

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.

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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.

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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.

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

case-patients were admitted to local government hospitals; the cholera attack rate was 183/1,000 population. Four deaths were reported (case-fatality rate 0.5%). The number of cases per cluster varied from 15 to 400, and adults were primarily affected (74%); 20% of patients had severe dehydration. *V. cholerae* O1 Ogawa was confirmed from stool cultures by using standard isolation, biochemical, and serotyping methods. Twenty-six isolates were phenotypically and genotypically characterized according to biotype. Phenotypic characterization included the Voges-Proskauer reaction, polymyxin B (50 U) susceptibility, chick cell agglutination, and sheep erythrocyte hemolysis; all isolates were confirmed as El Tor biotype. Genotypic characterization included PCR assays for *ctxA* and *tcpA* (2), *rstR* (3), and *ctxB* (8). All strains were toxigenic because each carried the *ctxA* gene. All strains also carried El Tor-specific *tcpA* (472 bp) and *rstR* genes (500 bp). MAMA-PCR showed the *ctxB* gene of both El Tor and the classical type in 21 (80%) of 26 isolates tested.

Similarly, we also tested 20 available isolates from the 2002 outbreak and 4 and 19 sporadic isolates from 2003 and 2004, respectively; all were phenotypically and genotypically confirmed as El Tor and had only *ctxB* of the El Tor type. Of 53 water samples tested during the 2007 outbreak, 4 grew *V. cholerae*. Three samples were

confirmed to be non-O1, non-O139 strains. Only 1 isolate was *V. cholerae* O1, which was positive for *tcp*, *ctxA*, and *ctxB* of both classical and El Tor types (Table).

During the cholera outbreak of 2002 in Chandigarh (9), only 1 death was reported (case-fatality rate, <0.01%); the attack rate was 20/1,000, 58.6% were children, and only 10% had severe dehydration. Before the most recent outbreak, the affected regions of Panjab and Haryana (Ambala, Nurpur, Kurali, Mohali, Panchkula, and Raili) had been free of cholera outbreaks since 1994, though sporadic cases had been reported. The 4 deaths from cholera in 2007, along with adult preponderance, high attack rate, more severe illness, and 6 different clusters, point towards a change in the disease's epidemiology. This change may be related to circulation of the hybrid vibrios in this region. In Bangladesh, all strains of *V. cholerae* O1 examined since 2001 belong to the altered El Tor type (6), which produces CT of the classical type. This altered type has replaced the seventh pandemic strain of the El Tor biotype that produced CT of the El Tor type, which indicates that a cryptic change has occurred in the seventh pandemic El Tor biotype strains of *V. cholerae* O1.

Newly emerged variants from Bangladesh (8) have the genetic makeup of El Tor with *ctxB* gene of only classical, whereas our strains are unique in having *ctxB* of both the clas-

sical and El Tor biotypes. Our strains appear to be different from the Mozambique variant *V. cholerae* O1 (10), which has *rstR* of the classical type, in that our strains have *rstR* of only the El Tor type. Of 5 Matlab variants analyzed with MAMA-PCR, 3 had classical *ctxB* and 2 had El Tor type. Our study highlights the different genetic recombinations possible in *V. cholerae* and the epidemiologic role of these recombinations.

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References

1. Keasler SP, Hall RH. Detecting and biotyping *Vibrio cholerae* O1 with multiplex polymerase chain reaction. *Lancet*. 1993;341:1661. DOI: 10.1016/0140-6736(93)90792-F

Table. Phenotypic and genotypic traits of *Vibrio cholerae* O1 clinical strains isolated from northern India, 2002–2007*

Year of isolation or type of strain	Phenotypic tests				PCR amplicons		
	VP	Polymyxin B (50 U) susceptibility	CCA	Sheep erythrocyte hemolysis	<i>tcpA</i>	<i>ctxB</i> (MAMA-PCR)	<i>rstR</i>
2002 (n = 20)	+	Resistant	+	+	E	E	E
2003 (n = 4)	+	Resistant	+	+	E	E	E
2004 (n = 19)	+	Resistant	+	+	E	E	E
2007 (n = 26)	+	Resistant	+	+	E	E + C (n = 21)†	E
Environmental (n = 1)	+	Resistant	+	+	E	E + C	E
Classical 569B	–	Sensitive	–	–	C	C	C
El Tor N16961	–	Resistant	+	+	E	E	E
Hybrid NICED, India	+	Resistant	+	+	E	C	E + C

*VP, Voges-Proskauer; CCA, chick cell agglutination; MAMA, mismatch amplification mutation assay; +, positive; –, negative; E, El Tor type; C, classical type; NICED, National Institute of Cholera and Enteric Diseases, Kolkata, India.

†Remainder had *ctxB* of El Tor type.

2. Nusrin S, Khan GY, Bhuiyan NA, Ansaruzzaman M, Hossain MA, Safa A, et al. Diverse CTX phages among toxigenic *Vibrio cholerae* O1 and O139 strains isolated between 1994 and 2002 in an area where cholera is endemic in Bangladesh. *J Clin Microbiol.* 2004;42:5854–6. DOI: 10.1128/JCM.42.12.5854-5856.2004
3. Nair GB, Faruque SM, Bhuiyan A, Kamruzzaman M, Siddique AK, Sack DA. New variants of *Vibrio cholerae* O1 biotype El Tor with attributes of the classical biotype from hospitalized patients with acute diarrhea in Bangladesh. *J Clin Microbiol.* 2002;40:3296–9. DOI: 10.1128/JCM.40.9.3296-3299.2002
4. Safa A, Bhuiyan NA, Alam M, Sack DA, Nair GB. Genomic relatedness of the new Matlab variants of *Vibrio cholerae* O1 to the classical and El Tor biotypes by pulsed-field gel electrophoresis. *J Clin Microbiol.* 2005;43:1401–4. DOI: 10.1128/JCM.43.3.1401-1404.2005
5. Faruque SM, Tam VC, Chowdhury N, Diraphat P, Dziejman M, Heidelberg JF, et al. Genomic analysis of the Mozambique strains of *Vibrio cholerae* O1 reveals the origin of El Tor strains carrying classical CTX prophage. *Proc Natl Acad Sci U S A.* 2007;104:5151–6. DOI: 10.1073/pnas.0700365104
6. Nair GB, Qadri F, Holmgren J, Svennerholm AM, Safa A, Bhuiyan NA, et al. Cholera due to altered El Tor strains of *Vibrio cholerae* O1 in Bangladesh. *J Clin Microbiol.* 2006;44:4211–3. DOI: 10.1128/JCM.01304-06
7. Safa A, Sultana J, Cam PD, Mwansa JC, Kong RYC. *Vibrio cholerae* O1 hybrid El Tor strains, Asia and Africa. *Emerg Infect Dis.* 2008;14:987–8.
8. Morita M, Ohnishi M, Arakawa E, Bhuiyan NA, Nusrin S, Alam M, et al. Development and validation of a mismatch amplification mutation PCR assay to monitor the dissemination of an emerging variant of *Vibrio cholerae* O1 biotype El Tor. *Microbiol Immunol.* 2008;52:314–7. DOI: 10.1111/j.1348-0421.2008.00041.x
9. Taneja N, Jasjit K, Kusum S, Singh M, Kalra JK, Sharma NM, et al. A recent outbreak of cholera due to *Vibrio cholerae* O1 Ogawa in and around Chandigarh, North India. *Indian J Med Res.* 2003;117:243–6.
10. Ansaruzzaman M, Bhuiyan NA, Nair GB, Sack DA, Lucas M, Deen JL, et al. Cholera in Mozambique, variant of *Vibrio cholerae*. *Emerg Infect Dis.* 2004;10:2057–9.

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Human Case of *Atopobium rimae* Bacteremia

To the Editor: The genus *Atopobium* (*I*) accommodates species formerly designated *Lactobacillus minutus*, *L. rimae*, and *Streptococcus parvulus* (2). Use of 16S rDNA sequence analysis showed these species to be closely related and to form a distinct line of descent within the lactic acid bacteria (3). *Atopobium* spp. usually have been recognized as part of the human gingival oral flora; some species, including *A. rimae* and *A. parvulum*, have been identified as agents of chronic periodontitis (4,5). *A. rimae*, formerly known as *L. rimae* (*I*), forms short, gram-positive, strictly anaerobic, elliptical bacteria with low G+C content (4). *A. rimae* produces large amounts of lactic acid and has been recovered previously from normal human gingival flora (4,5). Apart from periodontitis, it has not been implicated in other types of infection. We report an unusual case of *A. rimae* bacteremia.

In May 2007, a 77-year-old woman with a history of right thoracotomy for pneumothorax 2 years earlier was hospitalized for inhalation pneumonia caused by paralysis of the right vocal cord. During hospitalization, septic shock and a fever of 38°C developed in the patient, complicated by acute respiratory failure and stroke. She was transferred to an intensive care unit with a PaO₂/FiO₂ >300 mm Hg, and a tracheotomy was performed. Three anaerobic blood specimens, drawn at entrance into the intensive care unit, yielded gram-positive cocci after 24-h incubation of the first bottle and gram-positive bacilli after 48-h incubation of the 2 other bottles. The gram-positive cocci were identified as *Streptococcus gordonii* using API STREP (bioMérieux, Marcy l'Etoile, France). The gram-positive bacilli were catalase negative and oxidase positive

but remained unidentified with use of API ANA strip (bioMérieux). Minimum inhibitory concentrations of antibiotics were determined for the gram-positive bacilli using E-test assay (AB BIODISK, Solna, Sweden) on Columbia agar supplemented with 5% sheep blood. Minimum inhibitory concentrations were 0.064 µg/mL for penicillin G, 0.023 µg/mL for ampicillin, 0.012 µg/mL for amoxicillin-clavulanic acid, 0.032 µg/mL for imipenem, <0.016 µg/mL for azithromycin, <0.016 µg/mL for erythromycin, 0.06 µg/mL for ciprofloxacin, and 1.25 µg/mL for vancomycin. DNA was extracted from 1 colony by using a QIAamp tissue kit (QIAGEN, Hilden, Germany) as described by the manufacturer. The 1,454-bp 16S rDNA sequence obtained using the fD1 5'-AGAGTTTGATCCTGGCTCAG-3' and rP2 5'-ACGGCTACCTTGTTAC GACTT-3' primer pair (6,7) showed 99% sequence similarity with the 16S rDNA sequence of *A. rimae* (GenBank accession no. AF292371) by use of BLAST version 2.2.9 software (National Center for Biotechnology Information). A phylogenetic neighbor-joining tree based on the *Atopobium* spp. 16S rDNA sequences made with the MEGA software confirmed that the isolate belonged to *A. rimae* (Figure). Initial treatment by intravenous tazocilline-amikacin was changed to intravenous amoxicillin-clavulanic acid (2 g/200 mg). The fever resolved, and the patient's condition improved. The treatment was stopped after 7 days, and the patient remained afebrile.

In this case, phenotypic identification of gram-positive bacillus isolated from 2 blood cultures failed because the definite bacterial species *A. rimae* was not included in the API database used for the phenotypic identification. Final identification was achieved within 2 days by comparison of the almost complete 16S rDNA sequence with homologous sequences deposited in Genbank. This comparison yielded a 99% sequence similarity, regarded