(H5N1) strain (collected in December 2005; online Appendix Figure, panel A) is an HPAI and is closely related to the parent of the group of viruses isolated in the early 2006 Egypt outbreak, with an average identity of 99.4% with all other strains from Egypt and a bootstrap support value of 96% (online Appendix Figure, panel B). Despite the rapid spread of this clade (Qinghai-like strain) to many countries, since late 2005, strains analyzed in this study showed low-level genetic variation (<2%).

Brown et al. reported that species can vary greatly in their response to HPAI (*9*). At least in ducks, it appears that viral shedding is highest in birds with clinical signs of infection, and lowest, as seen in the common teal infected with the HPAI strain in this study, in birds with subclinical infections. These subclinical infections may be due to flock immunity from previous exposure to LPAI H5 virus or genetic factors. This suggestion is conceivable in light of the LPAI H5 virus detected in the other teal a few months earlier.

Such naturally resistant wild birds might serve as vectors for introduction of HPAI viruses into new locations. Data presented herein suggest that an HPAI virus may have been introduced into Egypt through a migratory bird. Whether poultry were infected before mid-February or the teal was infected with influenza A (H5N1) virus by a domesticated species is not unknown. The low degree of viral shedding indicates that detection of any influenza A (H5N1) virus in wild birds in a new region should be immediately followed up with efforts to characterize the virus to control the spread of new subtypes/strains of HPAI into new locations.

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Community-acquired Extended-Spectrum β-Lactamase Producers, United States

To the Editor: Extended-spectrum β-lactamase (ESBL)–producing organisms have become a common problem for patients in hospitals and other healthcare facilities (*1*). Community-onset ESBL infections have recently been described in Spain, the United Kingdom, Israel, and Canada (*2*,*3*). Typically, the infections are urinary tract infections (UTIs) with CTX-M–producing *Escherichia coli*. These organisms may be resistant to most or all antimicrobial agents commonly used to treat UTIs, such as ciprofloxacin, trimethoprim-sulfamethoxazole, gentamicin, and ceftriaxone.

Although CTX-M–producing *E. coli* have previously been found in the United States (*4*), clinical descriptions of community-acquired ESBL-producing *E. coli* infections have not been reported in this country. We describe 2 healthy young women in Pennsylvania in whom UTI with CTX-M-15–producing *E. coli* developed.

A 25-year-old woman was seen in October 2006 at the emergency department of a hospital in Pittsburgh reporting frequent urination, chills, and bilateral back pain. She had no relevant past medical history except for previous UTIs. Results of a physical examination were unremarkable.

Urinalysis showed >20 leukocytes per high-powered microscopic field. Urine culture grew >100,000 colonies/mL *E. coli*, resistant to trimethoprim/sulfamethoxazole and ciprofloxacin. The organism was positive by phenotypic confirmatory tests for ESBL production. Molecular characterization showed a gene encoding the CTX-M-15 β-lactamase. The patient was treated empirically with oral ciprofloxacin, 500 mg every 12 hours for 10 days and was lost to further follow-up. She has not had urine or blood cultures collected through our healthcare system in the 3 months since the time of her UTI.

A 24-year-old woman visited a student health service in Pittsburgh in September 2006 with urinary frequency, nausea, and back pain. There was mild costovertebral angle tenderness, and a clinical diagnosis of early pyelonephritis was made. The patient had no relevant past medical history except for previous UTIs (3 in the last 12 months). She was treated empirically with oral ciprofloxacin, 500 mg every 12 hours for 7 days. Urine culture grew >100,000 colonies/mL *Streptococcus agalactiae* and 25,000 colonies/mL *Klebsiella pneumoniae*. The *K. pneumoniae* was not an ESBL producer and was resistant to ampicillin and susceptible to ciprofloxacin, trimethoprim/sulfamethoxazole, ceftriaxone, and tobramycin.

Ten days after the initial visit, the patient returned for further assessment, and a follow-up urine sample was collected. There was no pyuria, but urine culture grew 15,000 colonies/mL *E. coli*. The organism was an ESBL producer that was resistant to ciprofloxacin and tobramycin and susceptible to trimethoprim/sulfamethoxazole. Given the lack of pyuria and the low intensity of symptoms, the patient was not treated with antimicrobial agents. She has not returned for follow-up, and no urine or blood cultures have been collected through our healthcare system in the 3 months since the time of her *E. coli* UTI. Molecular characterization of the gene encoding the ESBL indicated that it encoded CTX-M-15.

To our knowledge, these 2 cases represent the first cases of community-acquired ESBL-producing *E. coli* known to have occurred in the United States. In 2003, Moland et al. detected CTX-M–like ESBLs in the United States (*4*). The 9 *E. coli* isolates they described were from patients in 5 states—Virginia, Idaho, Ohio, Washington, and Texas—which suggests that CTX-M producers are geographically widespread in this country. Although some isolates were from a urinary source, these isolates were from a hospital surveillance study and the authors were unable to determine if these organisms were from community-acquired infections.

These 2 patients did not appear to have substantial clinical effects from their infections. However, the potential importance of community-acquired ESBL-producing *E. coli* is that UTI may be associated with bloodstream infection. Empiric antimicrobial therapy of bloodstream infection presumed to be of urinary tract origin typically comprises use of fluoroquinolones, aminoglycosides, or ceftriaxone. ESBL-producing *E. coli* may be resistant to all of these antimicrobial agents. In the United Kingdom, 25 of the first 108 patients with documented community-onset ESBL-producing *E. coli* infections died (*5*). Frequent occurrence of ESBL-producing *E. coli* in the United States would be an important public health problem and may necessitate changes in empiric antimicrobial therapy.

In Europe, many community-onset infections with ESBL producers actually appear to be healthcare associated, rather than truly community acquired. Patients with these infections may have been hospitalized in the recent past or have had relevant underlying diseases (*2*,*6*). It is also possible that the 2 infections we observed were not actually community-acquired and that these 2 women had unrecognized exposure to hospitals or healthcare facilities. However, the CTX-M producers we have previously isolated from hospitals in Pittsburgh produced CTX-M-9, not CTX-M-15 (data not shown). Our healthcare system provides coverage for most people in Pittsburgh and surrounding regions, and we found no records of previous hospitalization or chronic healthcare contact for either of the 2 persons. As far as we can ascertain, the 2 cases we describe appear to be truly community acquired, as determined by previous definitions differentiating healthcare-associated from community-acquired infections (*7*).

If these infections were truly community-acquired, how and why did they arise? The CTX-M-15 ESBL has been found in many countries. We do not know the travel histories of these 2 patients. Thus, the organisms may have been acquired overseas. We assessed the genetic relatedness of these 2 strains by pulsed-field gel electrophoresis but found no evidence of clonality (data not shown). Another possibility is that food was the source of infections. CTX-M-15–producing *E. coli* have been detected in food-producing animals (*8*), and we have recently found CTX-M-15–producing *E. coli* in chicken sold at a Pittsburgh area supermarket. We are currently conducting ongoing surveillance for community-acquired ESBL producers in our region in *E. coli* isolates from both humans and from foodstuffs to determine the prevalence of CTX-M producers in the United States.

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Japanese Encephalitis Outbreak, Yuncheng, China, 2006

To the Editor: Japanese encephalitis (JE) epidemics have occurred only in Asia. More than 50,000 cases of JE with ≈10,000 deaths have been reported since 1998 (*1,2*). The People's Republic of China reported 5,104 cases and 214 deaths in 2005. Most of these deaths occurred in infants (*3,4*).

During July and August 2006, an outbreak of viral encephalitis occurred in Yuncheng, Shanxi Province, People's Republic of China. A total of 66 cases (1.32/100,000 population) were reported, including 19 deaths (case-fatality rate 28.8%). The cases had a widespread distribution over 9 counties and involved 37 towns and 61 administrative villages. The ratio of male-to-female patients was 1:0.89. A distinct clinical feature of this outbreak was the age distribution. More than 86% of the patients were >30 years of age, with only 10% of patients <7 years of age; ≈95% of the deaths occurred in patients >50 years of age (*5*).

We report serologic and virologic findings for the 2006 outbreak of viral encephalitis. Forty-six clinical specimens collected from 34 patients who had a diagnosis of viral encephalitis, including 33 serum samples and 13 cerebrospinal fluid (CSF) samples, were studied. All serum samples were screened for immunoglobulin M (IgM) to West Nile virus (WNV) by using the WNV IgM-capture ELISA kit (PanBio, Brisbane, Queensland, Australia) and for IgM to dengue virus or Japanese encephalitis virus (JEV) by using the JE-Dengue IgM Combination ELISA kit (PanBio). Results for JEV were confirmed by using the JE Virus IgM-Capture ELISA kit (Shanghai B & C Enterprise Development Co. Ltd, Shanghai, People's Republic of China).

WNV-specific or dengue virus–specific IgM was not detected in any samples. JEV-specific IgM was detected in 27 (80%) patients, which indicated recent JEV infections. The other 7 patients were negative for JEV by ELISA and reverse transcription–PCR (RT-PCR). Increases ≥4-fold in neutralizing antibodies were detected in acute- and convalescent-phase serum samples from 9 patients (10 serum pairs were collected during the outbreak).

Attempts were made to detect virus in CSF of patients and in 2,400 mosquitoes. Mosquitoes (mainly *Culex* spp.) were collected in cow sheds and hog pens around houses and processed into pools of 100. Total RNA was extracted from CSF or mosquito homogenate by using the QIAamp viral RNA extraction kit (QIAGEN, Valencia, CA, USA) according to the manufacturer's specifications. RT was performed by using Ready-To-Go-You Prime First Strand Beads (Amersham Pharmacia Biotech, Piscatawy, NJ, USA) and a seminested PCR to amplify 492-bp gene fragments of the premembrane (PrM) sequence of JEV by using the Takara LA Taq PCR kit (Takara Bio Inc., Shiga, Japan). The primers were derived from Ishikawa strain genome sequences (GenBank accession no. AB051292). Primers PrMF: 5′-CGT TCT TCA AGT TTA CAG CAT TAG C-3′ (251–275), PrMR1: 5′-CGY TTG GAA TGY CTR GTC CG-3′ (724–743), and PrMR2: 5′-CCY RTG TTY CTG CCA AGC ATC CAM CC-3′ (901–925) were used.

JEV PrM gene was amplified from CSF of 6 (46%) of 13 patients and 10 of 24 pools of mosquitoes by using the same seminested RT-PCR. To identify JEV genotype(s) involved in this outbreak, PCR products were sequenced. Eleven sequences (GenBank accession nos. EF434264–EF434274) were obtained from 6 patients and 5 pools of mosquitoes. The 11 sequences were compared phylogenetically with17 known JEV strains of the 4 recognized