8. Currie BJ, Haslem A, Pearson T, Hornstra H, Leadem B, Mayo M, et al. Identification of melioidosis outbreak by multilocus variable number tandem repeat analysis. Emerg Infect Dis. 2009;15:169–74. http://dx.doi.org/10.3201/eid1502.081036
9. Tuanyok A, Auerbach RK, Brettin TS, Bruce DC, Munk AC, Detter JC, et al. A horizontal gene transfer event defines two distinct groups within *Burkholderia pseudomallei* that have dissimilar geographic distributions. J Bacteriol. 2007;189:9044–9. http://dx.doi.org/10.1128/JB.01264-07
10. Baker A, Pearson T, Price EP, Dale J, Keim P, Hornstra H, et al. Molecular phylogeny of *Burkholderia pseudomallei* from a remote region of Papua New Guinea. PLoS ONE. 2011;6:e18343. http://dx.doi.org/10.1371/journal.pone.0018343

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Hemolytic Uremic Syndrome Associated with *Escherichia coli* O8:H19 and Shiga Toxin 2f Gene

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DOI: http://dx.doi.org/10.3201/eid21001.140515

To the Editor: Gastroenteritis caused by Shiga toxin–producing *Escherichia coli* (STEC), associated with hemorrhagic colitis and hemolytic uremic syndrome (HUS), has been identified as a major health problem (*1*). Shiga toxin is essential for the development of HUS (*2*). Shiga toxin can be distinguished into Shiga toxin 1 (Stx1) and Shiga toxin 2 (Stx2). The stx_{2f} STEC variant is a distinct group within STEC (regarding virulence genes) and is known to cause relatively mild disease, although reports of human illness are scarce (*3*).

During autumn 2013, a healthy 9-year-old boy in the Netherlands experienced fever, vomiting, and bloody diarrhea which persisted for days; he was admitted to the pediatric ward of a local hospital because of clinical signs of HUS with renal insufficiency: serum creatinine level 439 μmol/L (reference range 31–68 μmol/L); blood urea nitrogen concentration 34.1 mmol/L (reference range 3.3–5.6 mmol/L); thrombocytopenia (46 platelets/nL); reference range 150–450/nL), and low haptoglobin level. Hemoglobin levels decreased within 48 hours from 7.4 mmol/L to 5.5 mmol/L (reference range 6.9–8.4 mmol/L). His blood pressure was 127/82 (99th percentile for age and height). Renal insufficiency worsened over time, evidenced by maximum urea levels of 57.3 mmol/L and maximum creatinine levels of 744 μmol/L. Vomiting increased, and feeding became difficult. The boy was transferred to an academic nephrology center, where he received erythrocyte and thrombocyte infusions, then peritoneal dialysis. He received 1 prophylactic dose of cefazolin during insertion of the dialysis catheter. After 2 days, he entered a polyuric phase of renal failure; renal function normalized within a few weeks, however. To improve proteinuria, physicians prescribed a 3-month course of angiotensin-converting enzyme inhibitors after discharge.

A fecal sample tested positive for STEC by PCR in a local laboratory. Five isolates were sent to the National Institute for Public Health and the Environment (RIVM) as part of the national STEC surveillance. By using PCR, 1 of the 5 tested positive for the stx_{2f} gene and the attaching and effacing gene (*eae*), and negative for the genes stx_1, $stx_{2a\text{-}e}$, H7, O157, and enterohemorrhagic *E. coli* hemolysin (*hly*). Serotyping identified O8:H19. The other 4 isolates tested negative for all of the above-mentioned genes and were not serotyped.

The family had stayed in a hotel in Turkey and returned to the Netherlands 5 days before onset of illness. The only reported contact with animals was with a parrot in the hotel. On return to the Netherlands, the boy had eaten filet américain, a sandwich spread made of raw beef. The day before disease onset, he attended a party where barbecue was served by a catering company.

Since 2007, besides this reported case, 8 cases of STEC O8 were registered within the STEC surveillance system in the Netherlands: O8:H– (4 cases), O8:H19 (2 cases), O8:H8 (1 case), and O8:H9 (1 case). All 8 isolates were $stx_{2a\text{-}e}$-positive and stx_1-, stx_{2f}-, *eae*-, and *hly*-negative. Disease associated with these cases was relatively mild. During 2007–2010, a total of 13,545 human STEC infections were reported in Europe: 20 were registered as STEC O8; HUS did not develop in these case-patients (*4*). HUS developed in 2 patients infected with STEC O8 (O8:H2, O8:H19) in Germany during 1996–2000 (*5*); these isolates and all other isolates from HUS and non-HUS case-patients in this period tested negative for stx_{2f}. During 2008–2011, 87 stx_{2f} STEC infections were registered in the Netherlands (*3*). These infections were relatively mild; no HUS cases were registered. The virulence genes seen in the isolate of the described case, stx_{2f} and *eae*, but no *hly* or other toxin genes, were also seen in 97% of stx_{2f} STEC infections reported in the Netherlands (*3*). Besides being detected in humans, stx_{2f} STEC has only been detected in pigeons (*6*).

The cause of the severity of disease in this stx_{2f} STEC case and the source of the infection could not be determined. The parrot in the hotel in Turkey could have been the source if birds are a reservoir of stx_{2f} STEC. Conversely, the uncooked beef and barbecue cannot be ruled out, because O8:H19 has been found in cattle, pigs, and sheep (*7*). This case shows that STEC subgroups known to cause relatively mild disease can occasionally cause severe disease and that surveillance based upon a small group of serotypes underestimates the number of severe STEC infections and increases the chance of missing emerging serotypes.

References

1. Karmali MA, Gannon V, Sargeant JM. Verocytotoxin-producing *Escherichia coli* (VTEC). Vet Microbiol. 2010;140:360–70. http://dx.doi.org/10.1016/j.vetmic.2009.04.011
2. Prager R, Annemuller S, Tschape H. Diversity of virulence patterns among Shiga toxin–producing *Escherichia coli* from human clinical cases—need for more detailed diagnostics. Int J Med Microbiol. 2005;295:29–38. http://dx.doi.org/10.1016/j.ijmm.2004.12.009
3. Friesema I, van der Zwaluw K, Schuurman T, Kooistra-Smid M, Franz E, van Duynhoven Y, et al. Emergence of *Escherichia coli* encoding Shiga toxin 2f in human Shiga toxin-producing *E. coli* (STEC) infections in the Netherlands, January 2008 to December 2011. Euro Surveill. 2014; 19(17): pii=20787. http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20787 [cited 2014 Nov 18]
4. European Food Safety Authority Panel on Biological Hazards. Scientific opinion on VTEC-seropathotype and scientific criteria regarding pathogenicity assessment. EFSA Journal. 2013;11:3138. http://dx.doi.org/10.2903/j.efsa.2013.3138
5. Friedrich AW, Bielaszewska M, Zhang WL, Pulz M, Kuczius T, Ammon A, et al. *Escherichia coli* harboring Shiga toxin 2 gene variants: frequency and association with clinical symptoms. J Infect Dis. 2002;185:74–84. http://dx.doi.org/10.1086/338115
6. Schmidt H, Scheef J, Morabito S, Caprioli A, Wieler LH, Karch H. A new Shiga toxin 2 variant (Stx2f) from *Escherichia coli* isolated from pigeons. Appl Environ Microbiol. 2000;66:1205–8. http://dx.doi.org/10.1128/AEM.66.3.1205-1208.2000
7. Bettelheim KA. The non-O157 Shiga-toxigenic (verocytotoxigenic) *Escherichia coli*; under-rated pathogens. Crit Rev Microbiol. 2007;33:67–87. http://dx.doi.org/10.1080/10408410601172172

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Monitoring Water Sources for Environmental Reservoirs of Toxigenic *Vibrio cholerae* O1, Haiti

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DOI: http://dx.doi.org/10.3201/eid2101.140627

To the Editor: In the March 2014 issue of Emerging Infectious Diseases, Alam et al. reported a survey of water sources in Haiti conducted to isolate *Vibrio cholerae* (*1*). Each month from April 2012 through March 2013, they sampled 15 sites at 3 rivers and 1 estuary in West Department. From 179 water samples and 144 aquatic animals and plants, they obtained 7 *V. cholerae* O1 isolates, including 3 *ctx*-positive toxigenic strains.

Unfortunately, the results for all 7 *V. cholerae* O1 isolates were aggregated, and no details were provided about the exact time and location of collection of samples corresponding to the 3 *ctx*-positive strains. The authors posed the question of whether *V. cholerae* O1 has become established in environmental reservoirs in Haiti, subsequently warning that "as long as the causative microorganism is present in the environment, eradication of the disease will not be possible."

However, after challenging their results with more accurate epidemiologic data, we found that these 3 *ctx*-positive toxigenic strains could more likely have been present in the sampled rivers as a result of recent fecal contamination (Figure, http://wwwnc.cdc.gov/EID/article/21/1/14-0627-F1.htm). Indeed, many cholera cases were reported in the corresponding communal sections (i.e., the smallest Haitian administrative unit, average 25 km^2) when the samples containing the 7 *V. cholerae* O1 isolates were collected. In this context of an ongoing cholera epidemic associated with persisting rainfall (Figure), generalized open-air defecation inevitably leads to contamination of water sources. It is therefore impossible to determine whether *V. cholerae*–positive rivers constitute perennial reservoirs of the bacteria or whether they act only as transient vectors of the pathogens.

The recent dramatic decrease in cholera transmission may provide a good opportunity to address this issue (*2*). We thus encourage Alam et al. to continue the search for *ctx*-positive toxigenic *V. cholerae* O1 strains in surface waters, especially during cholera-free periods.

Acknowledgments

We are grateful to the Haitian Directorate of Epidemiology Laboratory and Research and Doctors without Borders from Switzerland for providing cholera case data from the Leogane cholera treatment unit. We are indebted to Sandra Moore for her fine editing of this manuscript.

This work was co-financed by Assistance Publique–Hôpitaux de Marseille and the Haiti Office of the United Nations Children's Fund.

References

1. Alam MT, Weppelmann TA, Weber CD, Johnson JA, Rashid MH, Birch CS, et al. Monitoring water sources for environmental reservoirs of toxigenic *Vibrio cholerae* O1, Haiti. Emerg Infect Dis. 2014;20:356–63. http://dx.doi.org/10.3201/eid2003.131293