Prevalence of Nontuberculous Mycobacterial Lung Disease in U.S. Medicare Beneficiaries

Jennifer Adjemian¹, Kenneth N. Olivier¹, Amy E. Seitz¹, Steven M. Holland¹, and D. Rebecca Prevots¹

¹Laboratory of Clinical Infectious Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD

Rationale: Pulmonary nontuberculous mycobacteria (PNTM) are an important cause of morbidity among older adults in the United States, but national prevalence estimates are lacking.

Objectives: To describe the prevalence and trends of PNTM disease among adults aged 65 years or older throughout the United States.

Methods: A nationally representative 5% sample of Medicare Part B beneficiaries was analyzed from 1997 to 2007. Demographic and medical claims data were compiled and prevalence estimates for PNTM and selected comorbidities were calculated and trends over time evaluated. Logistic regression was used to identify demographic and geographic factors associated with PNTM.

Measurements and Main Results: From 1997 to 2007, the annual prevalence significantly increased from 20 to 47 cases/100,000 persons, or 8.2% per year. The period prevalence was 112 cases/100,000 persons, although prevalence was twofold higher among Asians/Pacific Islanders than among whites (228 vs. 116 cases/100,000 persons). Western states had the highest period prevalence at 149 cases/100,000 persons, with Hawaii having the highest prevalence at 396 cases/100,000 persons, followed by southeastern states, which had a period prevalence of 131 cases/100,000 persons. PNTM cases had more comorbid conditions than noncases and were 40% more likely to die than noncases. Women were 1.4 times more likely to be a PNTM case than men. Relative to whites, Asians/Pacific Islanders were twice as likely to be a case, whereas blacks were half as likely.

Conclusions: The prevalence of PNTM is increasing across all regions of the United States and among both men and women. Significant racial/ethnic and geographic differences suggest important gene-environment interactions.

Keywords: nontuberculous mycobacteria; pulmonary; epidemiology; prevalence; United States

Nontuberculous mycobacteria (NTM) have been increasingly recognized as an important cause of morbidity in the United States. NTM are ubiquitous in the environment, frequently detected in soil and water samples as well as in biofilms that form in municipal water sources (1). Exposure is typically believed to originate from exposure to environmental sources (2). Although severe infection can affect the lymph nodes, skin, soft tissues, bones, and joints, the vast majority of disease is pulmonary (3). More than 140 NTM species have been identified, although approximately 80% of pulmonary NTM (PNTM) infections in

AT A GLANCE COMMENTARY

Scientific Knowledge on the Subject

Recent studies suggest that the prevalence of pulmonary nontuberculous mycobacteria (PNTM) disease is increasing throughout the United States, especially among older adults; however, disease prevalence estimates vary widely by geographic region.

What This Study Adds to the Field

Using a data set of 2.3 million Medicare beneficiaries, this study found that the prevalence of PNTM is increasing significantly across all regions of the United States and among both men and women. Additionally, significant racial/ethnic differences exist that are independent of region.

the United States are associated with *Mycobacterium avium* complex, and prevalence varies by geographic region (4).

Recent studies suggest that the prevalence of PNTM disease is increasing throughout the United States (4, 5), especially among older adults (4, 5). Women appear to bear a disproportionate burden of PNTM disease (4, 6), with several studies identifying physical correlates of risk for PNTM (7). Although the increased risk of PNTM among older adults is well established, disease prevalence estimates vary widely by geographic region. Characterizing PNTM disease distribution by region, sex, age, and underlying or preexisting condition is essential to developing a pathophysiologic understanding of disease. Therefore, we sought to clarify the prevalence and trends of PNTM disease among adults aged 65 years or older in the United States by analyzing national Medicare claims data. Some preliminary results from this study were previously reported in the form of an abstract (8).

METHODS

Data were compiled from the Centers for Medicare and Medicaid's Carrier Standard Analytic File using a 5% sample of all Medicare Part B beneficiaries enrolled from 1997 to 2007. Beneficiaries were excluded if they were less than 65 years old, enrolled in a health maintenance organization (HMO), enrolled in Medicare Part B for less than 1 month, or resided outside of the United States. For each individual, sex, self-reported race/ethnicity, age, and county and state of residence were available. Individuals with race listed as "unknown" were excluded from analyses by race/ethnicity. Demographic data were summarized and compared with the general population using year 2000 U.S. Census Bureau data. Any deaths that occurred were also recorded.

All medical claims submitted for beneficiaries from noninstitutional outpatient care providers were scanned for the *International Classification of Diseases, 9th revision* (ICD-9) code associated with PNTM disease (031.0). Claims were also evaluated for the most frequently occurring comorbidities and for others known to be associated with PNTM. The occurrence of ICD-9 codes on claims was used to estimate the prevalence of PNTM disease and of comorbidities for individuals with and without PNTM. PNTM disease prevalence estimates and 95% confidence intervals (CIs) were calculated by dividing the total number of unique individuals for whom a PNTM-associated claim was submitted

(Received in original form November 16, 2011; accepted in final form January 19, 2012)

Supported by the Division of Intramural Research, National Institute of Allergy and Infectious Diseases.

Author Contributions: Study concept and design: J.A., K.O., D.R.P.; acquisition of data: J.A., A.E.S., D.R.P., S.M.H.; analysis and interpretation of data: J.A., K.O., A.E.S., D.R.P.; drafting of the manuscript: J.A., D.R.P.; critical revision of the manuscript for important intellectual content: J.A., K.O., A.E.S., S.M.H., D.R.P.; statistical analysis: J.A., D.R.P.; study supervision: D.R.P.

Correspondence and requests for reprints should be addressed to Jennifer Adjemian, Ph.D., Qrts 15 B-1, 8 West Drive, MSC 2665, Bethesda, MD 20892-2665. E-mail: jennifer.adjemian@nih.gov

Am J Respir Crit Care Med Vol 185, Iss. 8, pp 881–886, Apr 15, 2012

Published 2012 by the American Thoracic Society

Originally Published in Press as DOI: 10.1164/rccm.201111-2016OC on February 3, 2012

Internet address: www.atsjournals.org

(termed a PNTM case) by the total number of beneficiaries during the time period evaluated. Annual prevalence (AP) estimates were calculated for each year and period prevalence (PP) estimates for the total 11-year time period. Prevalence estimates were stratified by sex, race/ethnicity, age group, state of residence, and geographic region, and were mapped by states using ArcGIS Desktop 9.3.1 (Environmental Systems Research Institute, Inc., Redlands, CA).

Poisson regression models with allowance for overdispersion were used to calculate the annual percent change in prevalence over time. Logistic regression was used to estimate the risk of death in PNTM cases and to identify significant demographic and geographic factors associated with being a PNTM case. Racial/ethnic groups with 20 or fewer PNTM cases were excluded from these analyses. Variables with significant ($P < 0.05$) odds ratios (OR) in univariate regression analyses were included in a multivariate model built using backward stepwise selection procedures to calculate adjusted odds ratios (AOR) (9). Statistical analyses were conducted using SAS 9.2 (SAS Institute Inc., Cary, NC).

RESULTS

Medicare Part B Population

Nearly 2.3 million individuals were included from the sample of Medicare Part B beneficiaries. The demographic and geographic distribution of this sample did not differ from that of the general population (10, 11). Fifty-eight percent of beneficiaries were women, and the annual average age distribution was as follows: 50% were aged 65 to 74 years, 36% aged 75 to 84 years, and 13% aged 85 years or older. Approximately 86.7% of beneficiaries were white, 8.4% black, 1.7% Hispanic, 1.6% Asian/Pacific Islander, and 0.3% North American Native; race was unspecified for 1.2%. Beneficiaries came from all 50 states and the District of Columbia.

PNTM PP

Overall, 2,548 cases were identified from all 50 states and the District of Columbia (Figure 1). Cases were 65% women and 90% white. The overall PP was 112 (95% CI, 108–116) cases/100,000 persons, and was higher among women than men (127 [95% CI, 121–133] vs. 93 [95% CI, 86–98] cases/100,000 persons). Prevalence also varied by race/ethnicity (Figure 2). Asians/Pacific Islanders had a twofold increased prevalence relative to whites (228 [95% CI, 179–277] vs. 116 [95% CI, 111–121] cases/100,000 persons). Among Asians/Pacific Islanders, men had a higher prevalence than

women (263 [95% CI, 182–343] vs. 201 [95% CI, 141–262] cases/100,000 persons), whereas among whites, women had a higher prevalence than men (135 [95% CI, 129–142] vs. 89 [95% CI, 44–135] cases/100,000 persons). Among blacks, the PP of men was twofold that for black women (79 [95% CI, 59–98] vs. 40 [95% CI, 28–51] cases/100,000 persons).

Prevalence also varied geographically (Figure 1). One-third of PNTM cases resided in the Southeast, which had an overall PP of 131 (95% CI, 122–140) cases/100,000 persons. However, the highest PP was observed in the West at 149 (95% CI, 136–163) cases/100,000 persons. This was largely related to the very high PP in Hawaii (396 [95% CI, 170–211] cases/100,000 persons) and California (191 [95% CI, 170–211] cases/100,000 persons), which combined included 67% of Asian/Pacific Islander PNTM cases. When Asians/Pacific Islanders were excluded from analyses for these states, PP estimates remained high, though they dropped to 205 (95% CI, 166–485) and 168 (95% CI, 164–206) cases/100,000 persons for Hawaii and California, respectively. The Midwest had the lowest PP at 78 (95% CI, 71–86) cases/100,000 persons.

AP. The average AP from 1997 to 2007 was 31 (95% CI, 28–34) cases/100,000 persons. During this time period, the AP increased significantly by 8.2% (95% CI, 7.2–9.2) per year, from 20 (95% CI, 18–23) to 47 (95% CI, 44–51) cases/100,000 persons (Figure 3). The annual percent change for women was greater than for men (9.1% [95% CI, 6.5–11.7%] vs. 6.4% [95% CI, 3.7–9.2%] per year, respectively). All regions also showed a significant increase in prevalence over time, ranging from 1.7% (95% CI, 1.0–3.8%) per year in the West to 11.9% (95% CI, 9.5–14.8%) per year in the Northeast.

Comorbidity analysis. The majority of comorbidities evaluated were more frequent among PNTM cases than noncases, especially bronchiectasis, which occurred in 44% of PNTM cases compared with 1% of noncases (Table 1). On average, PNTM cases had four comorbidities, whereas noncases averaged only two. When evaluated by sex, among PNTM cases, 1.5 and 1.7 times more women had claims for rheumatoid arthritis and bronchiectasis, respectively, than men, whereas 1.5 times more men had claims for diabetes and lung cancer than women. The prevalence of chronic obstructive pulmonary disease (COPD), which included ICD-9 codes for COPD, emphysema, and chronic

Figure 1. Prevalence of pulmonary nontuberculous mycobacteria cases among a sample of U.S. Medicare Part B enrollees aged 65 and older, 1997 to 2007. NTM = nontuberculous mycobacteria.

Figure 2. Period prevalence of pulmonary nontuberculous mycobacteria cases among a sample of U.S. Medicare Part B enrollees aged 65 and older from 1997 to 2007 by sex and race/ethnicity. PNTM = pulmonary nontuberculous mycobacteria.

bronchitis, was 87% among PNTM cases and 44% among noncases; estimates did not vary significantly by sex or region. ICD-9 codes for pulmonary tuberculosis were also identified among 3.6% of the PNTM cases and 0.09% of noncases.

Mortality analysis. PNTM cases were 40% more likely to die over the period of study than noncases (OR, 1.4; 95% CI, 1.3–1.6). Among PNTM cases, the risk of death was greater in men (OR, 1.8; 95% CI, 1.6–2.2) than women, and relative to whites, was greater among blacks (OR, 2.1; 95% CI, 1.4–3.1) and Hispanics (OR, 2.4; 95% CI, 1.2–4.7). PNTM cases were more likely to die if they had three or more comorbidities (OR, 1.3; 95% CI, 1.0–1.6) or if they also had lung cancer (OR, 1.7; 95% CI, 1.4–2.1, asthma (OR, 1.7; 95% CI, 1.1–2.7), or pneumonia (OR, 2.9; 95% CI, 2.3–3.6). In contrast, PNTM cases with bronchiectasis, regardless of other comorbidities reported, were half as likely to die (OR, 0.5; 95% CI, 0.4–0.6) than cases without this diagnosis.

Demographic/geographic risk factor analysis. Significant regional differences in prevalence were observed, with the West (AOR, 1.8; 95% CI, 1.6–2.1) and Southeast (AOR, 1.8; 95% CI, 1.6–2.0) at similarly higher risk relative to the Midwest, followed closely by the Southwest (AOR, 1.7; 95% CI, 1.5–2.0). Women were 40% more likely to be PNTM cases than men (OR, 1.4; 95% CI, 1.3–1.5), and relative to whites, Asians/Pacific Islanders were twice as likely to be cases (OR, 2.0; 95% CI, 1.6–

2.5), whereas blacks were half as likely to be cases (OR, 0.5; 95% CI, 0.4–0.6). In a multivariate model looking at both sex and race/ethnicity, a significant interaction was observed ($P < 0.0001$): among both men and women, Asians/Pacific Islanders were more likely to have PNTM than whites, although the risk was greater among men (AOR, 2.9; 95% CI, 2.2–4.0) than women (AOR, 1.5; 95% CI, 1.1–2.0). When region was included in the multivariate model, Asian/Pacific Islander men remained at significantly greater risk for PNTM than white men (AOR, 2.6; 95% CI, 1.9–3.6). Although an increased risk among women was still observed, it was no longer statistically significant (AOR, 1.3; 95% CI, 0.95–1.8).

DISCUSSION

Using a nationally representative population-based sample of Medicare beneficiaries, we found that the prevalence of PNTM, as estimated from diagnostic codes on medical claims, is increasing significantly across all regions of the United States and among both men and women. Additionally, we identified significant racial/ethnic differences that are independent of region. Although other studies have estimated the prevalence of PNTM within specific geographic areas in the United States (4–6), this is the first study to provide nationally representative estimates among older adults from all 50 states plus the District of Columbia and across various racial/ethnic groups. The ability to

Figure 3. Annual prevalence of pulmonary nontuberculous mycobacteria cases among a sample of U.S. Medicare Part B enrollees by sex from 1997 to 2007. PNTM = pulmonary nontuberculous mycobacteria.

TABLE 1. FREQUENCY OF SELECTED MEDICAL CONDITIONS REPORTED THROUGH MEDICARE CLAIMS AMONG PULMONARY NONTUBERCULOUS MYCOBACTERIA AND NON-PULMONARY NONTUBERCULOUS MYCOBACTERIA CASES FROM A SAMPLE OF U.S. MEDICARE PART B ENROLLEES FROM 1997–2007

Medical Condition	ICD-9 Code(s)	PNTM Cases (%)	Non-PNTM Cases (%)	Relative Prevalence
Pneumonia	480 (480.0–480.3, 480.8, 480.9), 481, 482 (482.0–482.32, 482.4–482.42, 482.49, 482.8, 482.9), 483 (483.0, 483.1, 483.8), 484 (484.1, 484.3, 484.5–484.8), 485–487 (487.0, 487.1, 487.8)	82	33	2.5
Acute bronchitis	466 (466.0, 466.1, 466.11, 466.19)	66	32	2.1
Bronchiectasis	494 (494.0, 494.1)	44	1	44.0
Mitral valve disorder	424.0	37	21	1.8
Diabetes mellitus	250.00–250.03, 250.09	26	29	0.9
Cancer of the trachea, bronchus, and lung	162 (162.0, 162.2–162.5, 162.8, 162.9)	17	5	3.4
Pulmonary hypertension	416.8	7	2	3.5
Rheumatoid arthritis	714, 714.0	13	6	2.2
Asthma	493 (493.0–493.2, 493.8, 493.81, 493.82, 493.9)	4	1	4.0
Aspergillosis	117.3	2	0.1	20
Anomalies of the larynx, trachea, and bronchus	748.3	0.4	0.05	8.0
HIV	042	0	0.0004	0.0

Definition of abbreviations: ICD-9 = International Classification of Diseases, 9th revision; PNTM = pulmonary nontuberculous mycobacteria.

define the burden of PNTM across an 11-year period is important given the often chronic nature of this disease (12, 13).

Although clinic-based reports have suggested that the prevalence of PNTM has increased (14), few population-based studies have documented this (4, 15). du Moulin and colleagues (15) demonstrated a fivefold increase in the number of patients with *M. avium* complex isolates in Massachusetts from 1972 to 1983, suggesting that this trend of an increasing prevalence has been occurring before our study period, at least in some areas. More recently, an isolate-based study in four HMOs using American Thoracic Society (ATS) microbiologic criteria found an average annual prevalence of 30 and 23 cases/100,000 persons in women and men, respectively, which is similar to our average annual prevalence among women and men of 37 and 23 cases/100,000 persons, respectively (4). Although some individuals in our data set may have received a PNTM ICD-9 code without having met ATS criteria, our results most likely represent an underestimate of true disease prevalence, because using ICD-9 codes to identify PNTM cases meeting ATS criteria has been shown to miss approximately 25 to 75% of cases (4, 16). However, because NTM is a rare disease, those who do receive this diagnostic code are more likely to have clinical evidence of a pathogenic NTM species. Furthermore, using ICD-9 codes, which have been validated in other settings, allows for the estimation of prevalence in populations numbering millions of people, numbers that would not be feasible to study in depth in smaller clinical studies. Thus, despite the potential for some misclassification and underestimation of prevalence, ICD9 codes represent a critical tool for large-scale population studies of rare conditions. Outside the United States, increases in PNTM disease have also been noted in several Australian (17–19) and Asian studies (20–23). One Canadian study identified an annual increase in the isolation prevalence of PNTM of 8.4% per year from 1997 to 2003, which is consistent with our findings (24).

It is also important to note that the prevalence of PNTM in the United States exceeds that of tuberculosis (4), which has been decreasing in incidence in several major U.S. cities from 2000 to 2007 by 4 to 21% per year (25). It has been postulated that the decrease in tuberculosis may be contributing to the increase observed in NTM, as prior tuberculosis infection may possibly stimulate antimycobacterial immunity (26). Although 3.6% of the PNTM cases identified here also had an ICD-9 code for pulmonary tuberculosis, the use of diagnostic codes for this

condition is considered to be highly unreliable, with a very low positive predictive value (27). This is because a patient suspected to have active tuberculosis may have been coded as such before the disease was confirmed; the patient may have been evaluated for a latent tuberculosis infection; the patient had a history of treated tuberculosis; or the patient was actually infected with another species of *Mycobacterium* (27), as might have often been the case here.

The increase in PNTM may represent a true increase in disease or a combination of factors, including improved diagnostics and heightened awareness for NTM. For instance, improved mycobacterial diagnostics and increased use of imaging technology, such as computed tomography scans (28), may have resulted in more accurate detection and increased diagnosis of NTM infections among patients. Still, increases in NTM isolation, sensitization, and disease prevalence have been noted worldwide and throughout various points in time (4, 17–24), suggesting that at least some of the observed increase is likely true and beyond the contribution of potential surveillance and diagnostic biases. Numerous hypotheses have been advanced for the increase in NTM prevalence, such as the Clean Water Act in 1970, which led to widespread disinfection of drinking water that may have increased NTM prevalence by selection due to increased resistance to chlorination relative to competing microorganisms (1). Additionally, therapies that inhibit tumor necrosis factor- α (TNF- α), a cytokine that mediates host immune defense against opportunistic pathogens, have become widely used within the last decade to treat autoimmune inflammatory conditions such as rheumatoid arthritis, increasing the risk of NTM infection in these patients and perhaps contributing to its increasing prevalence (16, 29). However, TNF- α inhibitor exposure is unlikely to account for the overall increases that we found.

Prior clinic-based studies in the United States have identified an increase in PNTM among white women with no identified risk factors (7). Although more than 90% of the PNTM cases in this study were white, Asians/Pacific Islanders were at significantly greater risk for disease, with a prevalence twofold that of whites. Additionally, whereas white women had a 50% higher prevalence than white men, among Asians/Pacific Islanders, men were more affected than women. In fact, Asian/Pacific Islander men were twice as likely as white men to have PNTM. The difference in risk between Asian/Pacific Islander men and women is notable, and may be attributed to behavioral or cultural factors associated with

increased risk, such as frequent tobacco use, which among Asian/Pacific Islanders is higher in men than women (30, 31).

Assessing the role of race/ethnicity in disease can be complex, as racial/ethnic categorizations may be a surrogate measure for behavioral, cultural, and biological factors. Furthermore, the lack of clearly defined and consistently applied categorizations for racial/ethnic groups limits the ability to compare results across studies (32). In particular, the Asian/Pacific Islander category comprises a variety of subgroups that have significant health-related and demographic differences. Pacific Islanders are more likely than Asian Americans to be smokers, overweight, have lower income and education levels, and have higher chronic disease and mortality rates, despite no differences reported in access to care (33). It is also possible that some subgroups within the Asian/Pacific Islander population may share a common genetic ancestry and have increased susceptibility to PNTM disease. For instance, among patients in Japan with pulmonary *M. avium* complex infection, human leukocyte antigen-A33 and human leukocyte antigen-DR6 antigens, which have been linked to a role in host-defense mechanisms, were significantly more frequent than in the control population (34). PNTM disease dynamics are most likely driven by complex interactions among behavioral, cultural, genetic, and environmental factors.

In this study, the majority of Asian/Pacific Islander PNTM cases resided in Hawaii, which also had the highest PP at 396 cases/100,000 persons. However, even when Asians/Pacific Islanders were excluded, Hawaii still had the highest PP in the United States, suggesting that the regional exposure was dominant over the ethnic issues. Similarly, in 1979, a nationwide survey of state public health laboratories found Hawaii had the highest isolation rate of *M. avium* complex in the United States (35). Environmental conditions unique to Hawaii may contribute to greater levels of NTM exposure. For example, the soils of Hawaii are high in polycyclic aromatic hydrocarbons, environmental contaminants produced by fossil fuel combustion and human industry, but also by volcanic activity. Some mycobacteria have the unique property of being able to readily degrade polycyclic aromatic hydrocarbons, enabling them to thrive in this environment (36).

The higher prevalence of disease among older adults living in southeastern and western states is consistent with prior reports of higher human exposure and observed prevalence (5, 37, 38) and with studies isolating greater numbers of mycobacteria from southeastern states than from other locations surveyed (39). Although regional variation in Medicare practices relevant to PNTM may exist, the strength of our findings indicates that these results are unlikely to be explained by differences in regional practices alone. PNTM is prevalent throughout the United States, and exposure may often occur through drinking water distribution systems (40). Households with average water heater temperatures 50°C or lower have higher rates of NTM recovery, but it is unclear whether their inhabitants are at higher risk (41). Therefore, regional factors, individual behaviors, and local exposures likely contribute to the true risk of infection.

PNTM cases identified here had more reported comorbidities than noncases. Almost all PNTM cases were diagnosed with at least one other lung-associated condition, including 44% with bronchiectasis, an otherwise uncommon condition. This frequency is much higher than what was previously reported. Among Oregon residents with PNTM, only 16% had bronchiectasis (42), and in a study of non-AIDS PNTM-associated hospitalizations, 15% also had bronchiectasis (5), although these studies both included patients less than 65 years old. Among patients in Canada more than 56 years old meeting ATS criteria for NTM infection, 62% had nodular bronchiectasis (43). Although bronchiectasis may increase susceptibility to NTM infection, infection with NTM likely also exacerbates bronchiectasis. Similarly, rheumatoid arthritis, which was twice as prevalent among PNTM cases as noncases,

especially among women, may be associated with NTM infection, and this association may be increased by anti-TNF- α therapies (29). COPD is associated with PNTM, but this relationship could not be adequately assessed here due to identified challenges associated with its definition and subsequent coding (44). COPD is typically defined to include emphysema and chronic bronchitis, though other conditions, such as asthma and bronchiectasis, are also sometimes categorized as COPD by physicians (44) and may have been coded as such in some Medicare claims analyzed here, resulting in inflated estimates.

PNTM cases had higher mortality, especially among men and in those with greater numbers of comorbidities. Surprisingly, PNTM cases with bronchiectasis were half as likely to die as those without it. It is possible that those patients without bronchiectasis may be more likely to have fibrocavitary disease, which has been noted to have a worse prognosis (45). Black and Hispanic PNTM cases were more than twice as likely to die as whites, which may reflect racial/ethnic differences in the use of services among Medicare beneficiaries (46). Elderly white Medicare beneficiaries were more likely than blacks and Hispanics to receive care and seek out more services (46–48), regardless of urban or rural residence (46). Whites were also more likely to seek out care from private physicians, whereas blacks more frequently used community health centers (46), possibly resulting in barriers to access of specialized care, which may be more difficult to obtain in low-income areas. This might also account for the higher observed prevalence of bronchiectasis and PNTM in whites compared with blacks and Hispanics, as well as the lower death rate among those with a diagnosis of bronchiectasis, as whites might be more likely to obtain specialized testing, such as computerized tomography scans or X-rays, and to receive more intensive care and follow-up. In contrast, differences in use of Medicare services between whites and Asians/Pacific Islanders have not been consistently demonstrated (48–52).

Because PNTM is not a nationally notifiable disease, systematic monitoring of temporal trends for disease across all regions is difficult. However, with PNTM increasing among adults aged 65 years or older, a population that is expected to nearly double by 2030 (11), it is critical that this condition is recognized as an important public health issue with potentially significant consequences for affected patients.

Author disclosures are available with the text of this article at www.atsjournals.org.

Acknowledgment: The authors thank the Research Data Assistance Center (ResDAC), University of Minnesota, for supportive assistance on Medicare data collection methods.

References

1. Falkingham JO. Impact of human activities on the ecology of nontuberculous mycobacteria. *Future Microbiol* 2010;5:951–960.
2. Mangione EJ, Huitt G, Lenaway D, Beebe J, Bailey A, Figoski M, Rau MP, Albrecht KD, Yakus MA. Nontuberculous mycobacterial disease following hot tub exposure. *Emerg Infect Dis* 2001;7:1039–1042.
3. Tortoli E. Clinical manifestations of nontuberculous mycobacteria infections. *Clin Microbiol Infect* 2009;15:906–910.
4. Prevots DR, Shaw PA, Strickland D, Jackson LA, Raebel MA, Blosky MA, Montes de Oca R, Shea YR, Seitz AE, Holland SM, et al. Nontuberculous mycobacterial lung disease prevalence at four integrated healthcare delivery systems. *Am J Respir Crit Care Med* 2010;182:970–976.
5. Billinger ME, Olivier KN, Viboud C, de Oca RM, Steiner C, Holland SM, Prevots DR. Nontuberculous mycobacteria-associated lung disease in hospitalized persons, United States, 1998–2005. *Emerg Infect Dis* 2009;15:1562–1569.
6. Bodle EE, Cunningham JA, Della-Latta P, Schluger NW, Saiman L. Epidemiology of nontuberculous mycobacteria in patients without HIV infection, New York City. *Emerg Infect Dis* 2008;14:390–396.
7. Kim RD, Greenberg DE, Ehrmantraut ME, Guide SV, Ding L, Shea Y, Brown MR, Chernick M, Steagall WK, Glasgow CG, et al. Pulmonary

- nontuberculous mycobacterial disease: prospective study of a distinct preexisting syndrome. *Am J Respir Crit Care Med* 2008;178:1066–1074.
8. Adjemian J, Olivier K, Seitz A, Holland S, Prevots R. Prevalence of pulmonary nontuberculous mycobacterial infections among U.S. Medicare beneficiaries, 1997–2007. Presented at the Annual Meeting of the American Thoracic Society, May, 2011, Denver, CO.
 9. Hosmer DW, Lemeshow S. Applied logistic regression. New York: John Wiley & Sons; 2000.
 10. United States Census Bureau. Table DP-1. Profile of general demographic characteristics: 2000. Geographic area: United States. Washington, DC: United States Census Bureau; 2000.
 11. Colello K. Where do older Americans live? Geographic distribution of the older population. Washington, DC: Domestic Social Policy Division, Congressional Research Service; 2007.
 12. Field SK, Cowie RL. Lung disease due to the more common nontuberculous mycobacteria. *Chest* 2006;129:1653–1672.
 13. Field SK, Fisher D, Cowie RL. Mycobacterium avium complex pulmonary disease in patients without HIV infection. *Chest* 2004;126:566–581.
 14. Griffith DE, Aksamit T, Brown-Elliott BA, Catanzaro A, Daley C, Gordin F, Holland SM, Horsburgh R, Huitt G, Iademarco MF, et al. An official ATS/IDSA statement: diagnosis, treatment, and prevention of nontuberculous mycobacterial diseases. *Am J Respir Crit Care Med* 2007;175:367–416.
 15. du Moulin GC, Sherman IH, Hoaglin DC, Stottmeier KD. Mycobacterium avium complex, an emerging pathogen in Massachusetts. *J Clin Microbiol* 1985;22:9–12.
 16. Winthrop KL, Baxter R, Liu L, McFarland B, Austin D, Varley C, Radcliffe L, Suhler E, Choi D, Herrinton LJ. The reliability of diagnostic coding and laboratory data to identify tuberculosis and nontuberculous mycobacterial disease among rheumatoid arthritis patients using anti-tumor necrosis factor therapy. *Pharmacoepidemiol Drug Saf* 2011;20:229–235.
 17. Thomson RM. Changing epidemiology of pulmonary nontuberculous mycobacteria infections. *Emerg Infect Dis* 2010;16:1576–1583.
 18. O'Brien DP, Currie BJ, Krause VL. Nontuberculous mycobacterial disease in northern Australia: a case series and review of the literature. *Clin Infect Dis* 2000;31:958–967.
 19. Pang SC. Mycobacterium kansasii infections in Western Australia (1962–1987). *Respir Med* 1991;85:213–218.
 20. Wang HX, Yue J, Han M, Yang JH, Gao RL, Jing LJ, Yang SS, Zhao YL. Nontuberculous mycobacteria: susceptibility pattern and prevalence rate in Shanghai from 2005 to 2008. *Chin Med J (Engl)* 2010;123:184–187.
 21. Ding LW, Lai CC, Lee LN, Hsueh PR. Disease caused by nontuberculous mycobacteria in a university hospital in Taiwan, 1997–2003. *Epidemiol Infect* 2006;134:1060–1067.
 22. Sakatani M. [The epidemiology of pulmonary disease caused by mycobacterium avium complex in Japan]. *Kekkaku* 1993;68:43–46.
 23. Park YS, Lee CH, Lee SM, Yang SC, Yoo CG, Kim YW, Han SK, Shim YS, Yim JJ. Rapid increase of non-tuberculous mycobacterial lung diseases at a tertiary referral hospital in South Korea. *Int J Tuberc Lung Dis* 2010;14:1069–1071.
 24. Marras TK, Chedore P, Ying AM, Jamieson F. Isolation prevalence of pulmonary non-tuberculous mycobacteria in Ontario, 1997–2003. *Thorax* 2007;62:661–666.
 25. Oren E, Winston CA, Pratt R, Robison VA, Narita M. Epidemiology of urban tuberculosis in the United States, 2000–2007. *Am J Public Health* 2011;101:1256–1263.
 26. Horsburgh CR, Hanson DL, Jones JL, Thompson SE. Protection from Mycobacterium avium complex disease in human immunodeficiency virus-infected persons with a history of tuberculosis. *J Infect Dis* 1996;174:1212–1217.
 27. Sickbert-Bennett EE, Weber DJ, Poole C, MacDonald PD, Maillard JM. Utility of International Classification of Diseases, Ninth Revision, Clinical Modification codes for communicable disease surveillance. *Am J Epidemiol* 2010;172:1299–1305.
 28. Seitz A, Olivier K, Adjemian J, Holland S, Prevots R. Trends in bronchiectasis among medicare beneficiaries in the United States, 2000–2007. *Chest* (In press)
 29. Winthrop KL, Chang E, Yamashita S, Iademarco MF, LoBue PA. Nontuberculous mycobacteria infections and anti-tumor necrosis factor- α therapy. *Emerg Infect Dis* 2009;15:1556–1561.
 30. Centers for Disease Control and Prevention. Cigarette smoking among adults—United States, 2002. *MMWR Morb Mortal Wkly Rep* 2004;53:427–431.
 31. Centers for Disease Control and Prevention. From the Center for Disease Control and Prevention. Cigarette smoking among adults—United States, 1998. *JAMA* 2000;284:2180–2181.
 32. Centers for Disease Control and Prevention. Use of race and ethnicity in public health surveillance: summary of the CDC/ATSDR workshop. *MMWR Recomm Rep* 1993;42:1–16.
 33. Bitton A, Zaslavsky AM, Ayanian JZ. Health risks, chronic diseases, and access to care among us pacific islanders. *J Gen Intern Med* 2010;25:435–440.
 34. Takahashi M, Ishizaka A, Nakamura H, Kobayashi K, Nakamura M, Namiki M, Sekita T, Okajima S. Specific HLA in pulmonary MAC infection in a Japanese population. *Am J Respir Crit Care Med* 2000;162:316–318.
 35. Good RC. From the Center for Disease Control. Isolation of nontuberculous mycobacteria in the United States, 1979. *J Infect Dis* 1980;142:779–783.
 36. Hennessee CT, Seo JS, Alvarez AM, Li QX. Polycyclic aromatic hydrocarbon-degrading species isolated from Hawaiian soils: Mycobacterium crocinum sp. nov., mycobacterium pallens sp. nov., mycobacterium rutilum sp. nov., mycobacterium rufum sp. nov. And mycobacterium aromaticivorans sp. nov. *Int J Syst Evol Microbiol* 2009;59:378–387.
 37. Edwards LB, Acquaviva FA, Livesay VT, Cross FW, Palmer CE. An atlas of sensitivity to tuberculin, PPD-B, and histoplasmin in the United States. *Am Rev Respir Dis* 1969;99:1–132.
 38. Olivier KN, Weber DJ, Wallace RJ Jr, Faiz AR, Lee JH, Zhang Y, Brown-Elliott BA, Handler A, Wilson RW, Schechter MS, et al. Nontuberculous mycobacteria. I: Multicenter prevalence study in cystic fibrosis. *Am J Respir Crit Care Med* 2003;167:828–834.
 39. Kirschner RA Jr, Parker BC, Falkinham JO 3rd. Epidemiology of infection by nontuberculous mycobacteria. Mycobacterium avium, Mycobacterium intracellulare, and Mycobacterium scrofulaceum in acid, brown-water swamps of the southeastern United States and their association with environmental variables. *Am Rev Respir Dis* 1992;145:271–275.
 40. Falkinham JO 3rd. Nontuberculous mycobacteria in the environment. *Clin Chest Med* 2002;23:529–551.
 41. Falkinham JO 3rd. Nontuberculous mycobacteria from household plumbing of patients with nontuberculous mycobacteria disease. *Emerg Infect Dis* 2011;17:419–424.
 42. Winthrop KL, McNelley E, Kendall B, Marshall-Olson A, Morris C, Cassidy M, Saulson A, Hedberg K. Pulmonary nontuberculous mycobacterial disease prevalence and clinical features: an emerging public health disease. *Am J Respir Crit Care Med* 2010;182:977–982.
 43. Marras TK, Mehta M, Chedore P, May K, Al Houqani M, Jamieson F. Nontuberculous mycobacterial lung infections in Ontario, Canada: clinical and microbiological characteristics. *Lung* 2010;188:289–299.
 44. Mannino DM, Homa DM, Akinbami LJ, Ford ES, Redd SC. Chronic obstructive pulmonary disease surveillance—United States, 1971–2000. *MMWR Surveill Summ* 2002;51:1–16.
 45. Sohn H, Kim HJ, Jung Kwon O, Koh WJ, Shin SJ. High virulent clinical isolates of mycobacterium abscessus from patients with the upper lobe fibrocavitary form of pulmonary disease. *Microb Pathog* 2009;47:321–328.
 46. Escarce JJ, Epstein KR, Colby DC, Schwartz JS. Racial differences in the elderly's use of medical procedures and diagnostic tests. *Am J Public Health* 1993;83:948–954.
 47. Chen LM. Is there disparity in physician service use? A comparison of Hispanic and white medicare beneficiaries. *Health Soc Work* 2010;35:181–190.
 48. Virnig BA, Lurie N, Huang Z, Musgrave D, McBean AM, Dowd B. Racial variation in quality of care among medicare+choice enrollees. *Health Aff (Millwood)* 2002;21:224–230.
 49. Semrad TJ, Tancredi DJ, Baldwin LM, Green P, Fenton JJ. Geographic variation of racial/ethnic disparities in colorectal cancer testing among medicare enrollees. *Cancer* 2011;117:1755–1763.
 50. White A, Vernon SW, Franzini L, Du XL. Racial disparities in colorectal cancer survival: to what extent are racial disparities explained by differences in treatment, tumor characteristics, or hospital characteristics? *Cancer* 2010;116:4622–4631.
 51. Moy E, Greenberg LG, Borsky AE. Community variation: disparities in health care quality between Asian and white medicare beneficiaries. *Health Aff (Millwood)* 2008;27:538–549.
 52. Curtis E, Quale C, Haggstrom D, Smith-Bindman R. Racial and ethnic differences in breast cancer survival: how much is explained by screening, tumor severity, biology, treatment, comorbidities, and demographics? *Cancer* 2008;112:171–180.