

silent and, when not associated with outbreaks, most likely neglected by local physicians.

This work was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP) (grant no. 04/11098-2) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (grant no. 401396/2004-5). RVMB received fellowships from FAPESP (grant no. 05/03260-7). This work also was partially supported by the Viral Genetic Diversity Network (VGDN-FAPESP-Brazil).

**Ana Carolina Bernardes Terzian,
Roberta Vieira
de Moraes Bronzoni,
Betânia Paiva Drumond,
Mônica Da Silva-Nunes,
Natal Santos da Silva,
Marcelo Urbano Ferreira,
Márcia Aparecida Sperança,
and Maurício Lacerda Nogueira**

Author affiliations: Faculdade de Medicina de São José do Rio Preto, São Paulo, Brazil (A.C. Bernardes Terzian, R.V. de Moraes Bronzoni, M.L. Nogueira); IBILCE, São José do Rio Preto (A.C. Bernardes Terzian); Universidade Estadual de Montes Claros, Montes Claros, Brazil (B.P. Drumond); Instituto de Ciências Biológicas/Universidade de São Paulo, São Paulo (M. Da Silva-Nunes, N. Santos da Silva, M.U. Ferreira); and Faculdade de Medicina de Marília, São Paulo (M.A. Sperança)

DOI: 10.3201/eid1502.080401

References

1. Pinheiro FP, Travassos da Rosa APA, Vasconcelos PFC. Oropouche fever. In: Feigin RD, editor. Textbook of pediatric infectious diseases. Philadelphia: WB Saunders Co.; 2004. p. 2418–23.
2. Anderson CR, Spence L, Downs WG, Aitken THG. Oropouche virus: a new human disease agent from Trinidad, West Indies. *Am J Trop Med Hyg.* 1961;10:574–8.
3. Saeed MF, Wang H, Nunes M, Vasconcelos PF, Weaver SC, Shope RE, et al. Nucleotide sequences and phylogeny of the nucleocapsid gene of Oropouche virus. *J Gen Virol.* 2000;81:743–8.
4. Nunes MR, Martins LC, Rodrigues SG, Chiang JO, Azevedo RSS, da Rosa AP, et al. Oropouche virus isolation, southeast Brazil. *Emerg Infect Dis.* 2005;11:1610–3.
5. Azevedo RSS, Nunes MRT, Chiang JO, Bensabath G, Vasconcelos HB, Pinto AYN, et al. Reemergence of Oropouche fever, northern Brazil. *Emerg Infect Dis.* 2007;13:912–5.
6. Silva-Nunes M, Malafronte RS, Luz BA, Souza EA, Martins LC, Rodrigues SG, et al. The Acre Project: the epidemiology of malaria and arthropod-borne virus infections in a rural Amazonian population. *Cad Saude Publica.* 2006;22:1325–34. DOI: 10.1590/S0102-311X2006000600021
7. de Moraes Bronzoni RV, Baleotti FG, Nogueira RMR, Nunes M, Figueiredo LTM. Duplex reverse transcription-PCR followed by nested PCR assays for detection and identification of Brazilian alphaviruses and flaviviruses. *J Clin Microbiol.* 2005;43:696–702. DOI: 10.1128/JCM.43.2.696-702.2005
8. Moreli ML, Aquino VH, Cruz AC, Figueiredo LT. Diagnosis of Oropouche virus infection by RT-nested-PCR. *J Med Virol.* 2002;66:139–42. DOI: 10.1002/jmv.2122
9. Kumar S, Tamura K, Nei M. MEGA3: integrated software for molecular evolutionary genetics analysis and sequence alignment. *Brief Bioinform.* 2004;5:150–63. DOI: 10.1093/bib/5.2.150
10. Shope RE. The use of a micro-hemagglutination test to follow antibody response after arthropod-borne virus infection in a community of forest animals. [Rio J]. *Ann Microbiol.* 1963;11:167–71.

Address for correspondence: Maurício Lacerda Nogueira, Laboratório de Pesquisas em Virologia, Departamento de Doenças Infecciosas e Parasitárias, Faculdade de Medicina de São José do Rio Preto—FAMERP, Av Brigadeiro Faria Lima 5416, São José do Rio Preto—SP, Brazil 15090-000; email: mnogueira@famerp.br

The opinions expressed by authors contributing to this journal do not necessarily reflect the opinions of the Centers for Disease Control and Prevention or the institutions with which the authors are affiliated.

Use of trade names is for identification only and does not imply endorsement by the Public Health Service or by the U.S. Department of Health and Human Services.

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 meningeal signs. Leukocyte count was 19,900/mm³. Cerebrospinal fluid (CSF) had 284 × 10⁶/L 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 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). This isolate was sensitive to penicillin, cefepime, cefotaxime, ceftriaxone, linezolid, trimethoprim and sulfamethoxazole, vancomycin, meropenem, and levofloxacin; it was resistant to azithromycin, erythromycin, and tetracycline. The isolate was sent to the International Reference Laboratory at the Université de Montréal, Montréal, Québec, Canada, for *S. suis* serotyping, where it was identified by the coagglutination test as serotype 14 (5).

The patient recovered quickly, and her therapy was changed to penicillin G. She was transferred to her local hospital to complete her medication. Within a week of her initial visit, bilateral deafness and loss of balance developed and progressed over the next month and had not ameliorated after 1 year.

Human *S. suis* infections result primarily from direct contact (with wounds on skin or mucosa of the mouth and nasal cavity) with carrier pigs, sick pigs, or raw pork contaminated with *S. suis* (2). The infection rate among abattoir workers, pig breeders, meat processing workers, and veterinarians is ≈ 3 cases/100,000 (1,500 \times higher than the rate for the general population) (2). A striking sequela to *S. suis* meningitis is deafness or vestibular dysfunction (2,6). A consistently higher percentage

of persons experienced deafness after *S. suis* infection than after infection with other meningitis-causing bacteria, 50% and 65% in Europe and Asia, respectively (2).

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-

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

Test	<i>Streptococcus suis</i> (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	\pm
Starch	+	+
Glycogen	+	+
Hippurate	–	–
Acetoin	–	–
Optochin disk	Resistant	ND
Enzymes		
α -galactosidase	+	+
β -galactosidase	Variable	+
β -glucuronidase	+	+
Leucine arylamidase	+	+
N-acetylglucosaminidase	+	+
Acid phosphatase	–	–
Alkaline phosphatase	–	–
Pyrrrolidonylarylamidase	–	–
API Strep code		4640473 high degree (97%) confidence <i>S. suis</i>

*+, positive; –, negative; ND, not done; API Strep code, API 20 Strep, API System (bioMérieux, St.-Laurent, Quebec City, Canada).

licated by bilateral hearing loss with vestibular dysfunction. Preexisting medical conditions, such as alcoholism, liver cirrhosis, or splenectomy, have been described to predispose patients to severe infection and hearing loss (2). Our patient, however, did not have any predisposing conditions.

Meningitis in humans caused by *S. suis* serotype 14 is less common than that caused by serotype 2, but the consequences are similar and can be reduced by early treatment with antimicrobial drugs. Identifying this case of meningitis caused by *S. suis* serotype 14 in Canada raises concerns about the public health aspect of this infection. Guidelines may be required to ensure that staff working in hog plants are aware of the risk for this infection and that they use adequate personal protective equipment.

**Ahmed Haleis, Michelle Alfa,
Marcelo Gottschalk,
Kathryn Bernard, Allan Ronald,
and Kanchana Manickam**

Author affiliations: University of Manitoba, Winnipeg, Manitoba, Canada (A. Haleis, A. Ronald); St. Boniface General Hospital, Winnipeg (M. Alfa, K. Manickam); University of Montreal, St.-Hyacinthe, Quebec, Canada (M. Gottschalk); and National Microbiology Laboratory, Winnipeg (K. Bernard)

DOI: 10.3201/eid1502.080842

References

- Higgins R, Gottschalk M. Streptococcal diseases. In: Straw BE, D'Allaire S, Mengeling WL, Taylor DJ, editors. Diseases of swine. Ames (IA): Iowa State University; 2005. p. 769–83.
- Gottschalk M, Segura M, Xu J. *Streptococcus suis* infections in humans: the Chinese experience and situation in North America. *Anim Health Res Rev*. 2007;8:29–45. DOI: 10.1017/S1466252307001247
- Kilpper-Balz R, Schleifer KH. *Streptococcus suis* sp. nov., nom. rev. *Int J Syst Bacteriol*. 1987;37:160–2.
- Clinical and Laboratory Standards Institute. Performance for antimicrobial susceptibility testing; Eighteenth informational supplement. CLSI document M100-S18. Wayne (PA): The Institute; 2008.
- Gottschalk M, Higgins R, Boudreau M. Use of polyvalent coagglutination reagents for serotyping of *Streptococcus suis*. *J Clin Microbiol*. 1993;31:2192–4.
- Lee GT, Chiu CY, Haller BL, Denn PM, Hall CS, Gerberding JL. *Streptococcus suis* meningitis, United States. *Emerg Infect Dis*. 2008;14:183–5. DOI: 10.3201/eid1401.070930
- Trottier S, Higgins R, Brochu G, Gottschalk M. A case of human endocarditis due to *Streptococcus suis* in North America. *Rev Infect Dis*. 1991;13:1251–2.
- Michaud S, Duperval R, Higgins R. *Streptococcus suis* meningitis: first case reported in Quebec. *Can J Infect Dis*. 1996;7:329–31 [cited 2009 Jan 12]. Available from <http://www.pulsus.com/journals/journalHome.jsp?HCTYPE=Physician&jnlKy=3&/home.htm>
- Willenburg KS, Sentochik DE, Zadoks RN. Human *Streptococcus suis* meningitis in the United States. *N Engl J Med*. 2006;354:1325. DOI: 10.1056/NEJMc053089
- Messier S, Lacouture S, Gottschalk M. Groupe de Recherche sur les Maladies Infectieuses du Porc (GREMIP); Centre de Recherche en Infectiologie Porcine (CRIP). Distribution of *Streptococcus suis* capsular types from 2001 to 2007. *Can Vet J*. 2008;49:461–2.

Address for correspondence: Kanchana Manickam, St. Boniface General Hospital, Clinical Microbiology, 409 Tache Ave, Winnipeg, Manitoba R2H 2A6, Canada; email: kmanickam@sbgh.mb.ca

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.

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 toxin-coregulated pilus (TCP) gene, the *tcpA* allele of the TCP cluster (1), the *rstR* region (regulatory region for phage lysogeny) 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