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 *Aedes* infestation levels. We are stepping up our applied field research to prevent future outbreaks of chikungunya fever, as well as dengue fever.

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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 abcess material (1,2). 16S rRNA 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 ileoceleal 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-coile 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, 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. 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.

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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 surveillanc e 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, nunculture-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 (PremierEHEC, 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