7. Chow BD, Ou Z, Esper FP. Newly recognized bocaviruses (HBoV, HBoV2) in children and adults with gastrointestinal illness in the United States. J Clin Virol. 2010;47:143–7. doi:10.1016/j.jcv.2009.11.030
8. Han TH, Kim CH, Park SH, Kim EJ, Chung JY, Hwang ES. Detection of human bocavirus-2 in children with acute gastroenteritis in South Korea. Arch Virol. 2009;154:1923–7. doi:10.1007/s00705-009-0533-3
9. Ren L, Gonzalez R, Wang Z, Xiang Z, Wang Y, Zhou H, et al. Prevalence of human respiratory viruses in adults with acute respiratory tract infections in Beijing, 2005–2007. Clin Microbiol Infect. 2009;15:1146–53. doi:10.1111/j.1469-0691.2009.02746.x
10. Tamura K, Dudley J, Nei M, Kumar S. MEGA4: Molecular Evolutionary Genetics Analysis (MEGA) software version 4.0. Mol Biol Evol. 2007;24:1596–9. doi:10.1093/molbev/msm092

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Highly Virulent *Escherichia coli* O26, Scotland

To the Editor: Hemolytic uremic syndrome (HUS) is a rare disorder characterized by microangiopathic hemolytic anemia, microthrombi, and multiorgan injury. HUS is one of the commonest causes of acute renal failure in children worldwide and is most frequently precipitated by infection with verotoxin-producing *Escherichia coli* (VTEC) such as *E. coli* O157 (*1*). However, non-O157 VTEC serotypes have been increasingly found in the development of HUS (*2*–*4*).

Although previous surveillance of childhood HUS in Scotland identified *E. coli* O157 in >90% of cases, non-O157 serotypes have also been associated with HUS (*5*). In 2010, several particularly severe cases of HUS were reported to Health Protection Scotland by a consultant pediatric nephrologist. Subsequent tests identified the pathogen in these cases as *E. coli* O26. However, in a recent study of pediatric HUS cases in Europe, children infected with *E. coli* O26 did not exhibit different clinical signs and symptoms from patients infected with other VTEC serotypes (*6*). To establish whether the host pathophysiologic responses to *E. coli* O157 and *E. coli* O26 strains differed, we analyzed a cohort of children with HUS who were infected with these VTEC serotypes.

In Scotland, most patients with pediatric thrombotic microangiopathy are referred to a specialist pediatric hospital, which immediately reports cases of HUS to Health Protection Scotland as part of national surveillance. To test the hypothesis that *E. coli* O26 was more virulent than *E. coli* O157, we performed an age-matched, nested case–case study of HUS patients and compared host clinical markers, treatment, and outcomes from pediatric cases in 2010. Data collection has been described elsewhere (*5*). The statistical significances of associations between categorical variables were investigated by using χ^2, Fisher exact, Mann-Whitney, or *t* tests. All analyses were performed by using SPSS version 11 (SPSS Inc., Chicago, IL, USA) with a significance level of 5%.

Although initial signs and symptoms were similar for both sets of cases, i.e., bloody diarrhea and abdominal pain, statistical analysis showed that children with O26-HUS were more likely to have neurologic complications and diabetes mellitus and require admission to the intensive care unit than O157-HUS patients (p = 0.02 for neurologic complications and diabetes and p = 0.04 for admission to an intensive treatment unit; Table).

All patients with HUS were oligoanuric, and the 2 groups did not differ with respect to this parameter. However, O26-HUS patients had significantly longer periods of anuria than O157-HUS patients (p = 0.04; Table) and were more likely to require treatment with hemofiltration than with peritoneal or hemodialysis (p = 0.001; Table). One patient with O26-HUS also experienced cardiomyopathy resulting in reduced left ventricular function.

Our study illustrates the potential for increased severity of *E. coli* O26 infection in children. In Scotland, HUS is more commonly associated with *E. coli* O157 infection, and the outcome for children infected with this pathogen is much better than that reported in other studies (*7*,*8*). In this study, the clinical severity and outcomes for the children with O26-HUS were worse than for children requiring treatment for O157-HUS. We investigated the prehospital management of *E. coli* O157 and O26 patients in this cohort and found no difference in pharmacologic intervention or duration of delay in admission to hospital.

In our cohort, vtx_1 and vtx_2 genes were detected in isolates from 2 of 3 patients. A diagnosis was made in the third patient by detection of *E. coli* O26 lipopolysaccharide–specific immunoglobulin M in serum; it was therefore not possible to confirm the presence of *vtx* genes. However, it is not unusual for VTEC to be undetectable in stool samples from patients with HUS, most likely because of intrahost bacteriophage lysis. Therefore, serodiagnosis of VTEC is considered a necessary adjunct to bacteriological confirmation of infection (*9*). A recent study suggests *E. coli* O26 exists as a highly dynamic group of organisms that can undergo verotoxin gene loss and be transferred during infection in humans, resulting in new pathogenic clones (*10*). Therefore, vtx_2 gene acquisition by *E. coli* O26 may have contributed to increased virulence.

Table. Characteristics of infection in children with *Escherichia coli* O157 versus *E. coli* O26, Scotland, 2010*

Variable	O157 HUS, n = 12	O26 HUS, n = 3	p value
Age, y	4.5 ± 1.0	3.7 ± 1.3	
Clinical signs and symptoms			
Diarrhea	12	3	
Bloody diarrhea	8 (67)	2 (67)	NS
Abdominal pain	8 (67)	3 (75)	NS
Fever	2 (17)	1 (33)	NS
Neurologic involvement	3 (25)	3 (100)	0.02
Diabetes	1 (12)	2 (67)	0.02
Cardiomyopathy	0	1 (33)	0.04
Anuria, d	7.7 ± 2.4	15.7 ± 1.3	0.04
Clinical parameters			
Leukocyte count × 10^9/L	42.1 ± 14.4	25.9 ± 4.8	NS
C-reactive protein, mg/L	93.5 ± 28.4	151 ± 74	NS
Serum albumin, g/L	22.5 ± 1.3	22.3 ± 2.4	NS
Lactate dehydrogenase, IU/L	2,521 ± 362	1,991 ± 642	NS
Admission to ITU	3 (25)	3 (100)	0.04
Duration of hospitalization, d	13.2 ± 2.2	25 ± 3.8	0.03
Treatment			
Peritoneal dialysis, d	7.4 ± 1.9	10	NS
Hemodialysis, d	8.5 ± 1.5	8.3 ± 3.0	NS
Hemofiltration	0	3 (100)	0.001
Laparotomy	0	1 (33)	0.04
Postdischarge hypertension	0	2 (67)	0.01

*Values are mean ± SE or no. (%). Significance relates to difference between groups. HUS, hemolytic uremic syndrome; NS, not significant, ITU, intensive treatment unit.

Our study was limited by the small number of patients with pediatric O26-HUS. However, given the severity of the complications experienced by the children in this cohort, we believe it is necessary to communicate these findings promptly to the international community.

We suggest that infection with *E. coli* O26 in children can result in more severe and complicated forms of HUS than those caused by *E. coli* O157. In contrast to the findings of Gerber et al., we found that there was a significant difference in neurologic complications between the 2 groups (*2*). Epidemiologic investigations found that 2 of the 3 children lived on farms and may have acquired infection while playing near their homes (the other was acquired through foreign travel). Risk communication of VTEC infection to parents of young children who live in farming communities remains problematic, perhaps because of the perception that immunity has been acquired. Although this suggestion may be true for adults, children are likely to be immunologically naive. Salient public health messages on simple precautionary behavior need to be regularly reinforced because prevention of VTEC infection prevents HUS.

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References

1. Tarr PI, Gordon CA, Chandler WL. Shiga toxin producing *Escherichia coli* and the haemolytic uraemic syndrome. Lancet. 2005;365:1073–86.
2. Gerber A, Karch H, Allerberger F, Verweyen HM, Zimmerhackl LB. Clinical course and the role of Shiga toxin-producing *Escherichia coli* infection in the hemolytic-uremic syndrome in pediatric patients, 1997–2000, in Germany and Austria: a prospective study. J Infect Dis. 2002;186:493–500. doi:10.1086/341940
3. Aldick T, Bielaszewska M, Zhang W, Brockmeyer J, Schmidt H, Friedrich AW, et al. Hemolysin from Shiga toxin–negative *Escherichia coli* O26 strains injures microvascular endothelium. Microbes Infect. 2007;9:282–90. doi:10.1016/j.micinf.2006.12.001
4. Johnson KE, Thorpe CM, Sears CL. The emerging clinical importance of non-O157 Shiga toxin-producing *Escherichia coli.* Clin Infect Dis. 2006;43:1587–95. doi:10.1086/509573
5. Pollock KGJ, Young D, Beattie TJ, Todd WTA. Clinical surveillance of thrombotic microangiopathies in Scotland, 2003–2005. Epidemiol Infect. 2008;136:115–21. doi:10.1017/S0950268807008217
6. Zimmerhackl LB, Rosales A, Hofer J, Riedl M, Jungraithmayr T, Mellman A, et al. Enterohemorrhagic *Escherichia coli* O26:H11–associated hemolytic uremic syndrome: bacteriology and clinical presentation. Semin Thromb Hemost. 2010;36:586–93. doi:10.1055/s-0030-1262880
7. Garg AX, Suri RS, Barrowman N, Rehman F, Matsell D, Rosas-Arellano MP, et al. Long-term renal prognosis of diarrhea-associated hemolytic uremic syndrome: a systematic review, meta-analysis, and meta-regression. JAMA. 2003;290:1360–70. doi:10.1001/jama.290.10.1360
8. Weekly Report HPS. Clinical surveillance of haemolytic uraemic syndrome 2003–2009: renal prognosis at three-year follow up [cited 2011 Feb 1]. http://www.documents.hps.scot.nhs.uk/ewr/pdf2010/1010

9. Chart H, Cheasty T. Human infections with verocytotoxin-producing *Escherichia coli* O157—10 years of serodiagnosis. J Med Microbiol. 2008;57:1389–93. doi:10.1099/jmm.0.2008/003632-0
10. Bielaszewska M, Prager R, Köck R, Mellmann A, Zhang W, Tschäpe H, et al. Shiga toxin gene loss and transfer *in vitro* and *in vivo* during enterohemorrhagic *Escherichia coli* O26 infection in humans. Appl Environ Microbiol. 2007;73:3144–50. doi:10.1128/AEM.02937-06

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Perinatal Transmission of Yellow Fever, Brazil, 2009

To the Editor: Although urban cases of yellow fever have not been reported in Brazil since 1942, sylvatic yellow fever is still endemic to the northern and middle-western states. In the past decade, the endemic area has spread southward and eastward, approaching most populated states (*1*). In 2009, there was an outbreak of sylvatic yellow fever in São Paulo State that caused 28 cases and 11 deaths. In the affected area, there had been no reports of yellow fever since the 1930s (*2*). In the outbreak setting, a case of perinatal yellow fever transmission was diagnosed.

The mother was a 30-year-old woman exposed to yellow fever in late pregnancy in a sylvatic area near Piraju (23°11′44″S, 49°22′54″W), a city 100 km from Botucatu. The patient had not received yellow fever vaccine and had not traveled to yellow fever–endemic regions in the previous months. The exposure to yellow fever occurred during regular walks in a sylvatic area, a habit that continued until late pregnancy. She had fever, headache, and jaundice on March 14, 2009. Three days later, on March 17, she delivered a female infant through vaginal partum in a hospital in her hometown.

The mother´s symptoms were mild. She was admitted to Botucatu Medical School Hospital 7 days after delivery; she had fever, jaundice, and conjunctival suffusion. Liver enzymes were elevated (aspartate aminotransferase [AST] 246 U/L, alanine transaminase [ALT] 324 U/L, γ-glutamyl transpeptidase 221 U/L, and alkaline phosphatase 338 U/L). She was mildly anemic (hemoglobin level 10.2 g/dL), but leukocyte and platelet counts were within reference ranges. There were no other laboratory abnormalities. She was discharged after 7 days with complete recovery.

The infant girl was born asymptomatic on March 17, with a birthweight of 3,800 g and Apgar scores of 9–10. She was discharged from the hospital after 2 days of exclusive breast-feeding. On the third day of life, she had fever and cyanosis and was readmitted to the local hospital with suspected pneumonia. She received antimicrobial drugs but showed no improvement. On the 8th day of life, she had hematemesis, melena, bleeding at venipuncture sites, hypoglycemia, and oliguria.

The newborn was transferred to the Neonatal Intensive Care Unit at Botucatu Medical School Hospital. At admission, she had hypotension, tachycardia, cutaneous paleness, jaundice, hepatomegaly, and melena. The initial diagnostic hypothesis was congenital or hospital-acquired sepsis, but the mother´s diagnosis prompted doctors to investigate possible yellow fever. The infant was intubated for ventilatory support and received volume expansion, vasoactive amines, antimicrobial drugs, blood components (erythrocytes, platelets, fresh frozen plasma, cryoprecipitate), and drugs to control bleeding. Liver enzymes values were initially high (AST 4,072 U/L, ALT 1,420 U/L) but fell abruptly within 3 days (AST 150 U/L, ALT 114 U/L).

Despite the therapy, the newborn experienced liver and renal failure, disseminated intravascular coagulation, seizures, and finally coma. She died on the 12th day of life (4th day of hospitalization in the neonatal intensive care unit). Autopsy specimens showed massive liver necrosis, pulmonary hemorrhage, and acute tubular necrosis (Figure).

The mother had serologic tests (immunoglobulin M antibody capture ELISA) done on the 11th day of disease. Test results were positive for yellow fever and negative for dengue fever. The newborn had similar results from serum samples collected 5 days after onset of symptoms with confirmation by reverse transcription PCR (RT-PCR). RT-PCR was performed as described by Deubel et al. (*3*). Nucleotide sequencing showed a wild yellow fever virus belonging type I of South American genotype 1E, according to the classification proposed by Vasconcelos et al. (*4*). RT-PCR performed with samples of the mother´s serum did not amplify yellow fever virus sequences. However, the serum was collected on the 11th day post symptoms when the sensitivity of the test is low.

The vertical transmission of arboviruses has been documented. Pouliot et al. reviewed direct and indirect evidence for vertical transmission of dengue virus (*5*). Vertical transmission has also been reported for West Nile encephalitis and western equine encephalitis (*6*). This is not the case for yellow fever. Reports of yellow fever during pregnancy are scarce (*7*), and we found none that describe vertical transmission to newborns. However, vaccine virus was isolated from asymptomatic newborns from pregnant women who