Previous studies showed that pandemic (H1N1) 2009 may have become established in swine populations in Canada, Norway, and Hong Kong (*1*,*5*–*8*). The human-to-pig transmission of pandemic (H1N1) 2009 may substantially affect virus evolution and subsequent epidemiology. Although the pandemic was mild, the virus could develop further reassortment in swine and gain virulence. On the other hand, subtype H5N1 and H9N2 viruses have become established in pigs, so the introduction of pandemic (H1N1) 2009 virus to pigs has provided the possibility for the incorporation of avian virus genes into mammalian-adapted viruses. That transmission could occur from humans to pigs and vice versa is especially troublesome. Given the possible production of novel viruses of potential threat to public health, we should emphasize influenza surveillance in pigs and establishment of the genetic basis of the viral genome for rapidly identifying such reassortment events.

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References

1. Pasma T, Joseph T. Pandemic (H1N1) 2009 infection in swine herds, Manitoba, Canada. Emerg Infect Dis. 2010;16:706–8.
2. Itoh Y, Shinya K, Kiso M, Watanabe T, Sakoda Y, Hatta M, et al. In vitro and in vivo characterization of new swine origin H1N1 influenza viruses. Nature. 2009;460:1021–5.
3. Maines TR, Jayaraman A, Belser JA, Wadford DA, Pappas C, Zeng H, et al. Transmission and pathogenesis of swine-origin 2009 A (H1N1) influenza viruses in ferrets and mice. Science. 2009;325:484–7.
4. Tompkins SM, Lo CY, Tumpey TM, Epstein SL. Protection against lethal influenza virus challenge by RNA interference in vivo. Proc Natl Acad Sci U S A. 2004;101:8682–6. doi:10.1073/pnas.0402630101
5. Hofshagen M, Gjerset B, Er C, Tarpai A, Brun E, Dannevig B, et al. Pandemic influenza A (H1N1)v: human to pig transmission in Norway? Euro Surveill. 2009;14:pii:19406.
6. Howden KJ, Brockhoff EJ, Caya FD, McLeod LJ, Lavoie M, Ing JD, et al. An investigation into human pandemic influenza virus (H1N1) 2009 on an Alberta swine farm. Can Vet J. 2009;50:1153–61.
7. Weingartl HM, Berhane Y, Hisanaga T, Neufeld J, Kehler H, Emburry-Hyatt C, et al. Genetic and pathobiologic characterization of pandemic H1N1 2009 influenza viruses from a naturally infected swine herd. J Virol. 2010;84:2245–56. doi:10.1128/JVI.02118-09
8. Vijaykrishna D, Poon LL, Zhu HC, Ma SK, Li OT, Cheung CL, et al. Reassortment of pandemic H1N1/2009 influenza A virus in swine. Science. 2010;328:1529. doi:10.1126/science.1189132

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Pulmonary Disease Associated with Nontuberculous Mycobacteria, Oregon, USA

To the Editor: Nontuberculous mycobacteria (NTM) are environmental organisms ubiquitous in soil and water, including municipal water supplies. When inhaled, these organisms cause chronic, severe lung disease in susceptible persons (*1*). Recent epidemiologic studies suggest NTM pulmonary disease is increasingly prevalent in North America, with annual incidence rates of 13 cases per 100,000 population in persons ≥50 years of age and 2–4-fold higher in older age groups (*2*–*4*). The current distribution of pulmonary NTM disease has been poorly characterized with regard to environment, climate, and other factors.

We recently performed a statewide NTM surveillance project in Oregon, United States, where we documented higher pulmonary disease rates within the moister, temperate western regions of the state. Oregon is bisected north-south by mountains into 2 distinct climate zones. Western Oregon, where 87% of the state's population lives, is temperate and wet; eastern Oregon is primarily rural, with an arid, high desert climate. Our goal was to evaluate whether disease clustering within the state could be explained by population density.

For all Oregon residents who had newly diagnosed and existing pulmonary NTM disease during 2005 and 2006, we used case-patient home ZIP code and county of residence to construct statewide disease maps (*4*). We obtained state ZIP code and county-level census data for 2005 and 2006 from the Portland State University Population Research Center and used Oregon Office of Rural Health criteria to designate

ZIP codes as urban or rural and counties as rural (nonmetropolitan), micropolitan, or metropolitan (*5*,*6*). Unlike ZIP code data, which lacked age information, county census data were age stratified and consisted of population numbers aggregated in 5-year age groups (e.g., 0–4 years, 5–9 years). Because nearly all pulmonary NTM disease occurred in persons ≥50 years of age, we calculated age-adjusted disease prevalence rates (by using 95% Poisson exact confidence intervals) for patients ≥50 years of age in the county census data. We used the Cochran-Armitage test for trend to evaluate differences in rates by rural, micropolitan, and metropolitan county designations.

Statewide, 385 (94%) of 411 NTM cases occurred among residents of western Oregon, and the crude rate of annual disease prevalence was significantly higher in western than in eastern Oregon (6.0 vs. 2.7/100,000; $p<0.05$) (online Appendix Figure, www.cdc.gov/EID/content/17/9/101929-appF.htm). Within the western region, rates were significantly higher in urban than in rural ZIP codes. Using county-level data, we found that age-adjusted prevalence rates in western Oregon strongly correlated with increasing levels of population density (Table). In eastern Oregon, where only 26 cases occurred, age-adjusted rates among residents ≥50 years of age were similar (7.6 cases/100,000 population) to those in rural counties within western Oregon (6.5/100,000).

In Oregon, where most pulmonary NTM disease is caused by *Mycobacterium avium* complex (MAC), our findings suggest that the higher rates of disease in the wet western portion of the state are best explained by differences in population density (*4*). Disease rates there were highly correlated with increasing population density, and in rural areas of western Oregon, disease rates were similar to those in the arid, primarily rural eastern portion of the state.

Humans presumably are exposed to NTM daily through showering, bathing, and other activities where water or soil is aerosolized (*7*). Previous environmental studies suggest that persons living in urban areas could potentially have greater NTM exposure during these activities because NTM is more prevalent in piped networks of municipal water systems than in well-water systems primarily used in rural regions (*8*). A study in Japan in the 1980s found a similar association of pulmonary NTM disease (primarily MAC) with urban and wet environments compared with arid and rural regions in our study but unlike our study was not able to evaluate differences in disease rates between urban and rural areas independent of climate differences (*9*). A 1979 Texas study found an association of pulmonary NTM with rural living, although this result was driven by *M. kansasii* disease, and rates of MAC were actually higher in rural areas (*10*). These and other similar studies were conducted decades ago when the epidemiology of NTM was substantially different

Table. Prevalence of pulmonary nontuberculous mycobacterial disease, by geographic region and population density, Oregon, USA, 2005 and 2006*

Region/population density	Total no. cases	Prevalence† (95% confidence interval)		
		All age groups	Age <50 y	Age ≥50 y
Total	411	5.6 (5.0–6.1)	1.1 (0.8–1.4)	15.2 (13.6–16.9)
County-level analysis				
Western	385	6.0 (5.4–6.6)	1.2 (0.9–1.6)	16.5 (14.8–18.4)
Metropolitan	341	6.3 (5.7–7.0)	1.3 (1.0–1.8)‡	18.0 (16.0–20.2)§¶#
Micropolitan	39	4.5 (3.2–6.1)	0.2 (0.0–1.0)‡	11.1 (7.8–15.2)§¶
Rural	5	3.6 (1.2–8.3)	1.3 (0.0–7.1)	6.5 (1.8–15.9)§#
Eastern	26	2.7 (1.8–4.0)	0.2 (0.0–0.9)	7.6 (4.9–11.2)
Metropolitan	9	3.0 (1.4–5.6)	0.0 (0.0–1.8)	8.6 (3.9–16.3)
Micropolitan	12	2.5 (1.3–4.3)	0.3 (0.0–1.7)	6.8 (3.4–12.2)
Rural	5	2.9 (1.0–6.8)	0.0 (0.0–3.5)	7.8 (2.5–18.2)
ZIP code**				
Western	365	5.7 (5.2–6.4)	–	–
Urban	277	6.4 (5.7–7.2)††	–	–
Rural	88	4.3 (3.5–5.3)††	–	–
Eastern	26	2.8 (1.8–4.0)	–	–
Urban	5	2.7 (0.9–5.7)	–	–
Rural	21	2.8 (1.7–4.1)	–	–

*Annualized 2-year disease period prevalence, Oregon, 2005 and 2006.
†Cases per 100,000 population.
‡Significant differences ($p<0.05$) between western Oregon metropolitan and micropolitan counties, age <50 y.
§West, age ≥50 y, metropolitan, micropolitan, and rural (Cochran-Armitage test for trend, $p<0.01$).
¶Significant differences ($p<0.05$) between western Oregon metropolitan and micropolitan counties, age ≥50 y.
#Significant differences ($p<0.05$) between western Oregon metropolitan and rural counties, age ≥50 y.
**Included only the 391 patients for whom with ZIP code data were available. –, age-level data not available for ZIP codes.
††Significant differences ($p<0.05$) between western Oregon urban and rural ZIP codes.

(i.e., predominantly a disease of male patients) and before the formulation of the 2007 American Thoracic Society/ Infectious Diseases Society of America pulmonary NTM disease criteria (*1*).

We were limited in drawing firm conclusions about why pulmonary NTM is more common in urban areas because we were not able to evaluate patients or regional water systems within our study. Persons living rurally might be less likely to seek medical care and thus have NTM diagnosed, which would account for the differences in our study. However, given the reasonably close proximity of western Oregon's rural regions to major medical centers, we believe this scenario is unlikely.

Our findings suggest that pulmonary NTM disease is closely associated with urban living. We suspect the difference in disease rates between urban and rural areas might reflect differences in host exposure to these pathogens. Further studies should be undertaken to elucidate the environmental exposures associated with pulmonary NTM.

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References

1. Griffith DE, Aksamit T, Brown-Elliott BA, Catanzaro A, Daley C, Gordin F, 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. doi:10.1164/rccm.200604-571ST
2. Winthrop KL, McNelley E, Kendall B, Marshall-Olson A, Morris C, Cassidy M, et al. Pulmonary nontuberculous mycobacterial disease prevalence and clinical features: an emerging public health disease. Am J Respir Crit Care Med. 2010;182:977–82. doi:10.1164/rccm.201003-0503OC
3. Prevots DR, Shaw PA, Strickland D, Jackson LA, Raebel MA, Blosky MA, et al. Nontuberculous mycobacterial lung disease prevalence at four integrated health care delivery systems. Am J Respir Crit Care Med. 2010;182:970–6. doi:10.1164/rccm.201002-0310OC
4. Cassidy PM, Hedberg K, Saulson A, McNelly E, Winthrop KL. Nontuberculous mycobacterial disease prevalence and risk factors: a changing epidemiology. Clin Infect Dis. 2009;49:e124–9. doi:10.1086/648443
5. Population Research Center, Portland State University. Oregon population report 2005 and 2006 [cited 2010 Jan 27]. http://www.pdx.edu/prc/annual-oregon-population-report
6. Oregon Health and Science University, Oregon Office of Rural Health. Rural definitions [cited 2010 Aug 1]. http://www.ohsu.edu/xd/outreach/oregon-rural-health/data/rural-definitions/index.cfm
7. Falkinham JO III. Mycobacterial aerosols and respiratory disease. Emerg Infect Dis. 2003;9:763–7.
8. Falkinham JO III, Norton CD, LeChevallier MW. Factors influencing numbers of *Mycobacterium avium*, *Mycobacterium intracellulare*, and other mycobacteria in drinking water distribution systems. Appl Environ Microbiol. 2001;67:1225–31. doi:10.1128/AEM.67.3.1225-1231.2001
9. Tsukamura M, Kita N, Shimoide H, Arakawa H, Kuze A. Studies on the epidemiology of nontuberculous mycobacteriosis in Japan. Am Rev Respir Dis. 1988;137:1280–4.
10. Ahn CH, Lowell JR, Onstad GD, Shuford EH, Hurst GA. A demographic study of disease due to *Mycobacterium kansasii* or *M. intracelluare-avium* in Texas. Chest. 1979;75:120–5. doi:10.1378/chest.75.2.120

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Carriage of Meningococci by University Students, United Kingdom

To the Editor: *Neisseria meningitidis* causes septicemia and meningitis (*1*). Meningococci usually persist on the nasopharyngeal mucosa of asymptomatic carriers (*2*). Because carriers are the only reservoir of meningococci, carriage in at-risk populations should be monitored. Meningococcal carriage rates have been assessed during 1997–8 for first-year students at the University of Nottingham (*3*) and in autumn during 1999–2001 for >48,000 sixth-form students (pre-university, age range 15–17 years) throughout the United Kingdom (*4*). Serogroup B and nongroupable strains predominated; serogroup Y strains were found in only 1%–2% of participants.

From November 2008 through May 2009, to investigate persistence and spread of meningococcal strains in students living in dormitories, we conducted a longitudinal study in a cohort of 190 first-year students at the University of Nottingham. We found high rates of carriage and prevalence of serogroup Y strains (*5*).

During September 2009 (first week of term) through March 2010, we conducted a large repeated cross-sectional study analyzing pharyngeal swabs from students in all school-year groups at Nottingham University.