

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.

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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 km²) 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.

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Reservoir Host Expansion of Hantavirus, China

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DOI: <http://dx.doi.org/10.3201/eid2101.140960>

To the Editor: Hemorrhagic fever with renal syndrome (HFRS) is caused by hantavirus. During 1995–2005, China reported 20,000–50,000 cases of HFRS annually, which represents 90% of HFRS cases worldwide (1–3). In China, HFRS is caused mainly by 2 serotypes of hantavirus: Hantaan virus (HTNV) and Seoul virus (SEOV) (4). Pathogenic hantavirus serotypes are considered to be strictly associated with their serotype-specific reservoir hosts. HTNV is associated with the striped field mouse (*Apodemus agrarius*), and SEOV is associated with the brown rat (*Rattus norvegicus*) and the black rat (*Rattus rattus*) (4,5). HTNV causes a severe form of HFRS, characterized by renal failure that in some cases is followed by pulmonary edema and disseminated intravascular coagulation; the estimated death rate is 5%–15%. SEOV causes a moderate form of HFRS (6).

Jiaonan County in Shandong Province is one of the high-incidence HFRS areas in China. To detect the hantavirus infection in small mammals, we trapped rodents and shrews during December 2012–November 2013 using snap-traps in Jiaonan County (longitude 119°30′–120°30′, latitude 35°35′–36°08′).

We captured 1,276 animals comprising 5 rodent species and 1 shrew species (Table) and analyzed serum antibody against hantavirus of each animal using an antigen sandwich

ELISA Kit (Shanghai Jiahe Biotechnology, Shanghai, China). The serum was considered to contain antibodies against hantavirus when the optical density (OD)_{450nm} of the sample was greater than the threshold. The threshold was calculated by using the equation: threshold = the average OD of the negative control + 0.15. ELISA results showed that 23.3% of animals were seropositive to hantavirus antigen (Table). The seropositive rate to hantavirus was 44.0% in Asian house shrews (*Suncus murinus*), 25.3% in house mice (*Mus musculus*), 15.4% in Chinese hamsters (*Cricetulus griseus*), 10.3% in brown rats, 10.1% in striped field mice (*Apodemus agrarius*), and 3.0% in greater long-tailed hamsters (*C. triton*). The seropositivity rate for rodents was higher during summer (May–August) and lower during spring (March and April) and winter (October and November) but not significantly different among the months.

To determine what types of hantavirus infected the animals, we amplified viral RNA of HTNV and SEOV from animal lung samples using reverse transcription PCR with serotype-specific primers (7); 2.1% of animals had viral RNA of HTNV, and 2.1% had viral RNA of SEOV (Table). HTNV RNA was detected in striped field mice (6.3%), house mice (1.4%), and brown rats (0.6%). The hantavirus-positive animals were captured in February, April, and November for striped field mice; November for brown rats; and April and November for house mice. SEOV was detected in brown rats (8.2%) and Asian house shrews (1.7%). These SEOV-positive animals were captured in January, March, May, June, and July for brown rats and March and November for Asian house shrews. The phylogenetic analysis of sequences amplified by reverse transcription PCR is presented in the online Technical Appendix Figure (<http://wwwnc.cdc.gov/EID/article/21/1/14-0960-Techapp1.pdf>). The nucleotide sequences of the PCR products have been deposited in GenBank (accession nos. KM357423–KM357452).

Hantavirus had been considered to be strictly associated with specific reservoir hosts and to have the same geographic distribution pattern as these reservoir hosts. All hantaviruses that caused human diseases had been associated with rodents, including members of *Murinae*, *Arvicolinae*, and *Sigmodontinae* spp. Insectivore hantaviruses were not known to cause human disease. The rodent hantavirus and the insectivorous hantaviruses were thought to have co-evolved with their specific rodent and insectivorous hosts over millions of years (8). One observed geographic clustering of hantavirus strains, and the association of hantaviruses with their reservoirs, might have been caused by an isolation-by-distance mechanism (9,10) and mixture of both host switching and co-divergence (10). Our study demonstrated that HTNV not only infects its traditional host, the striped mouse, but also infects house mice and rats; SEOV infects not only rats but also shrews, suggesting host expansion for both HTNV

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