effective programs to curtail the spread of TB within the country and the region; in this era of globalization, it is required for the successful control of TB worldwide.

**Bassirou Diarra,[1]
Sophia Siddiqui,[1]
Dramane Sogoba,
Brehima Traore,
Mamoudou Maiga,
Janice Washington,
Anatole Tounkara,
and Michael A. Polis**

Author affiliations: Project SEREFO-NIAID/University of Bamako Research Collaboration on HIV/TB, Bamako, Mali (B. Diarra, D. Sogoba, B. Traore, M. Maiga, A. Tounkara); National Institute of Allergy and Infectious Diseases, Bethesda, Maryland, USA (S. Siddiqui, M.A. Polis); and US Food and Drug Administration, Silver Spring, Maryland, USA (J. Washington)

[1]These authors contributed equally to this article.

References

1. Filliol I, Driscoll JR, van Soolingen D, Kreiswirth BN, Kremer K, Valétudie G, et al. Global distribution of *Mycobacterium tuberculosis* spoligotypes. Emerg Infect Dis. 2002;8:1347–9.
2. European Concerted Action on New Generation Genetic Markers and Techniques for the Epidemiology and Control of Tuberculosis. Beijing/W genotype *Mycobacterium tuberculosis* and drug resistance. Emerg Infect Dis. 2006;12:736–43.
3. van Soolingen D, Qian L, de Haas PE, Douglas JT, Traore H, Portaels F, et al. Predominance of a single genotype of *Mycobacterium tuberculosis* in countries of east Asia. J Clin Microbiol. 1995;33:3234–8.
4. Glynn JR, Whiteley J, Bifani PJ, Kremer K, van Soolingen D. Worldwide occurrence of Beijing/W strains of *Mycobacterium tuberculosis*: a systematic review. Emerg Infect Dis. 2002;8:843–9.
5. Bifani PJ, Mathema B, Kurepina NE, Kreiswirth BN. Global dissemination of the *Mycobacterium tuberculosis* W-Beijing family strains [review]. Trends Microbiol. 2002;10:45–52. DOI: 10.1016/S0966-842X(01)02277-6
6. Kremer K, Glynn JR, Lillebaek T, Niemann S, Kurepina NE, Kreiswirth BN, et al. Definition of the Beijing/W lineage of *Mycobacterium tuberculosis* on the basis of genetic markers. J Clin Microbiol. 2004;42:4040–9. DOI: 10.1128/JCM.42.9.4040-4049.2004
7. Van Embden JD, Cave MD, Crawford JT, Dale JW, Eisenach KD, Gicquel B, et al. Strain identification of *Mycobacterium tuberculosis* by DNA fingerprinting: recommendations for a standardized methodology. J Clin Microbiol. 1993;31:406–9.
8. van der Spuy GD, Kremer K, Ndabambi SL, Beyers N, Dunbar R, Marais BJ, et al. Changing *Mycobacterium tuberculosis* population highlights clade-specific pathogenic characteristics. Tuberculosis (Edinb). 2009;89:120–5. Epub 2008 Dec 2. DOI: 10.1016/j.tube.2008.09.003
9. Werngren J, Hoffner SE. Drug-susceptible *Mycobacterium tuberculosis* Beijing genotype does not develop mutation-conferred resistance to rifampin at an elevated rate. J Clin Microbiol. 2003;41:1520–4. DOI: 10.1128/JCM.41.4.1520-1524.2003
10. Cowley D, Govender D, February B, Wolfe M, Steyn L, Evans J, et al. Recent and rapid emergence of W-Beijing strains of *Mycobacterium tuberculosis* in Cape Town, South Africa. Clin Infect Dis. 2008;47:1252–9. DOI: 10.1086/592575

Address for correspondence: Sophia Siddiqui, Collaborative Clinical Research Branch, National Institute of Allergy and Infectious Diseases, National Institutes of Health, 6700A Rockledge Dr, Rm 1105, Bethesda, MD 20892, USA; email: ssiddiqui@niaid.nih.gov

Hemorrhagic Fever with Renal Syndrome, Vietnam

To the Editor: Hantaviruses are primarily rodent borne and can cause hemorrhagic fever with renal syndrome (HFRS) in persons who inhale aerosolized excreta from infected rodents. The clinical characteristics of HFRS are fever, hemorrhage, and varying degrees of renal and hepatic dysfunction. Although HFRS is endemic primarily to Eurasian regions, there is serologic evidence of hantavirus infections in rodents and humans worldwide (*1*). Little is known about the occurrence of hantavirus infection in rodents or humans in Vietnam. One study found 5.4% prevalence of antibodies against Hantaan 76–118 and Puumala strains among residents of the Hanoi Metropolitan (*2*), whereas another study in southern Vietnam did not find evidence of hantavirus infection in humans (*3*). We describe autochthonous HFRS from Vietnam, possible reservoir hosts, and the follow-up investigation, which implies the presence of a strain of Seoul virus (SEOV).

The case-patient was a previously healthy 25-year-old nurse working in a referral hospital and residing in a semi-urban district of Ho Chi Minh City. On September 23, 2008, she was admitted to the referral hospital with a history of high fever, chills, myalgia, nausea, vomiting, hematuria, and abdominal and lower back pain for 3 days. Physical examination showed a body

temperature of >39°C, petechiae, mild dehydration and hypotension, with otherwise unremarkable vital signs. Hematologic tests showed 13,300 leukocytes/mm³, 167,000 thrombocytes/mm³, and hematocrit of 31%. Urinalysis showed grave hematuria (3+), proteinuria (2+), and leukouria (2+).

Three days after admission, acute renal failure with relative oliguria (0.85 L/24 h) developed, as well as uremia (26.4 mg/dL), creatinemia (0.98 mg/dL), and abnormal liver function (aspartate aminotransferase 49 U/L and alanine transferase 60 U/L). The following day the patient had dyspnea and became agitated. Ultrasound examination showed pleural effusion, parietal pericardial effusion, peritoneal ascites, hepatomegaly, and renal thickness. Six days after admission, diuretic problems developed in the patient (3.7 L/24 h), her dyspnea resolved, and she became afebrile. Ten days after admission, the patient's hematuria resolved, and renal and liver functions gradually recovered; she was discharged after 29 days of hospitalization.

Immunoglobulin (Ig) M and IgG against Hantaan recombinant nucleocapsid protein antigen were detected in the case-patient's acute-phase and convalescent-phase serum samples, respectively, by ELISA (4,5). The presence of antihantavirus IgG was confirmed by immunofluorescent antibody (IFA) assay using whole hantavirus antigen and Western blot using hantavirus CL-1 strain (6,7). In further analysis, neutralization antibodies against SEOV strain SR-11 were detected by focus reduction neutralization test (8). The viral RNA, however, was not detectable in the acute-phase blood sample by reverse transcription–PCR (RT-PCR) (9). Other serologic tests were performed for dengue fever, typhoid fever, hepatitis B, and malaria; results of culture of blood and urine were negative for bacteria.

Following confirmation of the diagnosis, close contacts of the patient were investigated. Two family mem-

bers of the patient did not have any symptoms compatible with HFRS; their serum samples were tested and found negative for antihantavirus IgG. Because the patient was a nurse, possible nosocomial transmission and sources were also investigated and excluded. On the basis of the patient's strong history of exposure to rodents at home, further investigation focused on the domestic rodent population.

From October 14 through 16, 2008, 110 rodent traps were set within and surrounding the patient's house.

The total catch was 32 rodents, of which 16 were *Rattus norvegicus*, 7 *R. exulans*, 5 *R. argentiventer,* and 4 *Bandicota indica*, respectively. By using ELISA, IFA, and Western blot, antihantavirus IgG was detected in serum from 7 rats, of which 5 were *R. norvegicus*, 1 *R. argentiventer*, and 1 *B. indica*. Further analysis using RT-PCR identified 2 SEOV strains from *R. norvegicus and R. argentiventer* captured in the patient's house. The M segment of 1 identified SEOV strain (24D1208) was sequenced and compared with 22
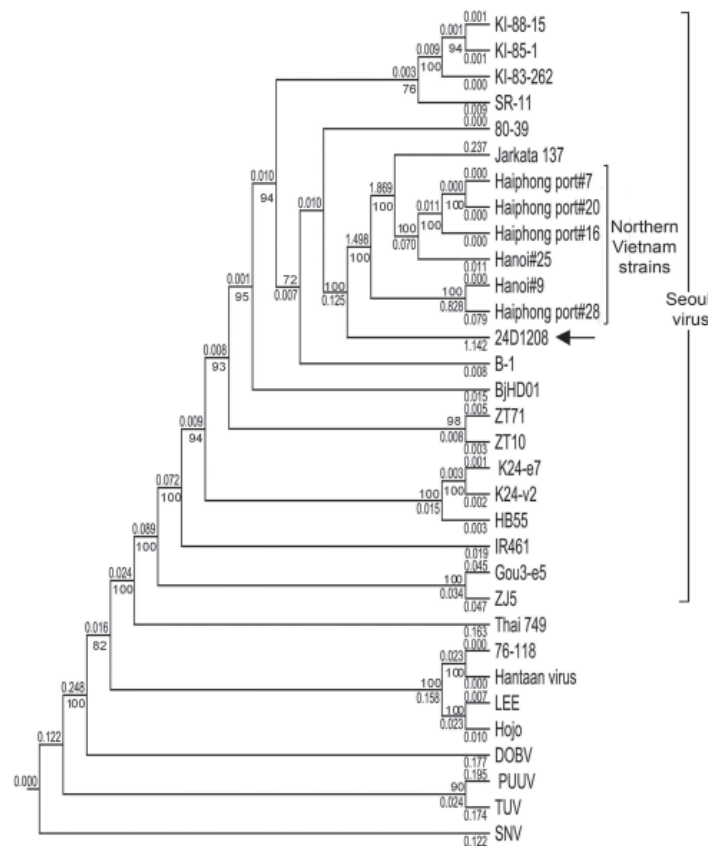

Figure. Phylogenetic tree (CLC-Combined Workbench 3) showing partial sequences of the medium segment (nt 810–2355). The newly identified Seoul virus (SEOV) was denoted as 24D1208 (arrow). The M segment sequences of the reference strains are: SEOV strains KI-88-15 (D17594), KI-85-1 (D17593), KI-83-262 (D17592), SR11 (M34882), 80–39 (S47716), Jakarta137 (AJ620583), Haiphong port #7 (AB355728), Haiphong port #20 (AB355730), Haiphong port #16 (AB355729), Hanoi #25 (AB355733), Hanoi #9 (AB355732), Haiphong port #28 (AB355731), B-1 (X53861), BjHD01 (DQ133505), ZT71 (EF117248), ZT10 (DQ159911), K24-e7 (AF288652), K24-v2 (AF288654), HB55 (AF035832), IR461 (AF458104), Gou3-e5 (AF288650), and ZJ5 (FJ811839); Thailand virus strain 749 (L08756); Hantaan virus strains 76–118 (M14627), Hantaan (NC005219), LEE (D00377) and Hojo (D00376); Dobrava virus (DOBV) strain Dobrava (L33685); Puumala virus strain Sotkamo (X61034); Tula virus (TUV) strain Tula/Moravia/5302v/95 (Z69993); and Sin Nombre virus (SNV) strain NMH10. The numbers at the nodes are bootstrap confidence levels for 1,000 replications. Only bootstrap support values >70% are shown.

SEOV strains, 6 of which were from *R. norvegicus* rodents captured in urban areas of North Vietnam. Phylogenetic analysis showed that this SEOV belonged to the Vietnamese SEOV genotype (Figure).

We describe a clinical case of hantavirus infection and its potential rodent reservoir occurring in Vietnam. The clinical manifestations of the case-patient were compatible with SEOV infection, which is responsible for a moderate form of HFRS (*10*). Also, HFRS caused by SEOV occurs in urban rather than rural areas, unlike other hantavirus infections. Our epidemiologic findings were compatible with other studies indicating the source of infection was the case-patient's home, the only place where she had a history of exposure to rodents. Although viral RNA could not be obtained from the case-patient for genotyping, the genomic comparison of the viral strains from rodents captured in the case-patient's home and elsewhere in Vietnam suggested that the source of infection was local rodents. This report provides additional evidence that hantavirus infection is a worldwide problem and is likely underdiagnosed in Vietnam and other countries where simple standardized laboratory diagnostics are not widely available.

**Vu Thi Que Huong,
Kumiko Yoshimatsu,
Vu Dinh Luan, Le Van Tuan, Le
Nhi, Jiro Arikawa,
and Tran Minh Nhu Nguyen**

Author affiliations: Pasteur Institute, Ho Chi Minh City, Vietnam (V.T.Q. Huong, V.D. Luan, L. Nhi); Hokkaido University Graduate School of Medicine, Sapporo, Japan (K. Yoshimatsu, J. Arikawa); World Health Organization Technical Office, Ho Chi Minh City (L.V. Tuan); and Vietnam Field Epidemiology Training Program Office, Hanoi, Vietnam (T.M.N. Nguyen)

References

1. Bi Z, Formenty PBH, Roth CE. Hantavirus infection: a review and global update. J Infect Dev Ctries. 2008;2:3–23. DOI: 10.3855/jidc.317
2. Rollin PE, Nawrocka E, Rodhain F. Serological data on hemorrhagic fever with renal syndrome in Southeast Asia. Bull Soc Pathol Exot Filiales. 1986;79:473–5.
3. Lee PW, Svedmyr A, Gajdusek DC, Gibbs CJ, Nystrom K. Antigenic difference between European and East Asian viruses causing haemorrhagic fever with renal syndrome. Lancet. 1981;2:256–7.
4. Araki K, Yoshimatsu K, Ogino M, Ebihara H, Lundkvist A, Kariwa H, et al. Truncated hantavirus nucleocapsid proteins for serotyping Hantaan, Seoul, and Dobrava hantavirus infections. J Clin Microbiol. 2001;39:2397–404. DOI: 10.1128/JCM.39.7.2397-2404.2001
5. Miyamoto H, Kariwa H, Araki K, Lokugamage K, Hayasaka D, Cui BZ, et al. Serological analysis of hemorrhagic fever with renal syndrome (HFRS) patients in Far Eastern Russia and identification of the causative hantavirus genotype. Arch Virol. 2003;148:1543–56. DOI: 10.1007/s00705-003-0113-x
6. Schmidt J, Jandrig B, Klempa B, Yoshimatsu K, Arikawa J, Meisel H, et al. Nucleocapsid protein of cell culture-adapted Seoul virus strain 80–39: analysis of its encoding sequence, expression in yeast and immunoreactivity. Virus Genes. 2005;30:37–48. DOI: 10.1007/s11262-004-4580-2
7. Yoshimatsu K, Arikawa J, Kariwa H. Application of a recombinant baculovirus expressing hantavirus nucleocapsid protein as a diagnostic antigen in IFA test: cross reactivities among 3 serotypes of hantavirus which causes hemorrhagic fever with renal syndrome (HFRS). J Vet Med Sci. 1993;55:1047–50.
8. Ogino M, Ebihara H, Lee BH, Araki K, Lundkvist A, Kawaoka Y, et al. Use of vesicular stomatitis virus pseudotypes bearing Hantaan or Seoul virus envelope proteins in a rapid and safe neutralization test. Clin Diagn Lab Immunol. 2003;10:154–60.
9. Zuo SQ, Zhang PH, Jiang JF, Zhan L, Wu XM, Zhao WJ, et al. Seoul virus in patients and rodents from Beijing, China. Am J Trop Med Hyg. 2008;78:833–7.
10. Lee HW. Hemorrhagic fever with renal syndrome in Korea. Rev Infect Dis. 1989;11(Suppl 4):S864–76.

Address for correspondence: Tran Minh Nhu Nguyen, Vietnam Field Epidemiology Training Program Office, 63 Hoang Cau St, Hanoi, Vietnam; email: tmnn69@yahoo.com

Origin of Highly Pathogenic Porcine Reproductive and Respiratory Syndrome Virus, China

To the Editor: A highly pathogenic porcine reproductive and respiratory syndrome virus (HP-PRRSV), which affected >2 million pigs, emerged in early 2006 in the People's Republic of China. The disease was characterized by high fever (41°C), high illness rates (50%–100%), and high death rates (20%–100%) for pigs of all ages (*1*). A number of HP-PRRSVs have been isolated from 2006 through 2009 from infected pigs in different provinces of China and confirmed to be the causative agent of the new outbreaks (*1,2*). These HP-PRRSVs have a deletion of 30 amino acids in nonstructural protein 2 (NSP-2). However, the evolutionary origin and path of the HP-PRRSV remain unknown.

We analyzed the full-length sequences of 67 PRRSVs: 35 HP-PRRSVs (HuN4 and LNSY-08-1 isolated in our laboratory and 33 viruses isolated in other laboratories), 28 classic PRRSVs (18 viruses isolated from China and 10 viruses representing other Asian countries and North