died at 5 dpi. The dead piglets showed similar lesions to those of the piglets infected with YS isolate in the stomach, intestines, and mesenteric lymph nodes. Piglets in the control group orally inoculated with Dulbecco minimal essential medium remained healthy during the experiment, and no obvious pathologic changes were observed.

Our investigation indicated that the recent diarrhea outbreaks were mainly caused by PEDV variants with novel genetic markers that distinguish them from classical strains. The YS and ZB isolates were highly virulent in piglets. Unlike CV777, the PEDV variants remained almost unchanged in the epithelium at positions 499–638; however, a 2-aa deletion, a 1-aa insertion, and 18 separate substitutions were identified in the epitope at positions 83–276 (7,8). These variations of amino acid sequences probably changed the immunogenicity of S protein and led to immunization failure of current commercial vaccines made from classical PEDV strains. However, how PEDV has evolved and varies in pig herds are not clear. Further studies, including extensive genomic sequence analyses and serologic cross-neutralization tests, should be conducted.

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References


New Delhi Metallo-β-Lactamase-1 in Carbapenem-Resistant Salmonella Strain, China

To the Editor: Carbapenem resistance in Enterobacteriaceae can occur through the production of carbapenem-hydrolyzing enzymes such as New Delhi metallo-β-lactamase-1 (NDM-1) (1). In recent years, plasmid-mediated NDM-1 has spread rapidly worldwide and into multiple Enterobacteriaceae species, such as Klebsiella pneumoniae and Escherichia coli (2).

NDM-1 has been reported in 2 strains of Salmonella spp., which were isolated from feces and urine specimens during screening for multidrug-resistant bacteria in patients from India (3,4). We report the isolation of 1 community-acquired NDM-1–bearing Salmonella strain isolated from a child with acute diarrhea.

The Salmonella strain was isolated from the feces of an 11-month-old girl at Lishui Central Hospital, Zhejiang Province, China, on July 25, 2012. Six days before admission, a fever ≤40°C, accompanied by a cough, developed in the patient. Four days before admission, physical examination showed fine rales in both lungs. The leukocyte count was 8,900 cells/μL, with 80% neutrophils. No obvious abnormalities were found on a chest radiograph.

The patient was given a diagnosis of acute bronchitis, and the condition was treated with parenteral cefoxitin for 3 days and parenteral piperacillin/tazobactam for 1 day, but fever persisted. Two days before admission, diarrhea (4–5 times/day with loose feces containing mucus and blood) developed. On admission day, fecal analysis showed 3–4 leukocytes and 1–3 erythrocytes per high-power field.
A *Salmonella* sp. was isolated from feces obtained at admission and identified as *S. enterica* subsp. *enterica* serovar Stanley by serotyping by the local Centers for Disease Control and Prevention.

The patient was then given a diagnosis of bacterial enteritis and received intravenous azithromycin and latamoxef. Fever and diarrhea resolved over the next 3 days. On the fifth day of hospitalization, a fecal culture was negative for *Salmonella* spp. and the patient was discharged. At a follow-up visit 3 months later, *Salmonella* spp. or other carbapenem-resistant bacteria were not isolated from feces samples from the patient or her grandmother and brother, who lived with her.

The patient and her family had not traveled to any country during the year, including countries with a high prevalence of NDM-1 producers. The patient was living in a small rural village in southern China and did not have a special diet. She was healthy before hospitalization for fever. She was born by cesarean section and did not have contacts with hospitalized patients.

MICs of antimicrobial drugs were determined by agar dilution and interpreted by using revised Clinical and Laboratory Standards Institute breakpoints (5). The *Salmonella* Stanley strain was resistant to all β-lactam antimicrobial drugs tested, including cephalosporins and carbapenems, but susceptible to chloramphenicol, ciprofloxacin, tetracycline, and fosfomycin, and had azithromycin MICs of 4 µg/mL (Table).

![Table. Antimicrobial drug susceptibility of *Salmonella* strain Stanley and transconjugants containing New Delhi metallo-β-lactamase-1, China](image)

<table>
<thead>
<tr>
<th>Drug</th>
<th>MIC, µg/mL</th>
<th><em>Salmonella</em> strain</th>
<th><em>Escherichia coli</em> C600</th>
<th><em>E. coli</em> C600 transconjugant</th>
<th><em>Klebsiella pneumoniae</em> 13883</th>
<th><em>K. pneumoniae</em> 13883 transconjugant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ceftazidime</td>
<td>&gt;128/2</td>
<td>1/2</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>2/8</td>
<td>&gt;128/2</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>&gt;128/2</td>
<td>&lt;0.06</td>
<td>128</td>
<td>&gt;128</td>
<td>1/2</td>
<td>&gt;128/2</td>
</tr>
<tr>
<td>Latamoxef</td>
<td>&gt;128/2</td>
<td>&gt;0.25</td>
<td>&gt;32</td>
<td>&gt;0.06</td>
<td>32</td>
<td>32</td>
</tr>
<tr>
<td>Cefepime</td>
<td>&gt;128/2</td>
<td>&gt;0.06</td>
<td>&gt;16</td>
<td>&gt;0.06</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Imipenem</td>
<td>&gt;128/2</td>
<td>&gt;0.06</td>
<td>&gt;8</td>
<td>&gt;0.06</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>Meropenem</td>
<td>&gt;128/2</td>
<td>&gt;0.06</td>
<td>&gt;4</td>
<td>&gt;0.06</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Fosfomycin</td>
<td>&lt;0.06/2</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
</tr>
<tr>
<td>Minocycline</td>
<td>0.5/4</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>1</td>
<td>0.5</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>&lt;0.06/4</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
<td>&lt;0.06</td>
</tr>
<tr>
<td>Chloramphenicol</td>
<td>4/4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>4/4</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Trimethoprim/sulfamethoxazole</td>
<td>&gt;128/2</td>
<td>0.3/0.015</td>
<td>&gt;152/8</td>
<td>1.2/0.06</td>
<td>&gt;152/8</td>
<td>&gt;152/8</td>
</tr>
</tbody>
</table>

A modified Hodge test result for *Salmonella* strain Stanley was weakly positive. Production of metallo-β-lactamase was detected by using an imipenem-EDTA double-disk synergy test. Carbapenemase-encoding genes, including *bla*<sub>KPC-2</sub>, *bla*<sub>VIM</sub>, *bla*<sub>IMP</sub>, *bla*<sub>SPM-1</sub>, *bla*<sub>GIM</sub>, and *bla*<sub>NDM-1</sub>, were detected by using PCR as described (2). Only the *bla*<sub>NDM-1</sub> gene was detected (with primers 5′-GGCGGAATGGCTCATCACGCA-3′ and 5′-CGCAACA-CAGCCTGACTTTC-3′). The PCR product sequence was consistent with that of NDM-1 (GenBank accession no. FN396876).

Conjugation experiments were conducted as described (6). Carbapenem resistance could be transferred from *Salmonella* strain Stanley to *E. coli* C600 Rif<sup>r</sup> and *K. pneumoniae* 13883 Rif<sup>r</sup> at frequencies of 1 transconjugant per ≈1.0 × 10<sup>8</sup> and 4.0 × 10<sup>7</sup> bacterial cells, respectively, after exposure for 15 min.

Plasmid DNA was extracted by using a Plasmid Midi Kit (QIAGEN, Hilden, Germany) according to the manufacturer’s instructions. Electrophoresis showed that donor and transconjugant strains had the same plasmid profile; both contained an ≈140-kb plasmid. A PCR-based method for plasmid replicon typing (7) indicated that the plasmid belonged to incompatibility group IncA/C.

Experiments of *bla*<sub>NDM-1</sub> stability in *Salmonella* spp., *E. coli* transconjugants, and *K. pneumoniae* transconjugants were conducted by using the method of Wang et al. (6). Twenty colonies were randomly selected every day during days 2–15. Only 2 colonies of the *Salmonella* Stanley strain lost carbapenem resistance; these colonies were collected on the second and ninth days of passage, respectively. Plasmids containing *bla*<sub>NDM-1</sub> were not present in these 2 colonies. No transconjugants lost carbapenem resistance during 14 days of passage.

Although *Salmonella* spp. have shown increased resistance to cephalosporins and quinolones, resistance to carbapenems is rare (3,4,8,9). Because of emerging resistance to traditionally recommended antimicrobial agents, azithromycin is increasingly used for treatment of invasive *Salmonella* spp. infections in children (10). The patient with carbapenem-resistant *Salmonella* infection and acute diarrhea was cured by treatment with azithromycin.

This report indicates ongoing spread of NDM-1–bearing *Salmonella* strains. If one considers the high conjugation frequency and stability of the IncA/C plasmid containing NDM-1 in *Salmonella* spp., one
would conclude that it might increase spread of bacterial drug resistance. Prompt recognition of carbapenem-resistant *Salmonella* spp. and initiation of appropriate infection control measures are essential to avoid spread of these organisms.

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**Contagious Caprine Pleuropneumonia in Endangered Tibetan Antelope, China, 2012**

**To the Editor:** Contagious caprine pleuropneumonia is a severe respiratory disease of goats caused by *Mycoplasma capricolum* subsp. *capripleuropneumoniae* (Mcpp), a member of the *M. mycoides* cluster (1). Mcpp infection is associated with a 60% mortality rate and 90% illness rate, and the disease can cause substantial losses of livestock (1,2). We report a 2012 outbreak of contagious caprine pleuropneumonia in endangered Tibetan antelope (*Pantholops hodgsonii*) in China.

In 2000, the International Union of Conservation of Nature first listed the Tibetan antelope as an endangered species (3), and in 2004, the number of these antelope was estimated at 150,000 (4). Most Tibetan antelopes live on China’s Qinghai–Tibet Plateau at an altitude of 3,700–5,500 m (3).

During September–December 2012, ≈2,400 endangered Tibetan antelopes were found dead in the Naqu area of Tibet; the dead animals represented 16% of the 15,000 Tibetan antelopes thought to live in the area. Necropsy was performed on 13 of the antelopes at sites within the Shenzha, Shuanghu, and Nima localities of the Naqu area (online Technical Appendix Table 1, wwwnc.cdc.gov/EID/article/20/1/13-0067-Techapp1.pdf). Gross pathologic lesions were localized exclusively to the lung, where severe pleuropneumonia with partial heparatization was observed (Figure, panel A). The lungs of some affected antelope displayed a thickening of the interlobular septa, pleuritis, and an accumulation of straw-colored pleural fluid. The pleural exudate solidified to form a gelatinous covering on the lung (Figure, panel B).

Samples of lung tissue from 5 of the antelope were selected for histologic examination. Four of the samples showed fibrinous pneumonia with serofibrinous fluid and an inflammatory cell infiltrate consisting mainly of lymphocytes in the alveoli (Figure, panel C) and bronchioles (Figure, panel D). One sample showed pulmonary edema with a protein-rich fluid effusion in alveoli.

Lung tissue from each of the 13 antelopes was minced and inoculated into modified Hayflick broth, which has been used extensively to isolate *Mycoplasma* spp. from animals. Cultures were incubated at 37°C in a humidified atmosphere of 5% CO2 (5). The medium was examined daily by comparing inoculated broth with an un inoculated control broth. Moderate turbidity, a color change from pink to yellow, and an appreciable swirl of the culture when rotated were used as indicators of

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1 These authors contributed equally to this article.