India experienced the first confirmed outbreak of chikungunya fever in 1963–1964 in Kolkata (*7*) and in 1965 in Chennai. The last epidemic in India was reported from Barsi in the state of Maharastra in 1973 (*8*). However, during these outbreaks, Andaman and Nicobar Islands were not affected. Outbreaks of dengue fever and chikungunya fever are known to occur simultaneously, as has happened in several parts of India. However, during the current outbreak in Andaman Islands, dengue infection was not detected. (Dengue has never been reported in the islands.) As chikungunya fever is known for its mysterious pattern of dramatic outbreaks interspersed by periods of prolonged absence, the introduction of this virus to an unexposed population has great public health importance.

This outbreak could be a warning about preparedness for health authorities not only in these islands but also in other areas where chikungunya fever has not occurred previously. With the extent of human travel to and from areas with active chikungunya virus transmission, many areas where the disease has not previously been reported could be at risk. As an outbreak response, the Regional Medical Research Centre and Directorate of Health Services, Andaman and Nicobar Administration, has undertaken a comprehensive community-based survey to assess the impact of chikungunya fever and *Aede*s infestation levels. We are stepping up our applied field research to prevent future outbreaks of chikungunya fever, as well as dengue fever.

Acknowledgments

We are thankful to the director and scientists of National Institute of Virology, Pune, for performing anti-CHIKV IgM tests.

This study was supported by the internal funds of Indian Council of Medical Research.

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References

1. Bessaud M, Peyrefitte CN, Pastorino BA, Tock F, Merle O, Colpart JJ, et al. Chikungunya virus strains, Reunion Island outbreak. Emerg Infect Dis. 2006;12:1604–6.
2. Lahariya C, Pradhan SK. Emergence of chikungunya virus in Indian subcontinent after 32 years: a review. J Vector Borne Dis. 2006;43:151–60.
3. Government of India, Ministry of Health and Family Welfare. Update on chikungunya: 13th October 2006. [cited 2007 Mar 11]. Available from http://www.nvbdcp.gov.in/doc/chikungunya%20-%20update.pdf
4. National Vector Borne Disease Control Programme. Directorate General of Health Services, Ministry of Health and Family Welfare, Government of India. Chikungunya trend. [cited 2007 Mar 11]. Available from http://www.nvbdcp.gov.in/chikun-trend.html
5. Shriram AN, Sehgal SC. *Aedes aegypti* (L) in Port Blair, Andaman and Nicobar Islands–distribution and larval ecology. J Commun Dis. 1999;31:185–92.
6. Hasebe F, Parquet MC, Pandey BD, Mathenge EGM, Morita K, Balasubramaniam V, et al. Combined detection and genotyping of Chikungunya virus by a specific reverse transcription polymerase chain reaction. J Med Virol. 2002;67:370–4.
7. Shah KV, Gibbs CJ Jr, Banerjee G. Virological investigation of the epidemic of haemorrhagic fever in Calcutta: isolation of three strains of Chikungunya virus. Indian J Med Res. 1964;52:676–83.
8. Padbidri VS, Gnaneswar TT. Epidemiological investigations of chikungunya epidemic at Barsi, Maharastra state, India. J Hyg Epidemiol Microbiol Immunol. 1979;23:445–51.

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Alistipes finegoldii in Blood Cultures from Colon Cancer Patients

To the Editor: *Alistipes finegoldii* was previously isolated from appendiceal tissue samples in children with acute appendicitis and from perirectal and brain abscess material (*1*,*2*). 16S rDNA sequencing studies showed that this bacterium clustered with *A. putredinis* (Figure) in the Bacteroidetes group (*4*). We describe the first cases, to our knowledge, of bacteremia due to *A. finegoldii* in 2 patients with colon cancer who underwent surgical resection.

The first patient was a 61-year-old woman with colorectal carcinoma and liver metastasis, who underwent chemotherapy consisting of 6 cycles of oxaliplatin (the FOLFOX scheme, a chemotherapy regimen consisting of fluorouracil [5 FU], folinic acid, and oxaliplatin). In September 2003, a left colectomy, resection of metastasis in the left side of the liver, and a ligation of the right portal vein were performed. Two months later, in a second step, a right hepatectomy was done. On postoperative day 5, the patient had a fever up to 39.8°C and leukocyte count of 8.49 g/L (68% polymorphonuclear leukocytes). Two blood cultures were performed before antimicrobial drug therapy based on amoxicillin/clavulanic acid and amikacin was started. After receiving this therapy, the patient recovered rapidly. One of the 2 anaerobic blood cultures was positive. Gram-negative bacilli were isolated (strain 3302398). Antimicrobial susceptibility testing showed decreased susceptibility to vancomycin, cefotetan, and penicillin G. The strain produced β-lactamase as determined by Cefinase test (Becton Dickinson, Le Pont de Claix, France).

The second patient was a 64-year-old man with colon cancer who was receiving palliative chemotherapy

(16th cycle, FOLFOX scheme); he was seen in March 2004 with a fever up to 39°C. An adenocarcinoma of the ileum had been diagnosed in June 2002 in this patient, and an ileocecal resection was performed followed by adjuvant chemotherapy. One year later, a local recurrence and peritoneal carcinomatosis were detected. The patient again underwent abdominal surgery by resection of ileo-colic anastomosis and sigmoid and peritoneal masses; a colostomy had to be created. The patient's leukocyte count was 14.94 g/L (84.6% polymorphonuclear leukocytes), and his C-reactive protein level was 268 mg/L. Before antimicrobial drug therapy with amoxicilline/clavulanic acid and ciprofloxacin was begun, blood cultures were taken. One of the 2 anaerobic blood cultures was positive. Gram-negative bacilli were isolated (strain 4401054). Antimicrobial drug resistance was detected only to vancomycin. After receiving this therapy, the patient recovered rapidly.

Biochemical characterization was conducted by using API 20A and rapid ID 32A strips (bioMérieux, Marcy l'Etoile, France). Results were compared with those obtained for the reference strain *A. finegoldii* CIP 107999^T. Strains 3302398 and 4401054 were indole positive and bile resistant, and they had positive enzyme reactions for N-acetyl-β-glucosaminidase, α-galactosidase, and β-galactosidase, as described for *A. finegoldii* (*4*). The 2 strains produced a brown pigment after 2 weeks' incubation on sheep blood agar plates (bioMérieux).

PCR amplification of the 16S rDNA was performed with the primer pair fD1/rp2 (*5*). The generated fragments were sequenced as previously described (*6*). Sequences were compared with those available in GenBank databases by using BLAST (www.ncbi.nlm.nih.gov/blast). They showed a 97% identity to the 16S rDNA of *A. finegoldii* (accession nos. AY643083 and AY643084).

A novel bacterium was characterized from appendiceal tissues samples from children with appendicitis and in 2 cases of perirectal and brain abscesses associated with other anaerobes (*1*). With routine tests, this organism resembled members of the *Bacteroides fragilis* group; however, the cellular fatty acid composition dominated by *iso*-C15:0 and production of brown pigment on media containing hemolyzed blood suggested that the organism was most closely related to the genus *Porphyromonas* (*1*). However, 16S rDNA sequence comparison showed highest sequence relatedness with *B. putredinis,* and the reclassification of *B. putredinis* in a novel genus, *Alistipes*, and the classification of the novel bacterium as *A. finegoldii* were proposed (*4*). *A. putredinis* was characterized in the indigenous flora of the human gut (*7*). The natural habitat of *A. finegoldii* is unknown but is probably the same. *B. fragilis* is the most frequent anaerobic bacterium isolated from blood samples, and the principal source of the bacteria is the gastrointestinal tract (*8*). Predisposing factors to *Bacteroides* species bacteremia include malignant neoplasms, recent gastrointestinal or obstetric-gynecologic surgery, intestinal obstruction, and use of cytotoxic agents or corticosteroids (*8*). In both of our patients, fever was noted and no other microorganisms were isolated, indications that the bacteria probably were pathogenic.

Phenotypic identification of *Alistipes* sp. is difficult in a routine microbiology laboratory. However, a molecular approach based on 16S rRNA gene sequence comparison is a good method for identifying anaerobic bacteria, as it has recently been reported for *B. fragilis* in anaerobic sepsis (*9*) and for *B. thetaiotaomicron* from a patient with a cholesteatoma and purulent meningitis (*10*). In our 2 patients, we also used molecular identification because *A. finegoldii* was not included in the API phenotypic database identification. *A. finegoldii* should be considered as an agent of bacteremia in patients with gastrointestinal pathologic conditions.

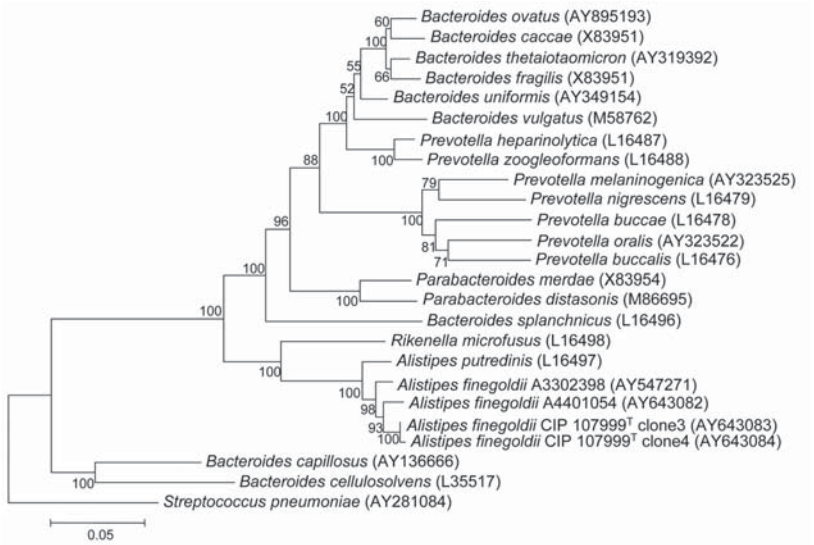

Figure. Phylogenetic tree inferred from comparison of the 16S rRNA gene sequences of genera *Bacteroides*, *Parabacteroidetes*, *Prevotella,* and *Alistipes*. Nucleotide accession numbers for the sequences used to construct this dendrogram are given in parentheses. The tree was constructed with MEGA version 2.1 (www.megasoftware.net). Distance matrices were determined following the assumptions described by Kimura (*3*) and were used to elaborate the dendrogram with the neighbor-joining method. Bar, 0.05-nt change per nucleotide position. *Streptococcus pneumoniae* was used as the outgroup.

This work was supported by grant PBBSB-102600 from the Swiss National Science Foundation.

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References

1. Rautio M, Lonnroth M, Saxen H, Nikku R, Väisanen ML, Finegold SM, et al. Characteristics of an unusual anaerobic pigmented gram-negative rod isolated from normal and inflamed appendices. Clin Infect Dis. 1997;25(Suppl 2):S107–10.
2. Rautio M, Saxen H, Siitonen A, Nikku R, Jousimies-Somer H. Bacteriology of histopathologically defined appendicitis in children. Pediatr Infect Dis J. 2000;19:1078–83.
3. Kimura M. A simple method for estimating evolutionary rates of base substitutions through comparative studies of nucleotide sequences. J Mol Evol. 1980;16:111–20.
4. Rautio M, Eerola E, Väisänen-Tunkelrott ML, Molitoris D, Lawson P, Collins MD, et al. Reclassification of *Bacteroides putredinis* (Weinberg et al., 1937) in a new genus *Alistipes* gen. nov., as *Alistipes putredinis* comb.nov., and description of *Alistipes finegoldii* sp. nov., from human sources. Syst Appl Microbiol. 2003;26:182–8.
5. Weisburg WG, Barns SM, Pelletier DA, Lane DJ. 16S ribosomal DNA amplification for phylogenetic study. J Bacteriol. 1991;173:697–703.
6. Fenner L, Roux V, Mallet MN, Raoult D. *Bacteroides massiliensis* sp. nov., isolated from blood culture of a newborn. Int J Syst Evol Microbiol. 2005;55:1335–7.
7. Rigottier-Gois L, Rochet V, Garrec N, Suau A, Doré J. Enumeration of *Bacteroides* species in human faeces by fluorescent in situ hybridisation combined with flow cytometry using 16S rRNA probes. Syst Appl Microbiol. 2003;26:110–8.
8. Brook I. Clinical review: bacteremia caused by anaerobic bacteria in children. Crit Care. 2002;6:205–11.
9. Wareham DW, Wilks M, Ahmed D, Brazier JS, Millar M. Anaerobic sepsis due to multidrug-resistant *Bacteroides fragilis*: microbiological cure and clinical response with linezolid therapy. Clin Infect Dis. 2005;40:67–8.
10. Feuillet L, Carvajal J, Sudre I, Pelletier J, Thomassin JM, Drancourt M, et al. First isolation of *Bacteroides thetaiotaomicron* from a patient with a cholesteatoma and experiencing meningitis. J Clin Microbiol. 2005;43:1467–9.

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Shiga Toxin–producing *Escherichia coli*, Idaho

To the Editor: Data collected from expanded surveillance study suggest that more than half of Idaho Shiga toxin–producing *Escherichia coli* (STEC) illnesses are caused by non-O157 serotypes. Using data from a regional medical center whose stool culture protocol included Shiga toxin testing, we predicted Idaho's STEC incidence to be significantly higher if non-O157 STEC *E. coli* were routinely detected by immunoassay. Recent findings suggest that the prediction was accurate in an expanded surveillance area.

Several studies have shown an increased incidence of non-O157 STEC infections in the United States. For example, a community hospital in Virginia detected non-O157 serotypes in 31% of patients with STEC from 1995–2002 (*1*). A 1998 Nebraska study that analyzed 30,000 diarrheal stool samples found that non-O157 and O157:H7 STEC were equally prevalent (*2*). Additionally, findings from a Connecticut study of laboratory-confirmed cases (*3*), STEC surveillance results from Montana (*4*), and a recent study from Michigan (*5*) indicate that non-O157 serotypes comprise a substantial percentage of STEC cases.

In other countries, nonculture-based methods are routinely used for STEC detection (*6*). However, *E. coli* O157:H7 culture methods remain the focus in the United Kingdom, Canada, and the United States (*6*). Reliance on culture methods can result in misleading interpretations of STEC prevalence. For example, 93% of STEC infections in Canada are reported to be *E. coli* O157:H7, yet a Manitoba 1992 study showed that when toxin assays were used, 35% of the recovered STEC isolates were non-O157 serotypes (*6*).

Analysis of reported non-O157 STEC cases in Idaho showed a similar trend. From 2002–2004, 66% of Idaho's non-O157 cases originated in Health District 7, where >70% of stool cultures are screened by enzyme immunoassay (EIA) for Shiga toxin (Premier EHEC, Meridian Bioscience, Cincinnati, OH, USA). This rate was disproportionately higher than that of the remaining 6 health districts, which primarily use culture methods to screen for *E. coli* O157:H7. We hypothesized that this disproportion was due to differences in stool culture protocol. To test this premise, we conducted enhanced surveillance for 16 months in a "low" STEC incidence area, Health District 5. A total of 2,065 stools submitted for culture were screened for Shiga toxin by EIA. With this approach, reported non-O157 STEC incidence rose from <1 case/year/100,000 population to 11 cases/year/100,000 population. Additionally, 56% of recovered STEC isolates were non-O157 serotypes, mirroring the proportion of non-O157 detected in District 7. Notably, this appears to be the endemic rate for District 5 because no non-O157 STEC outbreaks or matching pulsed-field gel electrophoresis patterns were detected during the surveillance period. Although our study captured only a portion of stool cultures in Idaho, our findings