tests from Panama City versus other areas of Panama and might result in a sampling bias. Despite these limitations, the recent Zika outbreak has shown the speed at which vectorborne diseases can spread and highlights the importance of detecting emerging viruses like PTVs.

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mcr-1 Colistin Resistance in ESBL-Producing Klebsiella pneumoniae, France

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We report intestinal carriage of an extended-spectrum β-lactamase–producing Klebsiella pneumoniae strain with high-level resistance to colistin (MIC 24 mg/L) in a patient in France who had been hospitalized for fungal meningitis. The strain had the mcr-1 plasmid gene and an inactivated mgrB gene, which are associated with colistin resistance.
R esistance to colistin in gram-negative bacteria stems mainly from structural modifications of bacterial lipopolysaccharide. These modifications include addition of 4-amino-4-deoxy-L-arabinose or phosphoethanolamine caused by chromosomal mutations in genes encoding the 2-component systems PhoPQ and PmrAB, or mutations in the mcr-1 gene, a negative regulator of PhoPQ (1).

The recent discovery of a horizontally transferable plasmid-mediated mcr-1 gene encoding a phosphoethanolamine transferase is a cause for concern, but few mcr-1–positive clinical strains of Klebsiella pneumoniae have been reported so far in Europe (2). Colocalization of carbapenemases or extended-spectrum β-lactamase (ESBL) genes and the mcr-1 gene on the same plasmids is of concern because it might lead to pandrug resistance (1,3). We report mcr-1 colistin resistance in ESBL-producing K. pneumoniae isolated from a patient in France.

The patient was a 38-year-old man who had chronic granulomatous disease that was diagnosed when he was 8 months old. Since then, he has had several minor and major diseases and conditions, including primitive femoral osteitis, hepatic abscesses, disseminated candidiasis, and bacteremia, which required several treatments with antimicrobial drugs. However, the patient was never given colistin.

In April 2016, he was hospitalized for surgical removal of a thyroid abscess. Fungal cultures of the abscess grew Aspergillus fumigatus. Despite antifungal treatment with amphotericin B and fluorocytosine, fungal meningitis, cerebral arterial vasospasm at the Willis polygon, and hydrocephalus developed. The patient also received immunosuppressive therapy (methyprednisolone and anakinra) and empiric antimicrobial drug therapy, including cotrimoxazole, clindamycin, meropenem, and vancomycin successively.

In August 2016, systematic culture of a rectal swab specimen showed an ESBL-producing strain of K. pneumoniae; 2 previous rectal screenings showed negative results. The strain was resistant to colistin (MIC >4 mg/L). Resistance was determined by using a broth microdilution assay (BD Phoenix Instrument; Becton Dickinson, Franklin Lakes, NJ, USA) (online Technical Appendix). None of these were localized with the mcr-1 gene on the IncX4 plasmid. Moreover, insertion of mobile element IS5 in the mgrB gene was detected, which is also associated with colistin resistance (3). No mutations were found in the prmA, prmB, phoP, and phoQ genes.

Table. Resistance genes identified by whole-genome sequencing of an ESBL-producing mcr-1–positive Klebsiella pneumoniae strain isolated from a 38-year-old man, France*

<table>
<thead>
<tr>
<th>Resistance gene</th>
<th>Target antimicrobial drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>mcr-1 and inactivation of mgrB by IS5 insertion</td>
<td>Colistin</td>
</tr>
<tr>
<td>bla&lt;sub&gt;SHV-106&lt;/sub&gt;</td>
<td>β-lactams</td>
</tr>
<tr>
<td>aac(3)-IId and aadA16-like</td>
<td>Aminoglycoside</td>
</tr>
<tr>
<td>aac(6’)-Ib-cr</td>
<td>Quinolone and aminoglycoside</td>
</tr>
<tr>
<td>fosA5</td>
<td>Fosfomycin</td>
</tr>
<tr>
<td>sul1 and folP</td>
<td>Sulfonamide</td>
</tr>
<tr>
<td>dfrA27</td>
<td>Trimethoprim</td>
</tr>
<tr>
<td>tetD</td>
<td>Tetracycline</td>
</tr>
</tbody>
</table>

*ESBL, extended-spectrum β-lactamase.
CNA+ medium did not identify intestinal carriage of ESBL-negative but mcr-1−positive enterobacteria in the index case-patient. On the basis of these results, rectal screening of 39 contacts was performed by using an ESBL-screening medium (BLSE agar [MacConkey agar and Drigalski agar], bioMérieux). All of the tests showed negative results.

The origin of the mcr-1 strain remains unknown. Nosocomial acquisition cannot be ruled out because colistin-resistant strains harboring the mcr-1 gene might have been isolated in the hospital but not identified because this resistance mechanism was initially reported in February 2016. Food might also be incriminated (I); one study identified a 21% mcr-1 prevalence among ESBL-producing E. coli in calves in France (10).

Multiple antimicrobial drug therapy for this patient might have selected for this multidrug-resistant bacteria. The presence of a plasmid containing the mcr-1 and ESBL or other resistance genes in the same strain might be involved in selection of colistin-resistant strains during administration of any ineffective antimicrobial drug (3). Development of efficient tools for rapid detection of mcr-1−harboring strains should be a priority to prevent dissemination of these strains in hospital settings.

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Chromosomal 16S Ribosomal RNA Methyltransferase RmtE1 in Escherichia coli Sequence Type 448

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We identified rmtE1, an uncommon 16S ribosomal methyltransferase gene, in an aminoglycoside- and cephalosporin-resistant Escherichia coli sequence type 448 clinical strain co-harboring bla<sub>KPC-2</sub>. Long-read sequencing revealed insertion of a 101,257-bp fragment carrying both resistance genes to the chromosome. Our findings underscore E. coli sequence type 448 as a potential high-risk multidrug-resistant clone.

RmtE (RmtE1 and its variant RmtE2) is an uncommon plasmid-mediated 16S rRNA methyltransferase (16S RMTase) found in gram-negative bacteria; only 4 strains have been reported to produce RmtE, all Escherichia coli, including 1 from the University of Pittsburgh Medical
**mcr-1 Colistin Resistance in ESBL-Producing *Klebsiella pneumoniae*, France**

**Technical Appendix**

**Sequencing of the *Klebsiella pneumoniae* Strain Genome and Plasmid Carrying the *mcr-1* Gene**

We used a whole-genome sequencing method (Illumina, San Diego, CA, USA) with 50-bp paired and 60× coverage. Gaps in the plasmid carrying the *mcr-1* gene were filled by using PCR and Sanger sequencing.

**Characteristics of the SHV-106 Plasmid**

Whole-genome sequencing identified a 57-kb plasmid that belonged to incompatibility group IncR and carried the *bla_{SHV-106}* gene. Genomic data were confirmed by extraction of plasmids according to the method of Kado and Liu (1) and hybridization with SHV and IncR probes.

**Antimicrobial Drug Susceptibilities of the *K. pneumoniae* Strain**

Antimicrobial drug susceptibilities were determined by using the BD Phoenix Instrument (Becton Dickinson, Franklin Lakes, NJ, USA). The strain showed susceptibility to amoxicillin/clavulanate (MIC 8/2 mg/L), piperacillin/tazobactam (≤4/4 mg/L), temocillin (8 mg/L), cefoxitin (≤4 mg/L), cefepime (≤1 mg/L), aztreonam (≤1 mg/L), ertapenem (≤0.25 mg/L), imipenem (≤0.25 mg/L), meropenem (≤0.125 mg/L), amikacin (≤4 mg/L), tigecycline (1 mg/L), and fosfomycin (32 mg/L); intermediate susceptibility to ticarcillin/clavulanate (16/2 mg/L) and ceftazidime (2 mg/L); and resistance to ampicillin (>8 mg/L), piperacillin (>64 mg/L), ceftriaxone (4 mg/L), tobramycin (>4 mg/L), gentamicin (>4 mg/L), nalidixic acid (>16 mg/L), ciprofloxacin (>1 mg/L), levofloxacin (>2 mg/L), norfloxacin (>2 mg/L), colistin (>4 mg/L), and trimethoprim/sulfamethoxazole (>4/76 mg/L).

**Reference**