for 2014. This prevalence is increasing compared to that described in France in 2012 (14%). We found 35 A–G substitutions at position 2058 or 2059, two A2062T mutations and one A2059C mutation (Table 1,9). Notably, in patients 15 and 33, who were infected with strains with macrolide resistance–associated mutations, *M. genitalium* infection was unsuccessfully treated with azithromycin, with treatment failures after azithromycin (1 g) and extended azithromycin (1.5 g for 5 d), but moxifloxacin treatment was effective. Patient 15 had been treated 1 year earlier with azithromycin (1 g) for nongonococcal urethritis.

Among the 168 patients whose isolates were examined for the 23S rRNA, gyrA, and parC genes, strains from 2 patients (patients 3 and 6) had both macrolide- and fluoroquinolone-associated mutations (1.2%; 95% CI 0.33%–4.24%). Both patients received azithromycin (1 g), and patient 6 received additional azithromycin (1.5 g) after failure of azithromycin (1 g). Patient 6 experienced azithromycin failure again after the extended regimen. *M. genitalium* multidrug resistance is described in France at a prevalence of 1.2%, lower than prevalence described in Australia (7.5%) (7) and Japan (30.8%) (10).

In conclusion, *M. genitalium* fluoroquinolone resistance is emerging in France, with a prevalence of 6% in 2013–2014. Further, macrolide resistance also increased during this period, to a rate of 17.2%. Patients infected with *M. genitalium* strains containing both macrolide and fluoroquinolone resistance mutations associated with therapeutic failure raise concerns about untreatable *M. genitalium* infections.

Acknowledgments
We thank Manon Zerbib and Manon Passard for technical assistance.

References

Address for correspondence: Cécile Bébéar, USc EA 3671, Mycoplasmal and Chlamydial Infections in Humans, University of Bordeaux, Campus Bordeaux Carreire, 146 rue Léo Saignat, 33076 Bordeaux CEDEX, France; email: cecile.bebear@u-bordeaux.fr

Possible Transmission of *mcr-1*–Harboring *Escherichia coli* between Companion Animals and Human

Xue-Fei Zhang, Yohei Doi, Xi Huang, Hong-Yu Li, Lan-Lan Zhong, Kun-Jiao Zeng, Yan-Fen Zhang, Sandip Patil, Guo-Bao Tian

Author affiliations: Sun Yat-Sen University Zhongshan School of Medicine, Guangzhou, China (X.-F. Zhang, X. Huang, L.-L. Zhong, K.-J. Zeng, Y.-F. Zhang, S. Patil, G.-B. Tian); Ministry of Education Key Laboratory of Tropical Diseases Control, Guangzhou (X.-F. Zhang, X. Huang, L.-L. Zhong, K.-J. Zeng, Y.-F. Zhang, S. Patil, G.-B. Tian); University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA (Y. Doi); Sun Yat-Sen University Memorial Hospital, Guangzhou (H.-Y. Li)

DOI: http://dx.doi.org/10.3201/eid2209.160464

To the Editor: Plasmid-mediated, colistin-resistance mechanism gene *mcr-1* was first identified in *Escherichia coli* isolates from food, food animals, and human patients in November 2015 (1). Reports on detection of *mcr-1* in *Enterobacteriaceae* from humans and food animals
soon followed from ≈12 countries (2–5). Here we report detection of mcr-1 in colistin-resistant *E. coli* isolated from companion animals and the possible transmission of mcr-1–harboring *E. coli* between companion animals and a person.

Three mcr-1–harboring *E. coli* clinical isolates were identified from specimens of 3 patients admitted to a urology ward of a hospital in Guangzhou, China. *E. coli* isolate EC07 was identified in the urine of a 50-year-old male patient with glomerulonephritis in October 2015. Isolate EC08 was cultured from the urine of a 48-year-old male patient with bladder cancer 3 weeks after EC08 was identified. Review of medical records identified the patient carrying *E. coli* isolate EC07 as a worker at a pet shop. In light of this finding, we collected a total of 53 fecal samples from 39 dogs and 14 cats in the pet shop where the man worked. We isolated and identified colonies consistent with *E. coli* from fecal samples on MacConkey agar plates (Thermo Fisher, Beijing, China) and API 20E system (bioMérieux, Durham, NC, USA). We prepared crude DNA samples of isolates for PCR testing by boiling cells in water. Among them, 6 were positive for mcr-1 by PCR and sequencing (4 from dogs and 2 from cats). All 6 isolates were resistant to colistin, polymyxin B, cephalosporin, gentamicin, and ciprofloxacin by using the agar dilution method, in accordance with the European Committee on Antimicrobial Susceptibility Testing (http://www.eucast.org) for colistin and polymyxin B and Clinical and Laboratory Standards Institute guidelines (http://www.clsi.org) for the other antimicrobial drugs. We identified various resistance genes accounting for the multidrug resistance in these 9 mcr-1–positive isolates (6, 7) (Table). We noted that *E. coli* isolate EC09 was also resistant to carbapenems and positive for *bla*<sub>IMP-4</sub>. We observed co-production of *mcr-1* and IMP-type metallo-β-lactamase in *E. coli*.

We subjected all isolates to multilocus sequence typing, in accordance with the protocol described at http://mlst.warwick.ac.uk/mlst/dbs/Ecoli, and pulsed-field gel electrophoresis as described previously (8–10). We identified 5 mcr-1–positive isolates from 4 dogs (PET02–04 and PET06) and isolate EC07 as sequence

### Table. Characteristics of 9 mcr-1–positive *Escherichia coli* isolates from companion animals and human patients, Guangzhou, China*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Isolate</th>
<th>PET01</th>
<th>PET02</th>
<th>PET03</th>
<th>PET04</th>
<th>PET05</th>
<th>PET06</th>
<th>EC07</th>
<th>EC08</th>
<th>EC09</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specimen source</td>
<td>Cat</td>
<td>Dog</td>
<td>Dog</td>
<td>Cat</td>
<td>Dog</td>
<td>Human</td>
<td>Human</td>
<td>Human</td>
<td>Human</td>
<td></td>
</tr>
<tr>
<td>Specimen type</td>
<td>Cat</td>
<td>Dog</td>
<td>Dog</td>
<td>Cat</td>
<td>Dog</td>
<td>Urine</td>
<td>Urine</td>
<td>Blood</td>
<td>Blood</td>
<td></td>
</tr>
<tr>
<td>Phylogenetic group</td>
<td>B2</td>
<td>D</td>
<td>D</td>
<td>B2</td>
<td>D</td>
<td>D</td>
<td>B1</td>
<td>B1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ST</td>
<td>ST93</td>
<td>ST354</td>
<td>ST354</td>
<td>ST354</td>
<td>New</td>
<td>ST354</td>
<td>ST354</td>
<td>ST156</td>
<td>ST156</td>
<td></td>
</tr>
<tr>
<td>PFGE type</td>
<td>IV</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td></td>
</tr>
<tr>
<td>Resistance genes</td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>qepA</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fosA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fosA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fosA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fosA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fosA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fosA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>fasA3</em>, <em>aac(6’)-Ib-cr</em></td>
<td><em>mcr-1</em>, <em>bla</em>&lt;sub&gt;TEM-1&lt;/sub&gt;, <em>bla</em>&lt;sub&gt;CTX-M-15&lt;/sub&gt;, <em>aosA3</em>, <em>mtfB</em>, <em>qepA1</em></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>MIC, μg/mL</th>
<th>Colistin</th>
<th>Polymyxin B</th>
<th>Ampicillin</th>
<th>AMX/CLV</th>
<th>Cefotaxime</th>
<th>Ceftazidime</th>
<th>Cefepime</th>
<th>Gentamicin</th>
<th>Amikacin</th>
<th>Ertapenem</th>
<th>Imipenem</th>
<th>Meropenem</th>
<th>Fosfomycin</th>
<th>Tigecycline</th>
<th>Nitrofurantoin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
<td>256</td>
</tr>
<tr>
<td></td>
<td>64</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
<td>&gt;256</td>
</tr>
<tr>
<td></td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
<td>&lt;0.25</td>
</tr>
<tr>
<td></td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
</tr>
<tr>
<td></td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
<td>&lt;2</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>32</td>
<td>32</td>
<td>32</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>128</td>
</tr>
<tr>
<td></td>
<td>256</td>
<td>128</td>
<td>128</td>
<td>128</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
<td>64</td>
</tr>
</tbody>
</table>

*AMX/CLV, amoxicillin clavulanic acid; PFGE, pulsed-field gel electrophoresis; ST, sequence type.
†By multilocus sequence typing.
type (ST) 354. Isolates PET01 and PET05, identified from cats, belonged to ST93 and a new ST strain, respectively. Isolates EC08 and EC09, from the patients who shared the same hospital room with the pet shop worker, were ST156 (Table). Results of pulsed-field gel electrophoresis were consistent with multilocus sequence typing results and showed that isolates consisted of 5 types (types I to V; online Technical Appendix, http://wwwnc.cdc.gov/EID/article/22/9/16-0464-Techapp1.pdf). Isolate EC07 was clonally related to 4 E. coli strains from dogs, according criteria described by Tenover et al. (10), suggesting possible transmission of mcr-1–harboring E. coli between dogs and the patient. Colistin resistance was successfully transferred to E. coli C600 through conjugation in all isolates, suggesting that mcr-1 was located on transferable plasmids.

These findings suggest that mcr-1–producing E. coli can colonize companion animals and be transferred between companion animals and humans. The findings also suggest that, in addition to food animals and humans, companion animals can serve as a reservoir of colistin-resistant E. coli, adding another layer of complexity to the rapidly evolving epidemiology of plasmid-mediated colistin resistance in the community.

Acknowledgments
We sincerely thank the patients and the owners of companion animals for giving written consent for publication.

This work was supported by research grants from the National Natural Science Foundation of China (no. 81471988), the 111 Project (nos. B13037 and B12003), the Guangdong Natural Science Foundation (no. S201310015810), and the Program of Science and Technology New Star of Guangzhou (no. 2014J2200038).

References

Address for correspondence: Guo-Bao Tian, Program of Immunology, Institute of Human Virology, Institute of Tuberculosis Control, Zhongshan School of Medicine, Sun Yat-Sen University, 74 Zhongshan 2nd Rd, Guangzhou 510080, China; email: tiangb@mail.sysu.edu.cn

Acetobacter indonesiensis
Bacteremia in Child with Metachromatic Leukodystrophy

Rebekka Kohlmann, Karin Barenberg, Agnes Anders, Sören G. Gatermann

Author affiliations: Ruhr-Universität Bochum, Bochum, Germany (R. Kohlmann, A. Anders, S.G. Gatermann); Institute of Medical Laboratory Diagnostics Bochum, Bochum (R. Kohlmann, S.G. Gatermann); Marienhospital Herne, Herne, Germany (K. Barenberg)

DOI: http://dx.doi.org/10.3201/eid2209.160566

To the Editor: Acetobacter indonesiensis, first described in 2000 (1), belongs to the group of acetic acid bacteria (AAB), which includes the genera Acetobacter, Gluconobacter, Asaia, Granulibacter, and others in the family Acetobacteriaceae. AAB are of great industrial interest for use in vinegar fermentation processes because they oxidize alcohols or sugars incompletely, which leads
Possible Transmission of *mcr-1*–Harboring *Escherichia coli* between Companion Animals and Human

**Technical Appendix**

**Technical Appendix Figure.** Pulsed-field gel electrophoresis analysis of 9 *mcr-1*-producing *Escherichia coli* isolates from companion animals and human patients, Guangzhou, China.