(H5N1) virus, suggestive of a cohort effect or otherwise, have yet to be published, although anecdotal reports of completed surveys point to a lack of widespread human infection with the virus (*8*). Current evidence indicates that pandemic influenza of humans since 1918 has been restricted to 3 influenza A virus subtypes: H1 (1918–57 and 1977–present); H2 (1957–68); and H3 (1968–present) (*9*,*10*). If an element of immunity to avian influenza A (H5N1) does exist in older populations, its possible association with geographically widespread (intercontinental) influenza A events before the late 1960s merits further investigation.

The work described has been undertaken as part of a program of research entitled Historical Geography of Emerging and Re-Emerging Epidemics, 1850–2000, funded by the History of Medicine Committee of the Wellcome Trust.

Matthew Smallman-Raynor* and Andrew D. Cliff†

*University of Nottingham, Nottingham, England; and †University of Cambridge, Cambridge, England

References

1. World Health Organization. Cumulative number of confirmed human cases of avian influenza A (H5N1) reported to WHO: 4 July 2006. Geneva: The Organization; 2006. Available from http://www.who.int/csr/disease/avian_influenza/country/en/index.html
2. World Health Organization. Epidemiology of WHO-confirmed human cases of avian influenza A (H5N1) infection. Wkly Epidemiol Rec. 2006;81:249–57.
3. World Health Organization. Avian influenza fact sheet (April 2006). Wkly Epidemiol Rec. 2006;81:129–36.
4. World Health Organization. Situation updates–avian influenza. Geneva: The Organization; 2004–6. Available from http://www.who.int/csr/disease/avian_influenza
5. World Health Organization. Avian influenza: assessing the pandemic threat. Geneva: The Organization; 2005.
6. Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat. World population prospects: the 2004 revision; and world urbanization prospects: the 2003 revision. New York: United Nations; 2006. Available from http://esa.un.org/unpp
7. Tukey JW. Exploratory data analysis. Reading (MA): Addison-Wesley; 1977.
8. Enserink M. Avian influenza: amid mayhem in Turkey, experts see new chances for research. Science. 2006;311:314–5.
9. Dowdle WR. Influenza A virus recycling revisited. Bull World Health Organ. 1999;77:820–8.
10. Hilleman MR. Realities and enigmas of human viral influenza: pathogenesis, epidemiology and control. Vaccine. 2002;20:3068–87.

Address for correspondence: Matthew Smallman-Raynor, School of Geography, University of Nottingham, University Park, Nottingham, NG7 2RD, England; email: matthew.smallman-raynor@nottingham.ac.uk

Toxoplasma gondii, Brazil

To the Editor: Recently, Jones et al. reported that past pregnancies increased risk for recent *Toxoplasma gondii* infection in Brazil (*1*). They did not, however, control for age. Previous seroepidemiologic studies have shown that age is a main confounding variable in analysis of risk factors for toxoplasmosis (*2*). Age can explain why mothers with more children are at higher risk for toxoplasmosis; the longer persons live in areas with high toxoplasmosis prevalence, the higher their risk for infection.

Also not explored were drinking water–related factors. Our recent study of pregnant women in Quindio, Colombia, found factors that explained attributable risk percent for infection to be eating rare meat (0.26%) and having contact with a cat <6 months of age (0.19%) (*3*). Drinking bottled water was more significantly protective for the group that did not consume undercooked or raw meat (odds ratio 0.06, 95% confidence interval 0.006–0.560, p = 0.008). We think that drinking water–related factors could explain up to 50% of toxoplasmosis infections in our region.

Jorge Gomez-Marin*

*Universidad del Quindio, Armenia, Quindio, Colombia

References

1. Jones JL, Muccioli C, Belfort R Jr, Holland GN, Roberts JM, Silveira C. Recently acquired *Toxoplasma gondii* infection, Brazil. Emerg Infect Dis. 2006;12:582–6.
2. Juliao O, Corredor A, Moreno GS. National study of health: toxoplasmosis in Colombia, Ministry of Health [in Spanish]. Bogota: National Institute of Health Press; 1988.
3. Lopez-Castillo CA, Diaz-Ramirez J, Gomez-Marín JE. Risk factors for *Toxoplasma gondii* infection in pregnant women in Armenia, Colombia [in Spanish]. Rev Salud Publica (Bogota). 2005;7:180–90.

Address for correspondence: Jorge Gomez-Marin, Universidad del Quindio, Centro de Investigaciones Biomedicas, Av Bolivar 12N Armenia 00, Quindio, Colombia; email: jegomezmarin@hotmail.com

In response: We thank Dr Gomez-Marin for his letter regarding our article on recently acquired *Toxoplasma gondii* infection in Brazil (*1*). Dr Gomez-Marin states that perhaps age could account for our finding that having had children was a risk factor for recent *T. gondii* infection among women. Studies have shown that age is a risk factor for prevalent *T. gondii* infection; i.e., infection prevalence increases with age (*2*). However, age is not necessarily a risk factor for recent (incident) infection.

Our study of risk factors for *T. gondii* infection was a case-control design to evaluate recent infection, not a cross-sectional study of *T. gondii* infection prevalence in a population. In our study, case-patients with recent infection were similar in age to *T. gondii*–negative control-patients, although among women the mean age of case-patients (33 years) differed slightly from that of control-patients (29 years) ($p = 0.03$, *t*-test). In addition, multivariate analysis comparing the case-patients with control-patients showed that age was not a significant factor. However, when we kept age in the multivariate model for women ($p = 0.87$ for age in the model), the odds ratio for having had children changed little, from 14.94 (95% confidence interval [CI] 3.68–60.73) to 14.01 (95% CI 2.88–68.08). Therefore, we do think that, in this study population, having had children is a risk factor for *T. gondii* infection among women.

Dr Gomez-Marin also states that we did not evaluate drinking water–related factors. However, in our methods section (*1*), we indicated that our questionnaire asked about a comprehensive set of risk factors related to drinking water. Specifically, the questionnaire asked about the types of water (city, private well, and others, including bottled water); chlorination; filtering of water; and ingestion of water from streams, lakes, rivers, ponds, or other sources. Although we evaluated numerous water-related factors, we did not find them to be significant in this study, which applies to 1 area of Brazil. In other areas of Brazil, however, studies in which 1 of our authors (J.L.J.) has been involved have found water to be a risk factor or a source of infection (*2*,*3*).

Again, we thank Dr Gomez-Marin for his letter. We sincerely appreciate his interest and work with toxoplasmosis.

Jeffrey L. Jones,* Cristina Muccioli,† Rubens Belfort Jr,† Gary N. Holland,‡ Jacquelin M. Roberts,* and Claudio Silveira§

*Centers for Disease Control and Prevention, Atlanta, Georgia, USA; †Federal University of São Paulo, São Paulo, Brazil; ‡University of California, Los Angeles, California, USA; and §Clinica Silveira, Erechim, Rio Grande do Sul, Brazil

References

1. Jones JL, Muccioli C, Belfort R, Holland GN, Roberts JM, Silveira C. Recently acquired *Toxoplasma gondii* infection, Brazil. Emerg Infect Dis. 2006;12:582–7.
2. Bahia-Oliveira LM, Jones JL, Azevedo-Silva J, Alves CF, Orefice F, Addiss DG. Highly endemic, waterborne toxoplasmosis in north Rio de Janeiro state, Brazil. Emerg Infect Dis. 2003;9:55–62.
3. de Moura L, Bahia Oliveira L, Wada MY, Jones JL, Tuboi SH, Carmo EH, et al. Waterborne toxoplasmosis, Brazil, from field to gene. Emerg Infect Dis. 2006;12:326–9.

Address for correspondence: Jeffrey L. Jones, Centers for Disease Control and Prevention, 4770 Buford Hwy NE, Mailstop F22, Atlanta, GA 30341-3724, USA; email: jlj1@cdc.gov

CTX-M Extended-spectrum β-Lactamases, Washington State

To the Editor: The CTX-M–type β-lactamases are non-TEM and non-SHV plasmid-mediated, class A, extended-spectrum β-lactamases (ESBLs). The CTX-M–type β-lactamases have recently emerged as the most common type of ESBLs, with a global distribution (*1*). In contrast, the CTX-M–type ESBLs are rarely reported in the United States and have not been identified in pathogens isolated from infected patients with gastroenteritis.

We screened 637 *Salmonella* and 126 *Shigella* isolates, collected in the state of Washington during 2003–2004, for CTX-M–type β-lactamases. Of these, 60 *Salmonella* isolates that exhibited an ESBL phenotype were further characterized by PCR for TEV, SHV, CTM-X, and CMY. All were positive for the CMY-2 or TEM-1 β-lactam genes. One *Shigella sonnei* isolate (WA7593), cultured from a fecal specimen in August 2004, tested positive with an ESBL confirmatory disk diffusion panel (ceftazidime 24 mm, ceftazidime/clavulanate 32 mm, cefotaxime 14 mm, and cefotaxime/clavulanate 34 mm; [*2*]). The patient had recently traveled to Pakistan and likely became ill there and returned to the United States while still sick. The transfer of extended-spectrum cephalosporin resistance was tested by conjugation to *Escherichia coli* J53 azi^R (*3*). The MIC for *S. sonnei* WA7593 and its transconjugant, WA7593TC1, were tested by using the E-test (AB Biodisk, Solna, Sweden). Both strains were resistant to cefotaxime and susceptible to ceftazidime and showed almost the same antimicrobial susceptibility patterns as β-lactam antimicrobial drugs (Table).

The type of ESBL produced by these strains was determined by using PCR specific for TEM and CTX-M (*4*,*5*). Both strains were PCR positive for TEM and CTX-M. The TEM type PCR products were then sequenced and identified as TEM-1; no variation was found on the promoter region of $bla_{\text{TEM-1}}$. The entire sequence of $bla_{\text{CTX-M}}$ from WA7593 was then sequenced (*1*), and the product showed 100% homology with $bla_{\text{CTX-M-15}}$ (GenBank accession no. AY960984). The mobile element associated with the transfer of $bla_{\text{CTX-M-15}}$ was investigated by sequencing the flanking regions.