume of 25 microliters per well were combined with primers and probes and amplified on an ABI PRISM® 7000 Sequence Detection System (Applied Biosystems, Inc.) for 45 cycles. Presence or absence and copy number of DQB1*0602 were scored for each sample.

Disease definitions

Current definitions distinguish between narcolepsy with and without cataplexy.[8] Sleep studies are required to make the diagnosis in patients without cataplexy, and were not always available in patients with a physician diagnosis of narcolepsy. Based on all the information collected on these patients, we estimated the date of onset of narcolepsy symptoms. Using several definitions of narcolepsy, we estimated prevalence under each definition on 2001 July 01 in King County in those 18 years or older, about 1.4 million people. Although current definitions of narcolepsy do not require the patient to carry HLA-DQB1*0602,[8] we also defined a group in which all patients were positive for HLA-DQB1*0602. This group is key for future epidemiologic studies of etiologic risk factors that may only be relevant in susceptible people defined by their carrying HLA-DQB1*0602.[9]

Statistical analyses

Using information on both recruited and non-recruited cases in our registry, we estimated prevalence for three definitions of narcolepsy: (1) with and without cataplexy, (2) with cataplexy, and (3) with HLA-DQB1*0602. To estimate age, gender, and race-specific prevalences of narcolepsy in King County, we imputed variables for which values were unknown. Our registry included 425 cases, 146 of whom were not recruited for the study. For those not recruited, age was missing for 85; race, for 123; and HLA status, cataplexy status and age of onset, for all. We multiply imputed age, race, HLA status, cataplexy status and age of onset using the method of chained equations[10] as implemented by the **ICE** package in Stata. The predictive mean matching option was used when imputing continuous variables, thus confining imputed values to the set of values actually observed among subjects without missing data. Imputation models for race, cataplexy, and HLA status included gender, age and referral source as predictors; models for age included gender and referral source; and models for age of onset included gender, age, and referral source. Ten sets of data were created and analyzed in parallel, and the results were combined using standard formulas[11] to obtain prevalence estimates that include non-recruited cases. Observations in which age of onset occurred after prevalence date were excluded from analyses.

Finally, we estimated approximate incidence of narcolepsy in King County, Washington based on date of symptom onset. Following the lead of other investigators,[5] we ignored the interval from 1996 to the time of recruitment given delays that may occur in making a diagnosis. We then tabulated the number of cases with symptom onset in each year over two decades from 1975 to 1994. The estimated population of King County in each year was used to calculate person-years for the denominator. This method is only approximate for several reasons. First, some of the cases counted as incident cases did not reside in King County in the year of onset. Second, some incident cases who did reside in King County during those years have since emigrated and were not captured among current prevalent cases. Third, some incident cases may have died since disease onset and no longer be available for study. For present purposes, we assumed that in and out-migration of cases were approximately equal and that under-enumeration of cases due to subsequent death was negligible. All analyses were conducted in Stata (version 9.2, College Station, Texas)

RESULTS

Over the recruitment period, 425 cases were entered into the registry. Initial contact about cases came from sleep medicine centers (74 percent), neurologists (8 percent), or patients (18 percent). We succeeded at recruiting 279 (66 percent) of those in the registry into the epidemiologic study. All underwent the interview and had HLA status established. Medical records were sought in all 279 patients and obtained in 223 (80 percent). Review of these records revealed that only 13 had had HLA-DQB1*0602 status established during their routine medical care. Based on all sources of information about the course of their illness, all 279 patients (100 percent) had experienced excessive daytime sleepiness, 186 (67 percent) had experienced cataplexy, and 138 (49 percent) carried at least one allele of HLA-DQB1*0602.

Table 1 shows demographics for patients in the registry broken down by those recruited into the epidemiologic study or not, along with similar information for all residents of King County 18 years or older. For 146 patients not recruited into the epidemiologic study, age was known in only 61. Those recruited were slightly older than those not recruited but of known age. Women were more likely than men to appear in the registry and to participate in the epidemiologic study. Race was unknown for 123 not recruited into the epidemiologic study. Cases who were self-referred were more likely than others in the registry to be recruited into the epidemiologic study.

The numbers used to estimate prevalence excluded three patients who were not known to have onset of their symptoms prior to prevalence date. Of the remaining 276 patients, 185 (67 percent) had cataplexy and 138 (50 percent) were positive for HLA-BQB1*0602. Among the 185 with cataplexy, 113 (61 percent) were positive for HLA-DQB1*0602, and among the 138 who were positive for HLA-DQB1*0602, 113 (82 percent) had cataplexy. Of the remaining 25 without cataplexy, all but two patients, who indicated that they had been diagnosed in the distant past, had seen a sleep medicine specialist and had formal sleep studies. Although medical records were available in these 23 patients, specific results of sleep studies were lacking in four. In nine patients, sleep studies met all criteria for narcolepsy without cataplexy. In the remaining 10 patients, one criterion was not met: duration of sleep the night before the multiple sleep latency test was unknown in four; mean sleep latency was more than eight minutes in two; and two or more sleep onset rapid eye movement sleep periods were not observed in four. For the subset of 14 patients less than 50 years old on prevalence date, seven met all criteria for narcolepsy without cataplexy, and only two had less than two sleep onset rapid eye movement sleep periods on their sleep studies.

Table 2 provides estimates of prevalence by age, sex, and race based on the 276 recruited cases in the first columns and based on recruited and non-recruited cases with imputed values in subsequent columns. Overall estimated prevalence per 100,000 people for physician-diagnosed narcolepsy with or without cataplexy was 30.6 (95 percent CI: 27.6, 33.5), with median age of onset at 15 years old (interquartile range (IQR): 11, 20). For narcolepsy with cataplexy, prevalence was 21.8 (95 percent CI: 18.8, 24.8), with median age of onset at 14 (IQR: 10, 18). For narcolepsy with HLA-DQB1*0602, prevalence was 15.3 (95 percent CI: 12.8, 17.9), with median age of onset at 15 (IQR: 11, 19). Prevalence was higher in women than men and in African-Americans than other racial groups. No clear pattern was evident with age.

Based on year of symptom onset among the 279 patients in the epidemiologic study, estimated incidence per 100,000 person-years for narcolepsy from 1975 to 1984 was 0.39 (95 percent confidence interval CI: 0.30, 0.51) and from 1985–1994 was 0.40 (95 percent CI: 0.24, 0.48). After imputing year of symptom onset for patients in the registry who were not recruited into the epidemiologic study, the corresponding overall estimated incidence per 100,000 person-years from 1975 to 1984 was 0.58 (95 percent CI: 0.43, 0.73) and from 1985 to 1994 was 0.62 (95 percent CI: 0.43, 0.73) and for the two decades combined was 0.60 (95 percent CI: 0.48, 0.69).

DISCUSSION

Using multiple overlapping techniques, we tried to identify all residents of King County, Washington who were 18 years or older with a physician diagnosis of narcolepsy. We entered 425 such patients into a narcolepsy registry and recruited 279 (66 percent) into a more in-depth epidemiologic study, seeking details about their disease through a structured interview and review of their medical records. Median age of onset was 15 years old, as described by others,[12,13] but with a single peak as described in the population-based study from Olmsted County, Minnesota.[13] We estimated the prevalence per 100,000 people on 2001 July 01 in residents 18 years or older of narcolepsy with or without cataplexy as 30.6 (95 percent CI: 27.6, 33.5), of narcolepsy with cataplexy as 21.8 (95 percent CI: 18.8, 24.8) and of narcolepsy with HLA-DQB1*0602 as 15.3 (95 percent CI: 12.8, 17.9).

The prevalence of narcolepsy with cataplexy that we estimated for King County, Washington is within the range of what has been estimated in studies with multistage direct screening of large numbers of people.[9] For example, in a landmark study of a Finnish twin registry, 11,354 members were screened, and three were found to have narcolepsy with cataplexy, yielding a prevalence of 26 per 100,000 (95 percent CI: 5, 77).[14] Another study approached the problem

as we did, trying to identify all residents of Olmsted County, Minnesota with a diagnosis of narcolepsy with cataplexy from 1960 to 1989 and estimated a prevalence of 36 per 100,000 (95 percent CI: 25, 50).[5] Although we attempted the same type of study in a more populous and somewhat more racially diverse county and identified more patients than in the previous study, the study from Olmsted County had the advantage of the well-established medical-records linkage system of the Rochester Epidemiology Project. We suspect that our estimates may be low because we did not identify or recruit all people affected by narcolepsy with cataplexy, whether diagnosed by a physician or not. Similarly, we believe that our estimate of the incidence of narcolepsy with or without cataplexy was probably low at 0.60 per 100,000 person-years compared to 1.37 found in Olmsted County, Minnesota.[5]

Prior studies have suggested that, if an imbalance exists, narcolepsy is likely more common in men than women.[9] Our finding prevalence higher in women than men may reflect a registry recruitment bias, which was also evident when looking at the percent of those who were in the registry and recruited into the epidemiologic study: 61 percent for men and 69 percent for women.

Narcolepsy was more common in African-Americans than in other racial groups despite our concern that African-Americans might have reduced access to medical care, in particular specialty care. These findings agree with a 1945 study of male United States military recruits, suggesting that narcolepsy was more common in African-American than white military personnel.[15] Race may also influence participation,[16] although results of a recent meta-analysis found that the proportion consenting for interview studies was decreased, but not significantly, among African-American subjects compared to whites.[17] We also found the lowest prevalence in Asian-Americans. However, the heterogeneous nature of this group should be borne in mind. For example, while high prevalences have been reported in studies from Japan, a low prevalence has been reported from Singapore.[9] The question of whether narcolepsy is more prevalent in certain racial or ethnic groups, which may provide etiologic clues, will require additional studies to be performed in more racially and ethnically diverse populations than available in Olmsted County, Minnesota and King County, Washington.

Narcolepsy is likely caused by key environmental exposures in genetically susceptible individuals defined by their carrying HLA-DQB1*0602. To address this gene-environment interactions, HLA-DQB1*0602 status must be known in studies seeking to identify etiologic risk factors. Including subjects who were negative for HLA-DQB1*0602 would weaken investigators' ability to identify risk factors that operate only in genetically susceptible individuals. Consequently, we established the HLA-DQB1*0602 status of all patients recruited into the epidemiologic study using DNA available from buccal swabs. None of the patients recruited into the epidemiologic study refused this aspect of it, although the DNA testing may have influenced patients' decisions to participate at all. Only rarely had the HLA-DQB1*0602 status been established as part of routine care. As would be expected, most of these patients (82 percent) with a physician diagnosis of narcolepsy and HLA-DQB1*0602 positivity had a history of cataplexy. Sleep medicine experts had evaluated most of the remaining patients to support their diagnosis of narcolepsy. This group defined by their HLA-DQB1*0602 positivity will be used in future epidemiologic case-control studies aiming to identify etiologic risk factors.

Population-based studies pose many challenges for diseases such as narcolepsy, which rarely results in hospitalization or death and for which few if any pre-existing registries exist. Despite our efforts, we likely failed to identify and recruit all patients with narcolepsy in King County and thus may have underestimated the prevalence. In addition, patients recruited into the study did not undergo a uniform assessment, and practicing sleep medicine experts, representing pulmonologists, psychiatrists, neurologists and others, may have had different thresholds for

assigning a diagnosis of narcolepsy in questionable cases, especially given the change in the diagnostic criteria over the course of the study and the imprecision of formal sleep studies.[8] As a result, some of the cases were likely misclassified as having narcolepsy when in fact they had some other hypersomnia. Even the presence of cataplexy does not afford protection from misclassification in that we had no way to apply a uniform definition of cataplexy throughout King County. Our most precise measurement was HLA-DQB1*0602. Future epidemiologic studies should consider including HLA-DQB1*0602 status in their case definition, even though it is not required for a clinical diagnosis.[8]

Population-based research has become more difficult to perform in recent years.[18] Participation by eligible study subjects has fallen. In this study, 66 percent of the eligible patients identified participated in the more detailed epidemiologic study. Using similar techniques in western Washington state, our Neuroepidemiology Group has achieved greater participation in the past from eligible patients with subarachnoid hemorrhage (93 percent, 149/160),[2] amyotrophic lateral sclerosis (97 percent, 174/180),[3] and intracranial meningioma (84 percent, 200/238).[4] The lower percent of patients participating in the current study compared to these older studies whose recruitment was completed prior to 2000 may reflect the disease being studied, the genetic test being requested, or a increasing reluctance of people to participate in epidemiologic research from which they stand to derive no direct benefit. Challenges recruiting study subjects, interacting with clinicians, and working with institutions were all greater than in previous studies in this region, making the conduct of such studies increasingly difficult and expensive.

Population-based epidemiologic investigations of uncommon conditions such as narcolepsy are essential if we are to learn more about its etiology and prevention. One possibility would be to conduct studies in large managed health care plans, as has been done for Parkinson disease and amyotrophic lateral sclerosis.[19,20] The prevalence of narcolepsy lies between that of amyotrophic lateral sclerosis at 4 per 100,000 and multiple sclerosis at 90 per 100,000,[21] and yet narcolepsy has been investigated epidemiologically far less than these and many other neurologic conditions. As in any epidemiologic study, validity is dependent in part on the proportion of eligible subjects who participate, which for narcolepsy could be improved. Ultimately those affected by narcolepsy are key to unraveling the mystery of their disease.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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TABLE 1

Age, sex and racial distribution of patients from the narcolepsy registry compared to general population 18 years and older in King County, Washington in 2001.

	Narcolepsy Registry (n = 425)		King County, WA
	Not recruited* N = 146 (%)	Recruited N = 279 (%)	Population N = 1,366,417 (%)
Age			
18–29	19 (31.2)	69 (24.7)	304,072 (22.2)
30–39	14 (23.0)	44 (15.8)	307,237 (22.5)
40–49	11 (18.0)	54 (19.4)	297,212 (21.8)
50–59	10 (16.4)	51 (18.3)	213,692 (15.6)
60–69	4 (6.6)	36 (12.9)	106,930 (7.8)
70–79	2 (3.3)	17 (6.1)	82,493 (6.0)
80+	1 (1.6)	8 (2.9)	54,781 (4.0)
Unknown	85 (58.2)		
Sex			
Men	67 (45.9)	103 (36.9)	674,269 (49.3)
Women	79 (54.1)	176 (63.1)	692,148 (50.7)
Race			
White	13 (56.5)	228 (81.7)	1,090,507 (79.8)
African-American	7 (30.4)	26 (9.3)	71,314 (5.2)
Asian-American	3 (13.1)	15 (5.4)	163,066 (11.9)
Other	0 (0.0)	10 (3.6)	41,530 (3.0)
Unknown	123 (84.3)		
Referral Source			
Sleep Medicine	120 (82.2)	194 (69.5)	
Clinic			
Neurologist	15 (10.3)	18 (6.5)	
Patient	11 (7.5)	67 (24.0)	

*Percent in age and race categories calculated among those with known ages and race except for “unknown” categories that are calculated using overall total.

TABLE 2

Estimates of prevalence for narcolepsy on 2001 July 01 in King County, Washington.

A. Narcolepsy with or without cataplexy

	Prevalence per 100,000					
	Recruited cases (n = 276)			**Recruited and non-recruited cases***		
Age	Men	Women	Total	Men	Women	Total
18–29	11.6	33.5	22.3	22.4	46.8	34.4
30–39	7.6	20.8	14.0	13.8	31.8	22.5
40–49	12.7	23.0	17.8	18.5	34.8	26.6
50–59	25.5	22.3	23.9	32.4	40.3	36.3
60–69	30.7	36.4	33.7	44.6	51.0	47.9
70–79	19.8	21.2	20.6	29.2	30.3	29.7
80+	21.2	11.1	14.6	13.4	37.2	21.5
Race						
White	15.7	25.4	20.6	26.8	37.5	32.2
African-American	19.2	54.6	36.4	25.2	61.2	42.8
Asian-American	9.1	9.3	9.2	11.9	17.8	15.0
Other	19.8	28.2	24.1	21.3	34.3	27.9
Total	15.3	25.0	20.2	24.9	37.2	30.6

B. Narcolepsy with cataplexy

	Prevalence per 100,000					
	Recruited cases (n = 185)			Recruited and non-recruited cases*		
Age	Men	Women	Total	Men	Women	Total
18–29	7.8	24.8	16.1	17.8	34.6	26.1
30–39	6.3	12.7	9.4	11.6	22.3	16.8
40–49	5.4	16.2	10.8	9.5	26.7	18.0
50–59	17.0	15.8	16.4	23.9	28.1	26.0
60–69	19.2	27.3	23.4	28.6	37.7	33.3
70–79	8.5	14.8	12.1	15.3	20.6	18.3
80+	15.9	5.6	9.1	6.4	25.5	13.0

A. Narcolepsy with or without cataplexy

	Prevalence per 100,000					
	Recruited cases (n = 276)			Recruited and non-recruited cases*		
Race						
White	9.6	17.1	13.4	18.3	27.0	22.7
African-American	13.7	43.2	28.0	18.4	46.5	32.1
Asian-American	5.2	8.1	6.7	7.8	14.6	11.4
Other	14.8	23.5	19.3	16.3	25.8	21.2
Total	9.5	17.5	13.5	17.0	27.2	21.8

C. Narcolepsy with HLA-DQB1*0602

	Prevalence per 100,000					
	Recruited cases (n =138)			Recruited and non-recruited cases*		
Age	Men	Women	Total	Men	Women	Total
18–29	5.2	14.7	9.9	10.9	20.7	15.7
30–39	3.2	8.7	5.9	6.3	14.5	10.3
40–49	3.4	10.1	6.7	6.3	15.7	11.0
50–59	17.9	13.9	16.9	18.6	25.9	22.2
60–69	17.3	20.0	18.7	25.0	26.0	25.5
70–79	8.5	12.7	10.9	14.7	17.2	16.1
80+	15.9	11.1	12.8	12.5	27.1	17.5
Race						
White	8.0	12.5	10.3	13.9	18.1	16.0
African-American	13.7	37.3	25.2	18.1	40.5	29.0
Asian-American	2.6	3.5	3.1	3.9	8.0	6.1
Other	9.9	4.7	7.2	8.9	11.4	10.1
Total	7.7	12.4	10.1	12.9	18.3	15.3

*Prevalence based on recruited and non-recruited cases using multiple imputation to generate 10 data sets from which an average was calculated. See Methods for details.