

References

1. Liao Q, Lam WT, Leung GM, Jiang C, Fielding R. Live poultry exposure, Guangzhou, China, 2006. *Epidemics*. 2009;1:207–12. <http://dx.doi.org/10.1016/j.epidem.2009.09.002>
2. Yuan J, Zhang L, Xu W, Shen J, Zhang P, Ma H. Reported changes in health-related behaviours in Chinese urban residents in response to an influenza pandemic. *Epidemiol Infect*. 2009;137:988–93. <http://dx.doi.org/10.1017/S0950268809002726>
3. Statistics Bureau of Guangzhou. The 6th national population census, Guangzhou, 2010 [in Chinese]. 2011 [cited 2013 Jul 2]. http://www.gzstats.gov.cn/tjgb/glpceb/201105/t20110517_25227.htm
4. Korn EL, Graubard BI. Analysis of health surveys. New York: Wiley; 1999.
5. Ministry of Health of the People's Republic of China. Shanghai and Anhui confirmed three cases of human infected with avian influenza A/H7N9 [in Chinese]. Beijing: the Ministry; 2013 [cited 2014 Feb 13]. <http://www.moh.gov.cn/mohwsyjbs/s3578/201303/44f25bd6bed14cf082512d8b6258fb3d.shtml>
6. Fielding R, Lam WWT. Reducing avian influenza risk: a qualitative exploration of issues in Hong Kong. *Health Educ*. 2007;107:437–47. <http://dx.doi.org/10.1108/09654280710778574>
7. Fielding R, Bich TH, Quang LN, Lam WWT, Leung GM, Tien TQ, et al. Live poultry exposures, Hong Kong and Hanoi, 2006. *Emerg Infect Dis*. 2007;13:1065–7. <http://dx.doi.org/10.3201/eid1307.061031>

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Socioeconomic Status and Campylobacteriosis, Connecticut, USA, 1999–2009

To the Editor: *Campylobacter* is the second most common bacterial cause of foodborne gastrointestinal illnesses in the United States and the leading cause of these illnesses in Connecticut (1). It is also the leading identifiable cause of Guillain-Barré syndrome in the United States and all industrialized countries in which it has been studied (2). According to the Foodborne Disease Active Surveillance Network (FoodNet), campylobacteriosis incidence in the United States is increasing (1). Clarification of the epidemiology of campylobacteriosis is needed to control and prevent infection.

Socioeconomic status (SES) measures have not been explored in the United States as determinants for *Campylobacter* infection. Although individual SES measures are not routinely collected in FoodNet, street address of patient residence is. Following the recommended method of the Public Health Disparities Geocoding Project (3), we used census tract-level poverty as an SES measure for analysis. We attempted to geocode patient residences for all campylobacteriosis cases reported in Connecticut during 1999–2009 and to categorize them into 4 groups on the basis of percentage of residents in the census tract living below the federal poverty line: 0–<5%, 5%–<10%, 10%–<20%, and ≥20%. The average annual age-adjusted (on the basis of 2000 US Census data for Connecticut) incidence rate was calculated for each of 4 census tract-level neighborhood SES (i.e., neighborhood poverty) categories for all years combined and for 3 periods (1999–2002, 2003–2005, and 2006–2009). In addition, age group-specific rates were calculated for case-patients in the 4 SES categories. We used the

χ^2 test for trend to assess the statistical significance of observed gradients of incidence across SES levels.

We geocoded 5,708 (95.9%) of the 5,950 campylobacteriosis cases reported during 1999–2009 to census tract level. The average annual crude incidence rate was 15.9 per 100,000 population; average age-specific incidence ranged from 9.4 in the 10–19-year age group to 18.1 in the ≥50-year age group. We found a strong dose-response relationship between higher campylobacteriosis incidence and higher neighborhood SES. Average annual age-adjusted incidence was 10.1 (95% CI 9.1–11.1) for the lowest SES group (≥20% below poverty), 11.9 (95% CI 11.0–12.9) for the 10%–<20% group, 14.8 (95% CI 14.0–15.7) for the 5%–<10% group, and 16.9 per 100,000 (95% CI 16.3–17.4) for the highest SES group (0–<5% below poverty) ($p<0.001$ by χ^2 for trend). A strong SES gradient was also consistent and significant ($p<0.001$ by χ^2 for trend) for each of the 3 periods.

Incidence within age groups by neighborhood SES level is shown in the Figure. For all age groups ≥10 years, incidence of campylobacteriosis increased as neighborhood SES increased ($p<0.001$ for each category by χ^2 for trend). However, for children 0–<10 years of age, the socioeconomic gradient seen in teenagers and adults reversed direction; incidence increased as neighborhood SES decreased ($p<0.001$ by χ^2 for trend). Because only 51% of case reports included information on race and ethnicity, we were unable to examine whether SES gradients occurred within each major racial/ethnic group in Connecticut.

Previous studies using similar area-based methods in Denmark; Manitoba, Canada; Queensland, Australia; and Scotland also found an association between *Campylobacter* infection incidence and higher area-based SES (4–7). A true higher prevalence of major campylobacteriosis risk factors among patients with a higher SES

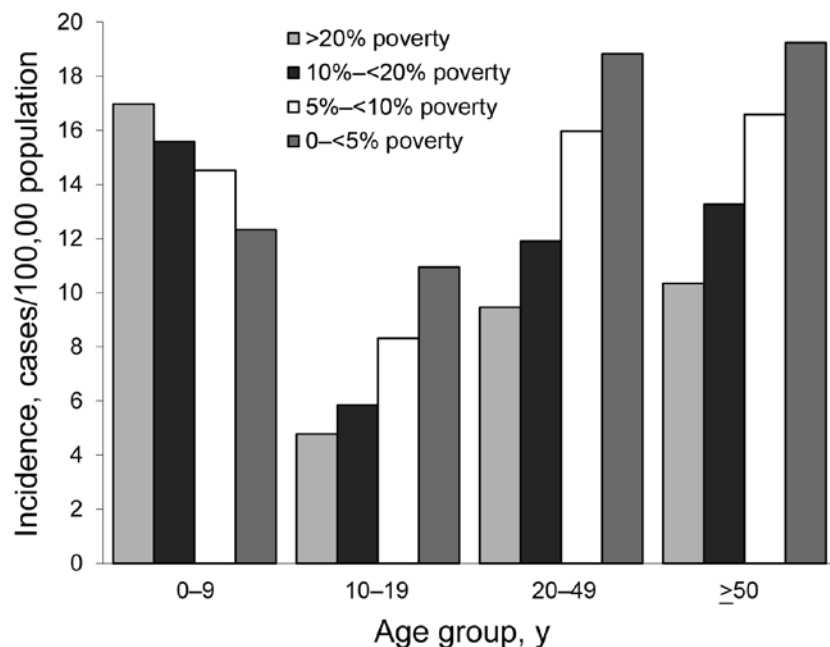


Figure. Average annual incidence rates for *Campylobacter* infection, by age group and neighborhood poverty level, Connecticut, 1999–2009. Census tract groupings were determined by percentage of residents living below the federal poverty level on the basis of data from the 2000 US Census.

might explain these findings, but these results could also indicate surveillance artifacts if persons at higher SES levels are more likely to seek health care and have an organism-specific diagnosis made. We believe the former hypothesis is more likely for several reasons. First, major risk factors for adult campylobacteriosis at FoodNet sites are international travel and eating out at restaurants (8). We examined these factors in Connecticut by using the 3 FoodNet population surveys (9) that occurred during the study period (2000–2001, 2002–2003, and 2006–2007) and found that these factors were associated with higher SES (K. Bemis, unpub. data). Second, we found that higher incidence in children <10 years of age was associated with lower SES, a finding that would not be expected if children living in poorer neighborhoods were less likely to receive a diagnosis of campylobacteriosis. Last, we examined Connecticut-specific data from the same 3 FoodNet population surveys (9) and

found that high-income adults who had diarrhea were no more likely than those with lower incomes to visit a healthcare provider and have a stool specimen taken (K. Bemis, unpub. data). The finding that children living in poorer census tracts were at higher risk than those in higher SES areas could conceivably reflect a higher rate of exposure to *Campylobacter* spp. in the home. However, this hypothesis needs verification. In addition, studies in other parts of the United States are needed to corroborate this study's findings.

We conclude that *Campylobacter* control efforts, at least in Connecticut, should take into consideration the groups with highest age-specific, SES-related incidence. Area-based SES measures should be more widely used when analyzing surveillance data.

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References

- Centers for Disease Control and Prevention. Incidence and trends of infection with pathogens transmitted commonly through food—Foodborne Diseases Active Surveillance Network. *MMWR Morb Mortal Wkly Rep.* 2013;62:283–7.
- Rees JH, Soudain SE, Gregson NA, Hughes RAC. *Campylobacter jejuni* infection and Guillain-Barré syndrome. *N Engl J Med.* 1995;333:1374–9. <http://dx.doi.org/10.1056/NEJM199511233332102>
- Krieger N, Chen JT, Waterman PD, Rehkopf DH, Subramanian SV. Painting a truer picture of US socioeconomic and racial/ethnic health inequalities: The Public Health Disparities Geocoding Project. *Am J Public Health.* 2005;95:312–23. <http://dx.doi.org/10.2105/AJPH.2003.032482>
- Simonsen J, Frisch M, Ethelberg S. Socioeconomic risk factors for bacterial gastrointestinal infections. *Epidemiology.* 2008;19:282–90. <http://dx.doi.org/10.1097/EDE.0b013e3181633c19>
- Green CG, Krause DO, Wylie JL. Spatial analysis of campylobacter infection in the Canadian province of Manitoba. *Int J Health Geogr.* 2006;5:2. <http://dx.doi.org/10.1186/1476-072X-5-2>
- Tenkate T, Stafford R, McCall B. A five year review of campylobacter infection in Queensland. *Commun Dis Intell Q Rep.* 1996;20:478–82.
- Bessel PE, Matthews L, Smith-Palmer A, Rotariu O, Strachan JC, Forbes KJ, et al. Geographic determinants of reported human campylobacter infections in Scotland. *BMC Public Health.* 2010 [cited 2013 Aug 26]. <http://www.biomedcentral.com/1471-2458/10/423>
- Friedman CR, Hoekstra RM, Samuel M, Marcus R, Bender J, Shiferaw B, et al. Risk factors for sporadic campylobacter

infection in the United States: a case-control study in FoodNet sites. *Clin Infect Dis*. 2004;38(Suppl 3):S285–96. <http://dx.doi.org/10.1086/381598>

9. Centers for Disease Control and Prevention. Foodborne Diseases Active Surveillance Network: population survey. 2012 July [cited 2013 Aug 26]. <http://www.cdc.gov/foodnet/studies/population-surveys.html>

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Legionnaires' Disease Caused by *Legionella pneumophila* Serogroups 5 and 10, China

To the Editor: Legionnaires' disease is a systemic infection caused by gram-negative bacteria belonging to the genus *Legionella*. The primary clinical manifestation is pneumonia. *Legionella* spp. are typically found in natural and artificially hydrated environments.

Legionella pneumophila is the species responsible for ≈90% of human cases of infection. *L. pneumophila* is divided into 15 serogroups, among which serogroup 1 is the most prevalent disease-causing variant (1). In contrast, rare cases are caused by other serogroups. We describe a case of Legionnaires' disease caused by co-infection with *L. pneumophila* serogroups 5 and 10 and the genotype characteristics of these strains.

The case-patient was a 77-year-old man who had chronic hepatitis B for 50 years, ankylosing spondylitis

for 40 years, and chronic cholecystitis for 5 years. On September 17, 2012, he was admitted to Wuxi People's Hospital (Wuxi, China) for treatment after a continuous cough for 15 days and a high fever for 2 days. At admission, the patient had a blood pressure of 130/65 mm Hg, a pulse rate of 102 beats/minute, and a body temperature of 37.4°C, which increased to 38.4°C four hours later. Laboratory tests showed a leukocyte count of 9,200 cells/μL (88.7% neutrophils) and a C-reactive protein level of 31 mg/L in serum. Lung inflammation was identified by computed tomography. The result of a urinary antigen test for *L. pneumophila* serogroup 1 (Binax, Portland, ME, USA) was negative. Bronchoalveolar lavage was performed, and fluid was collected for bacterial culture and molecular analysis.

Real-time PCRs were performed with primers specific for the 5S rRNA gene of the genus *Legionella* (2) and the *L. pneumophila*-specific *mip* gene (3). *Legionella* colonies isolated from bronchoalveolar lavage fluid grew on buffered-charcoal yeast extract agar. Nine *Legionella*-like colonies were isolated, and all showed positive results by PCRs. The colonies were identified as *L. pneumophila* serogroups 2–14 by using the *Legionella* latex test (Oxoid, Basingstoke, UK). Among these colonies, 5 were identified as *L. pneumophila* serogroup 5, and 4 were identified as serogroup 10 by using a monoclonal antibody (Denka Seiken, Tokyo, Japan). Environmental investigations were conducted in the patient's house and hospital room, but *L. pneumophila* serogroup 5 and 10 were not detected in any of the locations tested.

Pulsed-field gel electrophoresis (PFGE) (4) was used to investigate the 9 *L. pneumophila* strains. Two PFGE patterns that were 94% similar were observed; each pattern represented 1 serogroup. The PFGE patterns were compared with those of a reference

database of *L. pneumophila* for China. All *L. pneumophila* in the database, including 41 strains isolated from the city in which the patient resided in 2012, had patterns different from those of the 9 strains.

Two clinical *L. pneumophila* strains of different serogroups were further analyzed by sequence typing (5,6). Sequence type (ST) indicated that allele numbers for *flaA*, *pilE*, *asd*, *mip*, *mompS*, *proA*, and *neuA* genes were 6, 10, 15, 28, 21, 7, and 207 for serogroup 5 strains and 6, 10, 15, 10, 21, 40, and 207 for serogroup 10 strains. By querying the ST database for *L. pneumophila* (<http://www.ewgli.org>), we found that both profiles were new and assigned these 2 strains the numbers ST1440 (serogroup 5) and ST1439 (serogroup 10). STs of these 2 isolates differed from each other by only 2 alleles (3 nt in the *mip* gene and 1 nt in the *proA* gene), which suggested that the isolates might be more closely related to each other than suggested by serologic analysis.

Human infections with *L. pneumophila* serogroups 5 and 10 have been rarely reported (1,7). Our study confirms human infection with 2 *L. pneumophila* serogroups that did not involve serogroup 1. Results for this case-patient also indicated that a negative urinary antigen test result should not be a reason for ruling out Legionnaires' disease because the urinary antigen kit used detects only *L. pneumophila* serogroup 1 antigen. *L. pneumophila* serogroups 5 and 10 are probably underrecognized pathogenic serogroups. Culture and molecular analysis should be performed to obtain an accurate diagnosis. Rare co-infections with *L. pneumophila* serogroup strains have been identified by culture methods (8,9).

The cases reported previously and in this study indicate that co-infections with different serogroups are more common than currently recognized and that multiple colonies should be