ESBL-producing isolates of human and animal origins (*10*); no clonality was observed for the other 7 isolates by pulsed-field gel electrophoresis,

E. coli ST131 has emerged as a worldwide pathogen and causes mainly community-onset extraintestinal infections. Although the pandemic spread of *E. coli* ST131 was first identified in isolates producing CTX-M-15 ESBL, it is increasingly recognized that isolates belonging to this clone may also harbor other drug resistance determinants. Among acquired AmpC β-lactamases, CMY-2 has been most frequently reported in ST131 from human clinical isolates (*3*). Infections caused by CMY-producing *E. coli* are common but underrecognized because of the lack of standardized detection methods (*2*).

Given the rapid global spread of the ST131 clone and the possibility of its transmission from food animals to humans, coupled with an abundance of CMY-2–encoding plasmids in poultry environments, *E. coli* ST131 producing CMY-2 β-lactamase may have potential to spread to humans. Our results also show that *E. coli* producing CMY-2 continues to be found commonly among retail chicken products in our study area.

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Ilheus Virus Infection in Human, Bolivia

To the Editor: Ilheus virus (ILHV) was first isolated from mosquitoes of the genera *Ochlerotatus* and *Psorophora* near Ilheus, Bahia, Brazil, in 1944 (*1*). After its discovery, the virus was also isolated from other mosquito species, including the genera *Culex*, *Sabethes*, *Haemagogus*, and *Trichoprosopon*, and from a variety of birds in different countries in Latin America (*2*). Only a few reports describe isolation of this virus from humans in Central and South America with symptoms ranging from subclinical to severe febrile disease (*2–6*). In mild cases, patients often reported gastrointestinal or respiratory symptoms lasting ≈1 week. In severe cases, either the central nervous or cardiac system can be affected. However, long-term sequelae or deaths have not been described. No epidemics attributed to ILHV have been reported.

In November 2005, a 15-year-old boy (farmer) sought medical attention in a health clinic in Magdalena, Bolivia, after having fever for 5 days. The patient's symptoms included malaise, asthenia, conjunctival

injection, vesicular rash, facial edema, arthralgia, myalgias, bone pain, abdominal pain, headache, and earache. Signs of cardiac, neurologic, or renal damage were not detected. A blood specimen was obtained during the clinic visit, and a convalescent-phase sample was obtained 24 days after onset of symptoms. At that follow-up visit, the patient reported a full recovery from his symptoms. Both samples were sent to the Naval Medical Research Unit No. 6 in Lima, Peru, for processing as part of a clinic-based study to determine the etiology of febrile illnesses in Bolivia (7). The study was approved by the Naval Medical Research Unit No. 6 Institutional Review Board (Navy Medical Research Center Detachment 2000.0008) and conducted in collaboration with the Bolivia Ministry of Health.

Serologic analyses showed a 64-fold IgM seroconversion between the acute-phase (<100) and convalescent-phase samples (6,400) by using an IgM ELISA as described (8). Samples were also tested by ELISA for the following arboviruses: West Nile virus, dengue virus, Oropouche virus, Guaroa virus, Rocio virus, St. Louis encephalitis virus, yellow fever virus, Venezuelan equine encephalitis virus, and Mayaro virus. All test results were negative for these viruses. Virus isolation was attempted on the acute-phase serum sample by using Vero and C6/36 cells, but the culture did not yield any virus. Attempts to isolate virus by intracranial inoculation in suckling mice were also unsuccessful (University of Texas Medical Branch, Institutional Animal Care and Use Committee protocol 9505045).

Viral RNA was extracted from the acute-phase sample and reverse transcription PCR specific for a portion of the nonstructural protein 5 gene was performed by using a described method (9). A 189-bp PCR product was obtained, purified, and sequenced by using flavivirus primers FU1 and cFD2 (9) and further analyzed by using BLAST (www.ncbi.nlm.nih.gov/blast), resulting in ≈95% homology to ILHV. Phylogenetic analysis with neighbor-joining and parsimony methods grouped the nucleotide sequence of the ILHV virus from Bolivia with ILHV strains from Ecuador and Peru (Figure).

Magdalena is a tropical city in northern Bolivia that borders Brazil. The city is surrounded by rivers and chestnut fields, and agriculture and fishing are the main sources of employment. Despite having ecoepidemiologic conditions similar to those in other locations with a history of ILHV transmission, the virus had not been detected in the area. The patient had no travel history in the 30 days preceding his illness, indicating that the virus is probably endemic to the area.

Mild unspecific symptoms, a short viremic period, and lack of advanced confirmatory laboratory techniques in situ are some of the barriers impeding the diagnosis of ILHV in disease-endemic areas. High levels of antibody cross-reactivity among flaviviruses, which are also endemic to the area, might render diagnosis even more difficult. The presence of the main ILHV vector, *Psorophora* sp. mosquitoes, in the city suggests that much of the population that labors outdoors may be at risk for ILHV infection.

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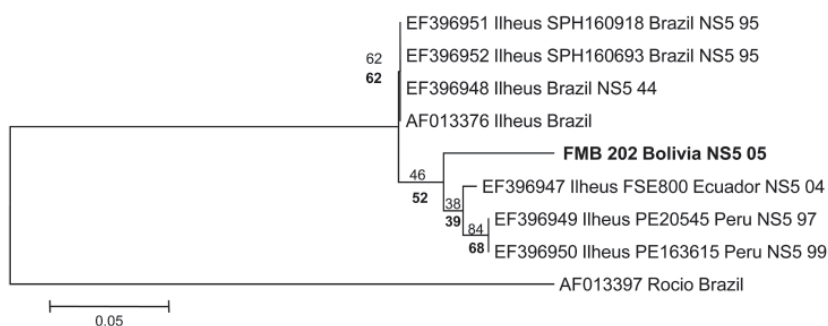

Figure. Phylogenetic analysis of the nonstructural protein 5 (NS5) gene region of 7 Ilheus virus isolates and a 189-bp nt sequence (FMB 202 Bolivia). Alignments were analyzed by using the neighbor-joining method with the Kimura 2-parameter algorithm in MEGA5 (www.megasoftware.net). Variation rate among sites was modeled with a gamma distribution (shape parameter = 1). Bootstrap confidence limits (from 1,000 replicates) are indicated at each node. Values in **boldface** below branches were obtained by parsimony analysis; **boldface** isolate name indicates virus from this study. Rocio virus (GenBank accession no. AF013397) was included as an outgroup on the basis of the phylogram of Kuno and Chang (10). Sequence generated in our study was deposited in GenBank under accession no. JN679229. Scale bar indicates nucleotide substitutions per site.

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Shared Human/Rabbit Ligands for Rabbit Hemorrhagic Disease Virus

To the Editor: Rabbit hemorrhagic disease virus (RHDV) is a calicivirus of the genus *Lagovirus* that causes epidemics of an acute disease and mortality rates of 50%–90% among rabbits. The disease, which was first described in 1984, is characterized by hemorrhagic lesions, mainly affecting the liver and lungs 24–72 h after infection (*1*).

Similar to human caliciviruses of the genus *Norovirus*, RHDV binds to histo-blood group antigens (HBGAs), and we recently showed that HBGAs serve as attachment factors (ligands) that facilitate RHDV infection (*2*). HBGAs are polymorphic carbohydrate structures representing terminally exposed portions of larger glycans linked to proteins or glycolipids. In many vertebrate species, they are mainly expressed on epithelial surfaces. Because phylogenetic conservation of receptors is a major risk factor for cross-species transmission (*3*), we analyzed the ability of RHDV strains to recognize human HBGAs expressed on epithelia.

We obtained 38 saliva samples from healthy persons with ABO, Secretor, and Lewis phenotypes, and we selected confirmed *FUT2* (secretor) and *FUT3* (Lewis) genotypes (*4*) to include ABO, secretor, and Lewis phenotypic diversity. Binding capacity of 6 RHDV strains representative of virus diversity (*2*) was tested against human saliva samples by using a method similar to that reported for human norovirus (*5*).

In brief, saliva samples diluted 1:1,000 or B type 2 bovine serum albumin–conjugated tetrasaccharide (positive control) were coated on ELISA plates. After blocking with milk diluted in phosphate-buffered saline, RHDV strains isolated from whole liver extracts of infected animals were incubated on coated plates at dilutions corresponding to 1×10^9 genome copies (0.2 µg/mL capsid protein equivalent) as determined by Nyström et al. (*2*). Monoclonal antibody 2G3, biotinylated anti-mouse IgG, and peroxidase-conjugated avidin were used for RHDV detection; 3,3′,5,5′-tetramethylbenzidine was used as a substrate; and optical density values at 450 nm were measured (*2*).

Binding to the B type 2 epitope was observed for all 6 strains (online Technical Appendix Figure, panel A, wwwnc.cdc.gov/EID/pdfs/11-1402-Techapp.pdf). Human saliva samples were recognized by 5 of 6 RHDV strains. Only G6, an RHDV antigenic variant also known as RHDVa (*6*), did not show binding to saliva. Strains G1 and G2 showed preferential binding to saliva from B secretors over that from O secretors, and A secretors were poorly recognized. Better recognition of A secretor saliva was obtained with the G3 strain. The G4 and G5 strains showed a clear preference for A secretors over B and O secretors, which indicated a shift in specificity toward recognition of the A antigen from the H and B antigens, as reported (*2*). None of the strains recognized nonsecretor saliva, which showed that binding to human saliva required A, B, or H motifs. This finding was confirmed by drastically decreased binding after removal of A, B, and H epitopes from secretor saliva by treatment with specific glycosidases. There was no relationship with the Lewis status.

To determine if human epithelial cells were recognized by RHDV, binding of the G3 strain to human tissue sections was assessed. Human trachea, lung, and gastroduodenal junction samples obtained from organ donors (before current French restrictions of December 1988) were used to prepare tissue microarrays. Tissues from 18 persons were used