

The molecular masses of the PrP^{res} moieties from the 2 cows were also clearly distinct from those from controls with L- and H-BSE (Figure). For samples from animals with H-BSE, enzymatic deglycosylation demonstrated PrP^{res} subtypes, 1 and 2, the latter being a C-terminal PrP^{res} fragment of ≈12–14 kDa (6). To investigate whether the novel PrP^{res} type corresponds to PrP^{res} subtype 2, we compared samples from cow 2 with those from the H-BSE control by Western blot. The PrP^{res} type from the 2 cows reported here and PrP^{res} subtype 2 from the H-BSE control were indeed distinct (Figure).

We report a novel PrP^{res} signature in 2 cows with BSE diagnoses determined according to established criteria. Combining Western blot analysis with an epitope mapping strategy, we ascertained that these animals displayed an N terminally truncated PrP^{res} different from currently classified BSE prions (Figure). The interpretation of these findings remains difficult because neuropathologic and systematic clinical data for the 2 cases are not available. Moreover, the tissue samples were autolyzed, and the question of whether this affected the PrP^{res} molecular signature is of concern. Nonetheless, our findings raise the possibility that these cattle were affected by a prion disease not previously encountered and distinct from the known types of BSE. To confirm this possibility and to assess a potential effect on disease control and public health, in vivo transmission studies using transgenic mouse models and cattle are ongoing. Until results of these studies are available, molecular diagnostic techniques should be used so that such cases are not missed.

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References

- Colby DW, Prusiner SB. Prions. Cold Spring Harb Perspect Biol. 2011; 3:a006833. doi:10.1101/cshperspect.a006833
- Jacobs JG, Langeveld JP, Biacabe AG, Acutis PL, Polak MP, Gavier-Widen D, et al. Molecular discrimination of atypical bovine spongiform encephalopathy strains from a geographical region spanning a wide area in Europe. J Clin Microbiol. 2007;45:1821–9. doi:10.1128/JCM.00160-07
- Seuberlich T, Heim D, Zurbriggen A. Atypical transmissible spongiform encephalopathies in ruminants: a challenge for disease surveillance and control. J Vet Diagn Invest. 2010;22:823–42. doi:10.1177/104063871002200601
- Schaller O, Fatzer R, Stack M, Clark J, Cooley W, Biffger K, et al. Validation of a Western immunoblotting procedure for bovine PrP(Sc) detection and its use as a rapid surveillance method for the diagnosis of bovine spongiform encephalopathy (BSE). Acta Neuropathol. 1999;98:437–43. doi:10.1007/s004010051106
- Office Internationale des Epizooties. OIE rules for the official confirmation of BSE in bovines (based on an initial reactive result in an approved rapid test) by using a second rapid test. 2009 [cited 2011 Oct 19]. http://vla.defra.gov.uk/science/docs/sci_tse_oie_bse.pdf
- Biacabe AG, Jacobs JG, Benesik A, Langeveld JP, Baron TG. H-type bovine spongiform encephalopathy: complex molecular features and similarities with human prion diseases. Prion. 2007;1:61–8. doi:10.4161/pri.1.1.3828

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Hantavirus in Bat, Sierra Leone

To the Editor: Hantaviruses (family *Bunyaviridae*) are transmitted from rodent reservoirs to humans. These viruses cause life-threatening human diseases: hantavirus cardiopulmonary syndrome in the Americas and hemorrhagic fever with renal syndrome in Asia and Europe (1). Since 2006, indigenous hantaviruses were reported also from Africa. Sangassou virus was found in an African wood mouse (*Hylomyscus simus*) in Guinea (2). Discovery of newer African hantaviruses, Tanganya virus and recently Azagny virus, was even more surprising because they were found in shrews (3,4).

The detection of hantaviruses in small mammals other than rodents, such as shrews and also moles (4), increasingly raises questions regarding the real hantavirus host range. Bats (order Chiroptera) are already known to harbor a broad variety of emerging pathogens, including other bunyaviruses (5). Their ability to fly

and social life history enable efficient pathogen maintenance, evolution, and spread. Therefore, we conducted a study on hantaviruses in bats from Africa.

A total of 525 tissue samples from 417 bats representing 28 genera were tested for the presence of hantavirus RNA. Samples originated from different regions in western and central Africa and were collected during 2009 and early 2011. Total RNA was extracted from tissue samples and reverse transcribed. cDNA was screened by PCR specific for sequences of the large genomic segment across the genus *Hantavirus* (2).

One sample yielded a product of the expected size and was subjected to cloning and sequencing. The positive sample (MGB/1209) was obtained from 1 of 18 investigated slit-faced bats (family Nycteridae). The animal was trapped at the Magboi River within Gola National Park, Sierra Leone ($7^{\circ}50.194'N$, $10^{\circ}38.626'W$), and the identification as *Nycteris hispida* has been verified with the voucher specimen (RCJF529). Histologic examination of organs of the animal showed no obvious pathologic findings.

The obtained 414-nt sequence covers a genomic region, which was found to correspond to nt position 2,918–3,332 in the large segment open reading frame of prototypic Hantaan virus. Bioinformatic analysis on the amino acid level showed highest degrees of identity to shrew- and mole-associated hantaviruses (Thottapalayam virus 73.0%, Altai virus 69.7%, Nova and Imjin virus 69.3%). On the basis of tree topology of a maximum-likelihood phylogenetic tree, the sequence does not cluster with rodent-associated hantaviruses but groups with those found in shrews and moles (Figure).

Considering that bats are more closely related to shrews and moles than to rodents (6), a certain genetic similarity of a putative

bat-borne hantavirus with shrew- and mole-associated hantaviruses seems reasonable. Notably, shrew-associated Thottapalayam virus (India) and Imjin virus (South Korea) seem to be closer relatives, and African Tanganya virus (Guinea) and Azagny virus (Côte d'Ivoire) are more distantly related. Additional sequence data is needed for more conclusive phylogenetic analyses.

Because the new amino acid sequence is at least 22% divergent

from those of other hantaviruses, we conclude that the bat was infected with a newly found hantavirus. We propose the putative name Magboi virus (MGBV) for the new virus because it was detected in an animal captured at the Magboi River in Sierra Leone. The MGBV nucleotide sequence is novel and has not been known or handled before in our laboratory. Before this study, hantavirus nucleic acid was found in lung and kidney tissues of bats from

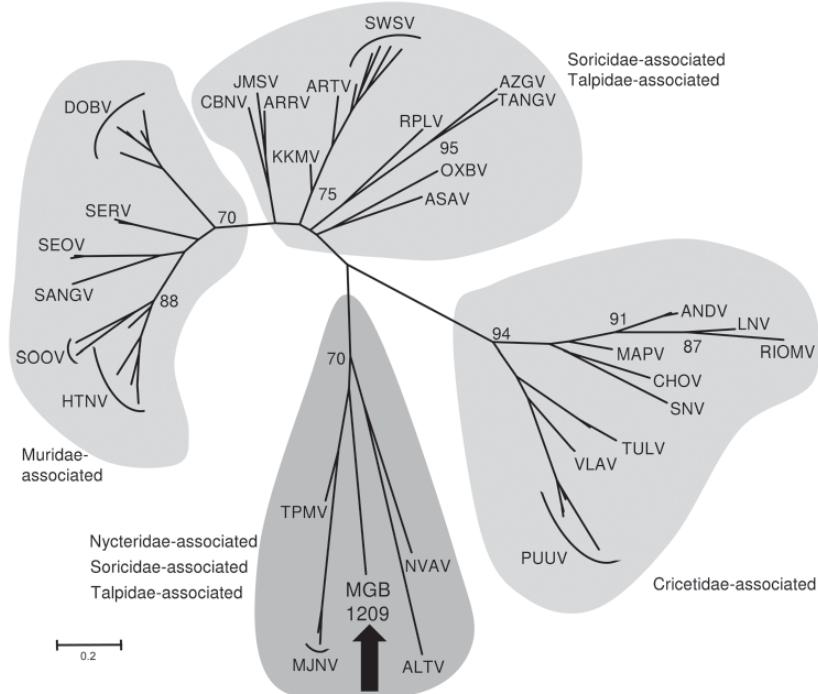


Figure. Maximum-likelihood phylogenetic tree of MGB/1209 virus based on partial large segment sequence (414 nt) and showing the phylogenetic placement of the novel sequence from *Nycteris* spp. bat compared with hantaviruses associated (i) with shrews and moles: Altai virus (ALTV), Artybash virus (ARTV), Asama virus (ASAV), Ash River virus (ARRV), Azagny virus (AZGV), Camp Ripley virus (RPLV), Cao Bang virus (CBNV), Imjin virus (MJNV), Jemez Springs virus (JMSV), Kenkeme virus (KKMV), Nova virus (NVAV), Oxbow virus (OXBV), Seewis virus (SWSV), Tanganya virus (TGNV), Thottapalayam virus (TPMV), and (ii) with rodents: Andes virus (ANDV), Chocho virus (CHOV), Dobrava-Belgrade virus (DOBV), Hantaan virus (HTNV), Laguna Negra virus (LNV), Maporal virus (MAPV), Puumala virus (PUUV), Rio Mamore virus (RIOMV), Sangassou virus (SANGV), Seoul virus (SEOV), Serang virus (SERV), Sin Nombre virus (SNV), Soochong virus (SOOV), Tula virus (TULV), Vladivostok virus (VLAV). The list of the accession numbers used in the analysis is available from the authors upon request. The tree was computed by using MEGA5 (<http://www.megasoftware.net>). The Tamura 3-parameter model with gamma-distributed rate heterogeneity and a proportion of invariant sites (T92 +G + I) was selected as the best fit evolutionary model according to the Bayesian information criterion calculated with MEGA5. The values at the tree branches are the bootstrap support values calculated from 500 replicates. Scale bar indicates an evolutionary distance of 0.2 substitutions per position in the sequence. The gray areas indicate association of hantaviruses with reservoir host families. The MGB/1209 partial sequence of the large genomic segment was deposited in GenBank under accession no. JN037851.

the genera *Eptesicus* and *Rhinolophus* in South Korea. However, nucleotide sequencing showed the presence of prototypical Hantaan virus indicating a spillover infection or laboratory contamination (7).

Further screening is necessary to confirm *N. hispida* as a natural reservoir host of the virus. Although the presented bat-associated sequence is obviously distinct from other hantaviruses, which suggests association with a novel natural host, a spillover infection from another, yet unrecognized host cannot be ruled out. However, detection of the virus exclusively in 1 organ (lung but not in liver, kidney, and spleen; data not shown) suggests a persistent infection that is typically observed in natural hosts of hantaviruses (8).

To date, only a few reports exist on cases of hemorrhagic fever with renal syndrome in Africa (9,10). However, underreporting must be assumed because the symptoms resemble those of many other febrile infections. Moreover, in cases of infections by non–rodent-associated hantaviruses, cross-reactivity with routinely used rodent-borne virus antigens should be limited and may hamper human serodiagnosis (1). The results suggest that bats, which are hosts of many emerging pathogens (5), may act as natural reservoirs for hantavirus. The effect of this virus on public health remains to be determined.

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References

- Krüger DH, Schonrich G, Klempa B. Human pathogenic hantaviruses and prevention of infection. *Hum Vaccin*. 2011;7:685–93. doi:10.4161/hv.7.6.15197
- Klempa B, Fichet-Calvet E, Lecompte E, Auste B, Aniskin V, Meisel H, et al. Hantavirus in African wood mouse, Guinea. *Emerg Infect Dis*. 2006;12:838–40.
- Klempa B, Fichet-Calvet E, Lecompte E, Auste B, Aniskin V, Meisel H, et al. Novel hantavirus sequences in shrew, Guinea. *Emerg Infect Dis*. 2007;13:520–2. doi:10.3201/eid1303.061198
- Kang HJ, Kadjo B, Dubey S, Jacquet F, Yanagihara R. Molecular evolution of Azagny virus, a newfound hantavirus harbored by the West African pygmy shrew (*Crocidura obscurior*) in Côte d'Ivoire. *Virology*. 2011;8:373. doi:10.1186/1743-422X-8-373
- Calisher CH, Childs JE, Field HE, Holmes KV, Schountz T. Bats: important reservoir hosts of emerging viruses. *Clin Microbiol Rev*. 2006;19:531–45. doi:10.1128/CMR.00017-06
- Murphy WJ, Eizirik E, Johnson WE, Zhang YP, Ryder OA, O'Brien SJ. Molecular phylogenetics and the origins of placental mammals. *Nature*. 2001;409:614–8. doi:10.1038/35054550
- Jung YT, Kim GR. Genomic characterization of M and S RNA segments of hantaviruses isolated from bats. *Acta Virol*. 1995;39:231–3.
- Schönrich G, Rang A, Lutteke N, Rafferty MJ, Charbonnel N, Ulrich RG. Hantavirus-induced immunity in rodent reservoirs and humans. *Immunol Rev*. 2008;225:163–89. doi:10.1111/j.1600-065X.2008.00694.x
- Coulaud X, Chouaib E, Georges AJ, Rollin P, Gonzalez JP. First human case of haemorrhagic fever with renal syndrome in the Central African Republic. *Trans R Soc Trop Med Hyg*. 1987;81:686. doi:10.1016/0035-9203(87)90455-X
- Klempa B, Koivogui L, Sylla O, Koulemou K, Auste B, Kruger DH, et al. Serological evidence of human hantavirus infections in Guinea, West Africa. *J Infect Dis*. 2010;201:1031–4. doi:10.1086/651169

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Outbreak of Porcine Epidemic Diarrhea in Suckling Piglets, China

To the Editor: Beginning in October 2010, porcine epidemic diarrhea (PED), caused by a coronaviral infection affecting pigs, emerged in China in an outbreak characterized by high mortality rates among suckling piglets. The outbreak overwhelmed >10 provinces in southern China, and >1,000,000 piglets died. This outbreak was distinguished by ≈100% illness among piglets after birth (predominantly within 7 days and

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