Meningitis Caused by *Streptococcus suis* Serotype 14, North America

To the Editor: *Streptococcus suis* is an opportunistic pathogen that can cause serious systemic infections in pigs and occupation-related infections in humans who work in close contact with pigs or pork by-products. Most *S. suis* organisms isolated from diseased pigs belong to serotypes 1–8 (1). The most prevalent strain worldwide is serotype 2, which causes invasive infections in pigs and humans (2). We report a case of human meningitis caused by *S. suis* serotype 14.

The patient was a 59-year-old woman from rural Manitoba, Canada; she worked at a hog plant and handled 300–400 piglets/day. In October 2007, when she sought care, she had a 2-day history of fever, vomiting, headache, neck pain, and reduced consciousness. She was febrile and confused and had meningeval signs. Leukocyte count was 19,900/mm³. Cerebrospinal fluid (CSF) had 284 × 10⁶ leukocytes (59% lymphocytes, 41% polymorphonuclear cells), 2.3 mmol/L glucose, and 1.85 g/L total protein. Gram stain of CSF showed gram-positive cocci in chains, were catalase negative, and in pairs; cefotaxime and vancomycin were prescribed empirically. Results of computed tomography of the head, chest radiograph, and transesophageal echocardiogram were within normal limits. Blood culture was negative after 5 days of incubation. The CSF culture grew small α-hemolytic colonies on blood agar and chocolate agar. The organisms were gram-positive cocci in chains, were catalase negative, and were identified as *S. suis* by Vitek II and API 20 Strep System (both from bioMérieux, St.-Laurent, Quebec City, Canada).

Identification of the organism as *S. suis* was confirmed at the National Microbiology Laboratory, Winnipeg, Manitoba, Canada, by conventional
biochemical tests (3), the results of which were consistent with that of the type strain (Table) and were also confirmed by 16S rRNA gene sequencing, which showed 100% homology with the *S. suis* type strain ATCC 43765, GenBank accession no. EU 477176.

Antimicrobial-drug susceptibilities were determined by microbroth dilution by using Sensititre STP3F panels (Nova Century Scientific Inc., Burlington, Ontario, Canada) and cation-adjusted Mueller Hinton broth with lysed horse blood (2%–5% vol/vol) by TREK Diagnostic Systems, Inc. (Nova Century Scientific Inc.) using manufacturer’s instructions and following Clinical and Laboratory Standards Institute guidelines for *Streptococcus* spp. other than *S. pneumoniae* (4).

Most cases of *S. suis* infection in humans have been attributed to serotype 2 strains. Only 4 human cases have been reported in North America: 2 in Canada (1 endocarditis, 1 meningitis) and 2 cases of meningitis in the United States (6–9). All 4 cases were attributed to *S. suis* serotype 2. Serotype 14 has been reported as a human pathogen in the Netherlands, Thailand, the United Kingdom, and Denmark and has been routinely isolated from diseased pigs in Canada (10).

Although in pigs the organism is present in the upper respiratory tract, particularly the tonsils, nasal cavities, genital tract, and alimentary tract, the mode of transmission to humans reported so far had been through cuts in the hands. Our patient handled hundreds of piglets every day and most likely acquired the infection through her hands. Her meningitis was com-

| Test                        | *Streptococcus suis* (3) | Patient isolate |
|-----------------------------|--------------------------|-----------------|
| α-hemolysis on sheep blood agar | +                        | +               |
| Motility                    | –                        | –               |
| Catalase                    | –                        | –               |
| Oxidase                     | ND                       | –               |

| Fermented                  |                          |                 |
|-----------------------------|--------------------------|-----------------|
| L-arabinose                 | –                        | –               |
| D-glucose                   | +                        | +               |
| Glycerol                    | –                        | –               |
| Inulin                      | +                        | +               |
| Lactose                     | +                        | +               |
| Maltose                     | +                        | +               |
| Mannitol                    | –                        | –               |
| Melezitose                  | –                        | –               |
| Melibiose                   | Variable                 | +               |
| Raffinose                   | Variable                 | +               |
| Ribose                      | –                        | –               |
| Salicin                     | +                        | +               |
| Sorbitol                    | –                        | –               |
| Sucrose                     | +                        | +               |
| Trehalose                   | +                        | +               |

| Hydrolyzed                  |                          |                 |
|-----------------------------|--------------------------|-----------------|
| L-arginine                  | +                        | –               |
| Esculin/bile esculin        | +/ND                     | ±               |
| Starch                      | +                        | +               |
| Glycogen                    | +                        | +               |
| Hippurate                   | –                        | –               |

| Acetoin                     | –                        | –               |
| Optochin disk               | Resistant                | ND              |

| Enzymes                     |                          |                 |
|-----------------------------|--------------------------|-----------------|
| α-galactosidase             | +                        | +               |
| β-galactosidase             | Variable                 | +               |
| β-glucuronidase             | +                        | +               |
| Leucine arylamidase         | +                        | +               |
| N-acetylglucosaminidase     | +                        | +               |
| Acid phosphatase            | –                        | –               |
| Alkaline phosphatase        | –                        | –               |
| Pyrrolidonylarylamidase     | –                        | –               |

| API Strep code              | 4640473 high degree      |
|-----------------------------|--------------------------|

Table. Identification of organism isolated from cerebrospinal fluid of 59-year-old woman with meningitis, Manitoba, Canada*  

*+, positive; –, negative; ND, not done; API Strep code, API 20 Strep, API System (bioMérieux, St.-Laurent, Quebec City, Canada).
Outbreaks Caused by New Variants of Vibrio cholerae O1
El Tor, India

To the Editor: Vibrio cholerae O1, the causative agent of cholera, has 2 biotypes (classical and El Tor), which have traditionally been distinguished by phenotypic tests and by genetic differences in the major toxincorregulated pilus (TCP) gene, the tcpA allele of the TCP cluster (I), the rstR region (regulatory region for phase lysis) of CTX phages (2), the type of cholera toxin (CT) produced, and the infection pattern of the disease they cause. However, 3 variants of the El Tor biotype have been described recently: Matlab (a place in Bangladesh) variants in 2002 (3), which could not be biotyped because they have a mixture of both classical and El Tor (4), Mozambique variant in 2004–2005, which has a typical El Tor genome but a tandem repeat of the classical CTX prophage in the small chromosome (5), and the altered El Tor type (a typical El Tor biotype and an El Tor CTX prophage that produces CT of the classical type) predominant in Bangladesh since 2001 (6). Hybrid vibrios have also been described in other regions of Asia and Africa (7).

CT, encoded by the ctxA and ctxB genes, is the principal toxin produced by V. cholerae O1 and O139. Methods for differentiating the biotype-specific CT-B subunit of V. cholerae O1 include sequencing the ctxB gene, performing an ELISA with a monoclonal antibody specific to the classical or El Tor CT, or by using a mismatch amplification mutation assay (MAMA)–PCR to distinguish between 2 kinds of ctxB genes. This assay detects sequence polymorphisms based on nt position 203 of the ctxB gene (8).

In Punjab and Haryana states of northern India, during July–September 2007, 6 clusters of cholera outbreak were identified. A total of 745

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Letters
Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article’s publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have 1 Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

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