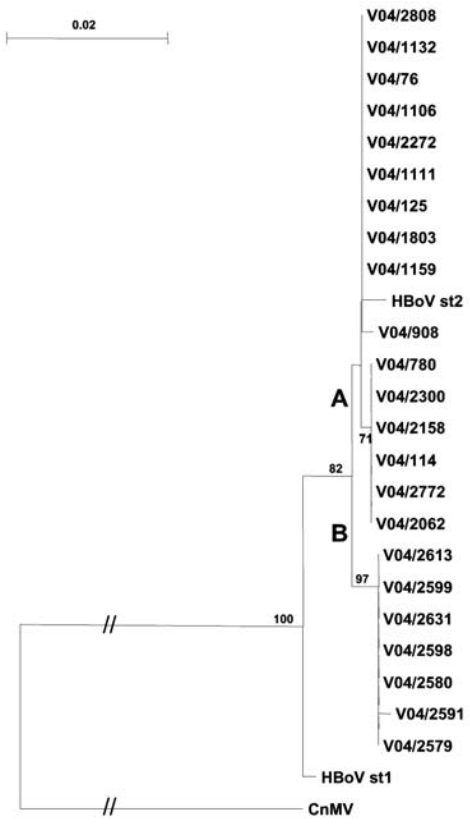

Figure. Phylogenetic analysis of a 980-bp region of the human bocavirus (HBoV) VP1/2 capsid gene from South African children with respiratory tract disease. The tree was constructed by using the neighbor-joining method with 1,000 bootstrap resamplings. All nucleotide sequences were submitted to GenBank (accession nos. DQ317539–DQ317561). CnMV, canine minute virus.

substantial. These findings suggest that HBoV may play a role in respiratory tract infections in young children who require hospitalization.

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References

1. van den Hoogen BG, de Jong JC, Groen J, Kuiken T, de Groot R, Fouchier RA, et al. A newly discovered human pneumovirus isolated from young children with respiratory tract disease. Nat Med. 2001;7:719–24.
2. Peiris JS, Lai ST, Poon LL, Guan Y, Yam LY, Lim W, et al. Coronavirus as a possible cause of severe acute respiratory syndrome. Lancet. 2003;361:1319–25.
3. van der Hoek L, Pyrc K, Jebbink MF, Vermeulen-Oost W, Berkhout RJM, Wolthers KC, et al. Identification of a new coronavirus. Nat Med. 2004;10:368–73.
4. Fouchier RAM, Hartwig NG, Besteboer TM, Niemeyer B, de Jong JC, Simon JH, et al. A previously undescribed coronavirus associated with respiratory disease in humans. Proc Natl Acad Sci U S A. 2004;101: 6212–6.
5. Woo PCY, Lau SKP, Chu C-M, Chan K-H, Tsoi H-W, Huang Y. Characterization and complete genome sequence of a novel coronavirus, coronavirus HKU1, from patients with pneumonia. J Virol. 2005;79:884–95.
6. Allander T, Tammi MT, Eriksson M, Bjerkner A, Tiveljung-Lindell A, Andersson B. Cloning of a human parvovirus by screening of respiratory tract samples. Proc Natl Acad Sci U S A. 2005;102:12891–6.
7. Kwok S, Higuchi R. Avoiding false positives with PCR. Nature. 1989;339:237–8.
8. Sloots TP, McErlean P, Speicher DJ, Arden KE, Nissen MD, Mackay IM. Evidence of human coronavirus HKU1 and human bocavirus in Australian children. J Clin Virol. 2006;25:99–102.

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Shigella sonnei Outbreak among Homosexual Men, London

To the Editor: In the summer of 2004, genitourinary medicine clinics in London reported cases of *Shigella sonnei* with a novel phage type pattern (later designated PTQ). Outbreak case finding involved local laboratories and genitourinary medicine physicians in London, as well as the national reference laboratory. A case was considered confirmed if *S. sonnei* PTQ was isolated from January 2004 through April 2005, and the patient had not traveled outside the country the week before illness. Possible cases were defined as for confirmed cases but were so designated when patient had a history of foreign travel in the week before illness or when travel history was unknown. From October 2004, when we became aware of the outbreak, until December 2004, we conducted telephone interviews with newly identified case-patients. For cases that occurred before October 2004, and from January 2005 through April 2005, information was obtained from laboratory records only.

Strains were phage typed by using the scheme described by Hammerstrom, Kallings, and Sjoberg, according to a protocol supplied by R. Wollin (*1*,*2*). The scheme consists of 11 phages and is based on the typing of the rough phase II variant of *S. sonnei*. The scheme comprises defined phage types (PT) 1–100 and provisional PTs A–P. Cultures were grown overnight on MacConkey agar, and a rough colony was placed in nutrient broth and grown for 18 hours at 37°C. The broth culture was then used to flood a nutrient agar plate and, once dry, spotted with the 11 phages and incubated at 37°C for 5 hours. The patterns of lysis were recorded and compared with those indicated on the typing chart. All isolates were

screened for resistance to a panel of antimicrobial agents by an agar incorporation method with Iso-Sensitest agar (Oxoid, Basingstoke, UK).

We identified 16 confirmed and 54 possible cases. Specimens from all 70 patients had the same unique pattern of lysis when phage typed, had the same profile when examined by pulsed-field gel electrophoresis, and were resistant to ampicillin, streptomycin, spectinomycin, sulfonamides, tetracyclines, and trimethoprim.

Cases occurred at a low frequency during the first half of 2004, followed by a large increase in August, September, and October (Figure). All case-patients (N = 48) were men, mean age 37 years (range 18–58 years). Five persons designated possible case-patients had traveled abroad in the week before illness (United States, France, Vietnam, Turkey, and 1 unknown destination). Of patients for whom HIV status information was available, nearly all were HIV positive (n = 30/32).

From October 2004 through December 2004, we identified 20 case-patients and interviewed 17 (85%). All were men who had sex with men (MSM). Reported symptoms were diarrhea (n = 15), abdominal pain (n = 14), fever (n = 10), blood in stools (n = 7), and vomiting (n = 6). In the week before illness, 15 reported sex with another man, about half with a casual partner, and mostly with 1 (9/15) or 2 (3/15) different men. No common sex venue was identified. Most (12/15) reported participation in oral and anal sex, and 6 reported oral-anal contact. Three patients recalled that their partner had had diarrhea around the time of sexual intercourse. Of 7 respondents who were asked, 3 reported using a condom during anal intercourse, and none reported using any barrier during oral intercourse.

That all cases were men, and many were HIV-positive MSM, who reported having sex the previous week, strongly suggests that male homosexual sex was the mode of transmission. The shape and timeframe of the epidemic curve indicates person-to-person transmission and rules out foodborne transmission linked to a gay venue. The predominance of HIV-positive homosexual men in the outbreak may be due to more symptomatic disease (from compromised cell-mediated immunity or achlorhydria [*3*]), more unprotected sex with other HIV-positive men (*4*), and greater likelihood of seeking healthcare.

Sexual transmission of shigellosis between MSM was first reported in the United States during the 1970s (*5*), and recent outbreaks have been reported in San Francisco (*6*), Canada (*7*), Australia (*8*), and Germany (*9*). The London outbreak coincided with an outbreak of *S. sonnei* in Berlin, Germany (*10*). Of the 17 Berlin case-patients, 14 were MSM. Isolates from 10 Berlin patients were subsequently tested by the same reference laboratory in London and confirmed to also be PTQ, which suggests a link between these 2 outbreaks, even though none of the London interviewees reported travel to Berlin.

Although the earliest identified case occurred in January 2004, *S. sonnei* PTQ may have been circulating among the MSM community for a longer period. The discovery of an outbreak of a novel phage type underlines the importance of prompt strain-typing for public health investigations and the benefit of good links between local clinicians, laboratories, and public health professionals. Additionally, local gay media and voluntary organizations were valuable partners for disseminating preventative health messages across London when the outbreak was in the early stages. This outbreak raises the possibility that the mobility and increased high-risk sexual practices among MSM in Europe (*4*) might facilitate mixing between sexual networks, thus causing potential for international outbreaks of sexually transmitted infection.

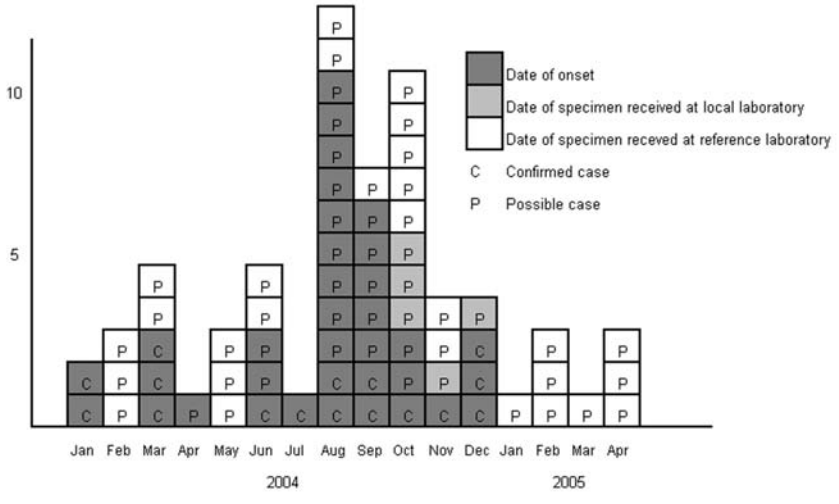

Figure. Confirmed and possible cases of *Shigella sonnei* PTQ by earliest recorded date, London, January 2004–April 2005.

Acknowledgments

We thank all those who agreed to participate in the interviews. This investigation also would not have been possible without the close cooperation of clinical colleagues and microbiologists across London. We are grateful to Valerie Delpech for helping develop the interview questionnaire.

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References

1. Hammerstrom E. Phage typing of *Shigella sonnei*. Acta Med Scand. 1949;Suppl 223:133.
2. Kallings L, Lindberg A, Sjoberg L. Phage typing of *Shigella sonnei*. Arch Immun Ther Exp. 1968;16:280–7.
3. Baer JT, Vugia DJ, Reingold AL, Aragon T, Angulo FJ, Bradford WZ, et al. HIV infection as a risk factor for shigellosis. Emerg Infect Dis. 1999;5:820–3.
4. Laporte A. A new decline in preventive behaviours among homosexual men: the role of highly active antiretroviral therapy? Euro Surveill. 2002;7:15–6.
5. Dritz SK, Back AF. *Shigella enteritis* venereally transmitted [letter]. N Engl J Med. 1974;291:1194.
6. Centers for Disease Control and Prevention. *Shigella sonnei* outbreak among men who have sex with men—San Francisco, California, 2000–2001. MMWR Morb Mortal Wkly Rep. 2001;50:922–6.
7. Outbreak of *Shigella flexneri* and *Shigella sonnei* enterocolitis in men who have sex with men, Quebec, 1999 to 2001. Can Commun Dis Rep. 2005;31:85–90.
8. O'Sullivan B, Delpech V, Pontivivo G, Karagiannis T, Marriott D, Harkness J, et al. Shigellosis linked to sex venues, Australia. Emerg Infect Dis. 2002;8:862–4.
9. Marcus U, Zucs P, Bremer V, Hamouda O, Prager R, Tschaepe H, et al. Shigellosis—a re-emerging sexually transmitted infection: outbreak in men having sex with men in Berlin. Int J STD AIDS. 2004;15:533–7.
10. Robert Kock Institut. Shigellose: Gehäuftes Auftreten bei Männern in Berlin irn Jahr 2004. Epidemiologisches Bulletin. 2005;8: 59–63.

Perinatal Toxoplasmosis, Northern Taiwan

To the Editor: Toxoplasmosis is caused by a protozoan parasite known as *Toxoplasma gondii*, which is found in animals worldwide and is readily transmitted to humans. The prevalence of *T. gondii*–specific immunoglobulin (IgG) antibodies in women ranges from ≈15% in the United States (*1*) to ≈55% in Europe (*2*). Rate of transmission to a fetus in the first, second, and third trimesters is 8%, 25%, and 60%, respectively (*3*). The rate of congenital toxoplasmosis in the United States is 1–10 per 10,000 live births (*4*). Most infants infected in utero are born without obvious signs of toxoplasmosis, and learning or visual disabilities do not develop in up to 80% until their second or third decade of life (*5,6*).

In 1985 in Taiwan, the prevalence rates of *T. gondii*–specific IgG, as determined by ELISA, for pregnant women and their neonates were 10.2% and 11.6%, respectively. No samples from mothers or neonates were screened for IgM titers (*7*). During the past 20 years, however, the lifestyle, socioeconomic environment, and healthcare system have changed substantially in Taiwan. Overseas traveling has become more convenient, and Taiwan residents often travel to toxoplasmosis-endemic areas. The number of babies born to immigrant mothers has also recently increased in Taiwan. Our objective was to estimate the seroprevalence of perinatally transmitted *T. gondii* in northern Taiwan.

We tested sera collected from consecutive samples of women and their neonates (live births only) at 1 medical center, 1 local hospital, and 2 obstetric clinics in northern Taiwan from April 2004 through January 2005, which was 1 investigation of the Taiwan Birth Panel Study. Informed consent was obtained from either parent before enrollment in the study. Serum samples from cord blood of 483 neonates and paired samples from their mothers were analyzed for *T. gondii*–specific IgG and IgM titers by ELISA (Diagnostic Products Corporation, Los Angeles, CA, USA) (IgG sensitivity 94%, specificity 100%; IgM sensitivity 96.9%, specificity 91%) (*8*). Samples from the mothers were tested within 2 days of delivery. Additional data about health measures and conditions were collected by trained interviewers using structured questionnaires.

Among the study population, 93% were Taiwanese, 0.6% were Taiwanese aboriginals, 2.5% were mainland Chinese, and 3.9% were immigrants from southeastern Asia. Of the 483 mothers, 0.6% worked as farmers, 76% were 25–35 years of age, >50% had a university-level education, 77.7% encountered pets daily, and 9.7% owned cats. Of the 483 infants, the male:female ratio was 50.8:49.2, delivery was premature for 8.6%, and 5.8% had a low birthweight. For the mothers, the *T. gondii*–specific IgG prevalence rate was 9.1% (95% confidence interval [CI] 6.5%–11.7%), and 5 mothers (1.0%; 95% CI 0.1%–1.9%) were IgM positive. The *T. gondii*–specific IgG prevalence rate for the neonates was 9.3% (95% CI 6.7%–11.9%), and 1 neonate (0.2%; 95% CI 0%– 0.6%) was IgM positive (Table).

We identified 2 risk factors for seropositive mothers: being from