should be initiated immediately because β-lactam antimicrobial drugs are inefficient for the treatment of rickettsioses (9).

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Pandemic Vibrio parahaemolyticus, Maryland, USA, 2012

To the Editor: Since 1996, an increasing number of infections caused by Vibrio parahaemolyticus strains belonging to a pandemic clonal complex (CC), CC3, typically O3:K6, have been observed worldwide (I–3); most of these strains are sequence type (ST) 3. In the summer of 1998, outbreaks linked to O3:K6 occurred in Galveston Bay, Texas, and Oyster Bay, New York, USA; the illnesses were associated with oyster consumption (4). Strains belonging to CC36 are the leading cause of V. parahaemolyticus infections in the United States. These strains are endemic to the West Coast (2) and have been historically linked to outbreak-associated V. parahaemolyticus infections caused by consumption of raw oysters harvested from the region (5).

In August 2012, a V. parahaemolyticus outbreak involving 6 persons occurred in Maryland, USA. The patients (members of 2 dining parties) had eaten in the same restaurant on the same day; raw and cooked seafood was served at the restaurant. Party A comprised 4 diners, of whom 2 had laboratory-confirmed illness and 2 were probable case-patients. Party B comprised 2 diners, of whom 1 had laboratory-confirmed illness and 1 was a probable case-patient. Probable case-patients were epidemiologically linked to confirmed case-patients, but V. parahaemolyticus was not detected in their stool samples. The epidemiologic investigation did not conclusively identify the specific food responsible for the outbreak. The affected diners had not eaten oysters, lobster, or mussels, but they had eaten cooked clams, fish, crab, and shrimp. Because the patients had not eaten oysters, a traceback investigation was not conducted. The outbreak possibly was caused by cross-contamination during food preparation. No other cases were reported from this restaurant or the surrounding area. V. parahaemolyticus was isolated from stool samples of 3 of the patients. The isolates were characterized by real-time PCR for virulence-related genes (tdh and trh). All 3 isolates were tdh positive and lacked the trh gene. Pulsed-field gel electrophoresis (PFGE) was run, using SfiI and NotI; the resulting K16S12.0138 (SfiI) and K16N11.0143 (NotI) patterns were indistinguishable. The PFGE pattern combination was queried against combination entries made in PulseNet (www.cdc.gov/pulsenet/) during February 4, 2010–April 16, 2013, and found to be indistinguishable from other clinical entries (online Technical

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The whole genomes of the 3 Maryland strains were sequenced by using the Ion Torrent personal genome machine (Life Technologies, Grand Island, NY, USA); in silico multilocus sequence typing (MLST) (2) showed that the isolates were all ST3, the most common ST belonging to CC3. Bioinformatic analysis of the whole genomes was conducted with the Bacterial Isolate Genome Sequence Database (BIGsdb) genome comparator tool available within the Vibrio parahaemolyticus MLST database (http://pubmlst.org/vparahaemolyticus) (7, 8). Results confirmed that these outbreak isolates were linked to the O3:K6 pandemic clone of V. parahaemolyticus (Figure). We identified 2,613 variable loci in this analysis by using as reference genome the prototype pandemic V. parahaemolyticus clonal strain RIMD2210633 (available from GenBank, www.ncbi.nlm.nih.gov/genome/?term=vibrio parahaemolyticus) (10). Differences in variable loci and the absence of certain genes indicated that, although indistinguishable by MLST and PFGE, these strains are easily differentiated from RIMD2210633 (online Technical Appendix 1). The draft genome sequences for the 3 strains are available at the V. parahaemolyticus MLST database (identification nos. 1187 [Vp16MD], 1188 [Vp17MD], and 1189 [Vp18MD]).

V. parahaemolyticus strains belonging to the pandemic CC have caused thousands of infections and a V. parahaemolyticus pandemic (3). Foodborne illnesses caused by pandemic V. parahaemolyticus are uncommonly reported in the United States. In Maryland, 12 and 21 cases of V. parahaemolyticus–associated gastroenteritis were reported in 2012 and 2013, respectively. We report that the pandemic CC was still causing US outbreaks as recently as August 2012. It is possible that complete availability of PFGE patterns during the outbreaks (online Technical Appendix 1) could have provided additional insight into the scope of the outbreak and implicated food sources. The application of rapid, whole-genome sequencing technology aided our discovery that the Maryland outbreak strains were part of the pandemic CC and likely related to V. parahaemolyticus strains that shared common PFGE patterns and that were reported as the cause of illnesses in several states around the same time as the Maryland outbreak.

The presence of this virulent V. parahaemolyticus strain in Maryland is an ongoing public health concern, requiring continued microbiological surveillance. This pandemic strain also indicates the need for establishing a V. parahaemolyticus genome database that is accessible worldwide. Such a database would enable improved tracking and faster responses to emergent and dangerous pandemic clonal strains.
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**Serologic Evidence of Leptospirosis in Humans, Union of the Comoros, 2011**

To the Editor: Leptospirosis is a worldwide bacterial zoonosis caused by infection with pathogenic *Leptospira* spp. (*Spirochaetales, Leptospiraceae*). Most mammals can be infected, but rats are considered the main reservoir, maintaining *Leptospira* spirochetes in the lumen of renal tubules and contaminating the environment with bacteria-infected urine. Transmission to humans is accidental, occurring through contact with animal secretions or with contaminated environmental materials.

In temperate countries, human leptospirosis is a sporadic disease; incidence is much higher in the tropics because climate and environmental conditions are conducive to the survival of bacteria, resulting in increased exposure of humans to leptospirosis-causing pathogens (1). Among islands in the southwestern Indian Ocean, human leptospirosis is endemic to Mayotte, France, and La Réunion (2–4) and to the Seychelles, where the incidence of leptospirosis is one of the highest worldwide (5). Leptospirosis is poorly documented in other islands in the region, including Mauritius, Madagascar, and the Union of the Comoros (2,6–8). Whether the scant documentation indicates underdiagnosis or reflects local epidemiologic specificities is unknown. To improve knowledge of *Leptospira* infection in the region, we conducted a study in the Union of the Comoros to serologically assess the presence or absence of leptospirosis in humans. The Union of the Comoros consists of 3 islands: Grande-Comore, Mohéli, and Anjouan. Together with a fourth, southern island, Mayotte, these islands form the Comoros Archipelago.

For feasibility reasons, we used excess serum samples. Seventy-six samples were from healthy volunteers who gave informed consent; 318 clinical blood samples from patients had been obtained by private laboratories and by the surveillance laboratory of the National Malaria Control Programme (PNLP) during August 1–October 8, 2011. The Ministère de la Santé, de la Solidarité et de la Protection du Genre of the Union of the Comoros, authorized the serologic investigation (authorization no. 1175/MSSPG/DNS).

We used the microscopic agglutination test (MAT) to test serum samples; the MAT was based on a panel of 15 *Leptospira* strains, enabling the screening of all recently reported serogroups for human and animal cases on neighboring Mayotte (2,4,9). A list of the tested strains follows, shown as *Genus species* Serogroup/Serovar (type strain): *L. borgdorferi* Ballum/Castellonis (Castellon 3), *L. borgdorferi* Sejroe/Hardjobovis (Sponselee), *L. borgdorferi* Sejroe/Sejroe (M 84),
## Technical Appendix

Table. Isolates that matched PFGE patterns K16S12.0138 and K16N11.0143 combination against PulseNet entries uploaded during February 4, 2010–April 16, 2013 and associated data*

| Isolate          | Source state | Source type | Patient age, y | Patient sex | Isolate date | Upload date | PFGE-SfiI-pattern | PFGE-NotI-pattern |
|------------------|--------------|-------------|----------------|-------------|--------------|-------------|------------------|------------------|
| TX__TXACB1000107 | TX           | Human       | 42             | M           | 1/15/2010    | 2/4/2010    | K16S12.0138      | K16N11.0143      |
| CDC__AM47597     | WA           | Human       | 33             | M           | 1/15/2010    | 2/4/2010    | K16S12.0138      | K16N11.0143      |
| CDC__AM47598     | WA           | Human       | 38             | F           | 7/11/2011    | 10/19/2011  | K16S12.0138      | K16N11.0143      |
| CDC__101215235   | NY           | Human       | 33             | M           | 7/9/2011     | 8/2/2012    | K16S12.0138      | K16N11.0143      |
| CDC__Pi11200004  | AZ           | Human       | 38             | F           | 7/11/2011    | 10/15/2012  | K16S12.0138      | K16N11.0143      |
| CDC__AM47605     | WA           | Human       | 38             | F           | 7/11/2011    | 10/15/2012  | K16S12.0138      | K16N11.0143      |
| TX__TXACB1102640 | TX           | Human       | 42             | M           | 8/8/2011     | 9/8/2011    | K16S12.0138      | K16N11.0143      |
| CDC__AM47611     | WA           | Human       | 33             | M           | 8/18/2011    | 11/2/2011   | K16S12.0138      | K16N11.0143      |
| CDC__AM47614     | WA           | Human       | 38             | F           | 8/18/2011    | 11/2/2011   | K16S12.0138      | K16N11.0143      |
| CDC__AM47617     | WA           | Human       | 27             | M           | 9/23/2011    | 8/2/2012    | K16S12.0138      | K16N11.0143      |
| CDC__Pi11272013  | AZ           | Human       | 39             | M           | 6/15/2012    | 7/23/2012   | K16S12.0138      | K16N11.0143      |
| CA__M12X02339    | CA           | Human       | 39             | M           | 7/6/2012     | 7/27/2012   | K16S12.0138      | K16N11.0143      |
| MD__MDA12147539  | MD           | Human       | 53             | M           | 8/21/2012    | 9/9/2012    | K16S12.0138      | K16N11.0143      |
| MD__MDA12148581  | MD           | Human       | 49             | F           | 8/22/2012    | 9/9/2012    | K16S12.0138      | K16N11.0143      |
| MD__MDA12162046  | MD           | Human       | 51             | F           | 8/24/2012    | 9/9/2012    | K16S12.0138      | K16N11.0143      |
| TX__TXACB1202640 | TX           | Human       | 67             | F           | 8/20/2012    | 10/10/2012  | K16S12.0138      | K16N11.0143      |
| CDC__Pi12255002  | AZ           | Human       | 49             | M           | 3/29/2013    | 4/16/2013   | K16S12.0138      | K16N11.0143      |
| CDC__Pi12256006  | AZ           | Human       | 49             | M           | 3/29/2013    | 4/16/2013   | K16S12.0138      | K16N11.0143      |

* PFGE, pulsed-field gel electrophoresis; NK, not known.