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 epitope 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

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).

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*KPC-2, *bla*VIM, *bla*IMP, *bla*SPM-1, *bla*GIM-1, and *bla*NDM-1, were detected by using PCR as described (2). Only the *bla*NDM-1 gene was detected (with primers 5′-GGCGGAATGGCT-CATCACGA-3′ and 5′-CGCAACAC-CAGCCTGACTTTT-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 and *K. pneumoniae* 13883 Rif at frequencies of 1 transconjugant per ≈1.0 × 10⁴ and 4.0 × 10³ 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*NDM-1 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*NDM-1 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

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**Table. Antimicrobial drug susceptibility of *Salmonella* strain Stanley and transconjugants containing New Delhi metallo-β-lactamase-1, China**

<table>
<thead>
<tr>
<th>Drug</th>
<th><em>Salmonella</em> strain Stanley</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</td>
<td>0.25</td>
<td>&gt;128</td>
<td>0.25</td>
<td>&gt;128</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>&gt;128</td>
<td>&lt;0.06</td>
<td>128</td>
<td>&lt;0.06</td>
<td>128</td>
</tr>
<tr>
<td>Latamoxef</td>
<td>64</td>
<td>0.25</td>
<td>32</td>
<td>&lt;0.06</td>
<td>32</td>
</tr>
<tr>
<td>Cefepime</td>
<td>16</td>
<td>&lt;0.06</td>
<td>16</td>
<td>&lt;0.06</td>
<td>16</td>
</tr>
<tr>
<td>Imipenem</td>
<td>8</td>
<td>&lt;0.06</td>
<td>8</td>
<td>&lt;0.06</td>
<td>16</td>
</tr>
<tr>
<td>Meropenem</td>
<td>4</td>
<td>&lt;0.06</td>
<td>4</td>
<td>&lt;0.06</td>
<td>8</td>
</tr>
<tr>
<td>Fosfomycin</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>2</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</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>2</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Trimethoprim/sulfamethoxazole</td>
<td>&gt;152/8</td>
<td>0.3/0.015</td>
<td>&gt;152/8</td>
<td>1.2/0.06</td>
<td>&gt;152/8</td>
</tr>
</tbody>
</table>
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. *capri pneumoniae* (Mccp), a member of the *M. mycoides* cluster (1). Mccp 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 antelope 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 antelope were found dead in the Naqu area of Tibet; the dead animals represented 16% of the 15,000 Tibetan antelope thought to live in the area. Necropsy was performed on 13 of the antelope at sites within the Shenzha, Shuanghu, and Nima localities of the Naqu area (online Technical Appendix Table 1, wwwnc.cdc.gov/EID/article/20/13-0067-Techapp1.pdf). Gross pathologic lesions were localized exclusively to the lung, where severe pleuropneumonia with partial hepatization 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 antelope 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. The medium was examined daily by comparing inoculated broth with an uninoculated 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...