that the prevalence of EBOV in these tested bat species is greater than that previously detected in *E. helvum* bats (1/262 serum samples) (*1*). The higher estimated prevalence in these species occurred despite the fact that *E. helvum* bats live in large colonies comprising several million animals, which make the species an ideal host for acute RNA virus infections. The relatively low seroprevalence of EBOV among *E. helvum* bats compared with that among sympatric species is contrary to our findings for a lyssavirus and an uncharacterized henipavirus (*3*,*4*). Our results, therefore, lead us to question what factors (e.g., host, ecologic) limit EBOV circulation in straw-colored fruit bats. Virus isolation is required to characterize EBOVs circulating among fruit bats in Ghana, and additional testing, including longitudinal sampling of bats, is required to further investigate the epidemiology of EBOV in West Africa. Possible public health threats should also be investigated and addressed. These initial findings, however, suggest that the risk for human infection with EBOV might be greater from bat-human contact in rural and forest settings than from urban-roosting *E. helvum* bats.


### Acknowledgments

We thank the Wildlife Division of the Forestry Commission, Ghana, and the Veterinary Services Directorate for their continued support for this study, and 2 anonymous reviewers for their comments.

This study was funded by the Wellcome Trust (to D.T.S.H.), UK Department for Environment, Food and Rural Affairs (grant VT0105), and the Research and Policy for Infectious Disease Dynamics program (RAPIDD [managed by the US Department of Homeland Security and the Fogarty International Center, National Institutes of Health]). A.A.C. is supported by a Royal Society Wolfson Research Merit award.



**David T.S. Hayman, Meng Yu, Gary Crameri, Lin-Fa Wang, Richard Suu-Ire, James L.N. Wood, and Andrew A. Cunningham**

Author affiliations: University of Cambridge, Cambridge, UK (D.T.S. Hayman, J.L.N. Wood); Zoological Society of London, London, UK (D.T.S. Hayman, A.A. Cunningham); Animal Health and Veterinary Laboratories Agency, Weybridge, UK (D.T.S. Hayman); Colorado State University, Fort Collins, CO, USA (D.T.S. Hayman); CSIRO Livestock Industries, Geelong, Victoria, Australia (M. Yu, G. Crameri, L.-F. Wang); and Ghana Forestry Commission, Accra, Ghana (R. Suu-Ire)


DOI: http://dx.doi.org/10.3201/eid1807.111654

### References


1. Hayman DTS, Emmerich P, Yu M, Wang LF, Suu-Ire R, Fooks AR, et al. Long-term survival of an urban fruit bat seropositive for Ebola and Lagos bat viruses. PLoS ONE. 2010;5:e11978. http://dx.doi.org/10.1371/journal.pone.0011978
2. Richter HV, Cumming GS. First application of satellite telemetry to track African straw-coloured fruit bat migration. J Zool (Lond). 2008;275:172–6. http://dx.doi.org/10.1111/j.1469-7998.2008.00425.x
3. Hayman DTS, Fooks AR, Horton DL, Suu-Ire R, Breed AC, Wood JL, et al. Antibodies against Lagos bat virus in megachiroptera in West Africa. Emerg Infect Dis. 2008;14:926–8. http://dx.doi.org/10.3201/eid1406.071421
4. Hayman DTS, Suu-Ire R, Breed AC, McEachern JA, Wang L, Wood JL, et al. Evidence of henipavirus infection in West African fruit bats. PLoS ONE. 2008;3:e2739. http://dx.doi.org/10.1371/journal.pone.0002739
5. Marsh GA, Haining J, Robinson R, Foord A, Yamada M, Barr JA, et al. Ebola Reston virus infection of pigs: clinical significance and transmission potential. J Infect Dis. 2011;204(Suppl 3):S804–9. http://dx.doi.org/10.1093/infdis/jir300
6. Leroy EM, Kumulungui B, Pourrut X, Rouquet P, Hassanin A, Yaba P, et al. Fruit bats as reservoirs of Ebola virus. Nature. 2005;438:575–6. http://dx.doi.org/10.1038/438575a
7. Le Guenno B, Formenty P, Wyers M, Gounon P, Walker F, Boesch C. Isolation and partial characterisation of a new strain of Ebola virus. Lancet. 1995;345:1271–4. http://dx.doi.org/10.1016/S0140-6736(95)90925-7
8. Ksiazek TG, West CP, Rollin PE, Jahrling PB, Peters PB. ELISA for the detection of antibodies of antibodies to Ebola viruses. J Infect Dis. 1999;179(Suppl 1):S192–8. http://dx.doi.org/10.1086/514313
9. Towner JS, Sealy TK, Khristova ML, Albariño CG, Conlan S, Reeder SA, et al. Newly discovered Ebola virus associated with hemorrhagic fever outbreak in Uganda. PLoS Pathog. 2008;4:e1000212. http://dx.doi.org/10.1371/journal.ppat.1000212
10. Negredo A, Palacios G, Vázquez-Morón S, González F, Dopazo H, Molero F, et al. Discovery of an Ebolavirus-like filovirus in Europe. PLoS Pathog. 2011;7:e1002304. http://dx.doi.org/10.1371/journal.ppat.1002304


Address for correspondence: David T.S. Hayman, University of Cambridge, Madingley Rd, Cambridge, CB3 0ES, UK; email: davidtshayman@gmail.com

# Outbreak-associated Novel Duck Reovirus, China, 2011

**To the Editor:** In 2011, an unidentified disease in Pekin ducks (*Anas platyrhynchos*) was reported in People's Republic of China. The infection caused death in 40% of ducks of various age and 35%–40% mortality in different flocks. Clinical signs included unstable gait, weakness in legs, and diarrhea. At necropsy, large necrotic foci were observed in the spleens. All classical endemic and emerging viruses, such as duck enteritis virus, duck hepatitis virus, duck flavivirus, duck parvovirus, and avian influenza virus, could be excluded as the causative agent by PCR and serologic methods. To identify the cause of the disease, we tested tissue from affected ducks and subsequently isolated a novel duck-pathogenic orthoreovirus from the livers of affected ducks.

Avian orthoreoviruses (ARVs) belong to the family *Reoviridae*, genus *Orthoreovirus* (*1*). The virions are nonenveloped, with icosahedral symmetry and a double capsid containing 10 double-stranded RNA segments that can be separated by polyacrylamide gel electrophoresis into 3 size classes: large (L1–L3), medium (M1–M3), and small (S1–S4) (*2*,*3*). ARVs cause a range of diseases in chicken, including viral arthritis/tenosynovitis, and are associated with respiratory disease, enteric disease, inclusion body hepatitis, hydropericardium, runting stunting syndrome, malabsorption syndrome, and sudden death. ARVs also have been isolated from the Muscovy duck (*Cairina moschata*). Muscovy duck reovirus infection caused illness in 30% and death in 20% of ducks on poultry farms in Israel (*4*). In China, reovirus infection has been reported in Muscovy ducklings, with a resulting death rate of 10%–30% since 1997 (*5*). The isolated reovirus was highly pathogenic to 1-day-old Muscovy ducklings by experimental infection. However, the Muscovy duck reovirus isolate was nonpathogenic for Pekin ducks when inoculated subcutaneously (*4*).

Since 2007, three isolates of orthoreovirus were confirmed in Pekin ducks from several duck farms in China. However, experiment infection with the isolates did not cause death (*6*). In 2011, farmers and veterinarians in China reported to the Animal Health Services and National Research Institutes an unidentified disease in ducks that spread rapidly around the county. We conducted further investigation to identify the causative agent of this disease. The diseased ducks showed depression and leg weakness. Large necrotic foci were observed in the spleens of the dead ducks. Histopathologic examination showed necrotic foci and granulomas in the spleen. Focal hepatic necrosis and proliferation of bile ducts were seen in the liver. Virus isolation from liver homogenate was conducted in duck embryo fibroblast cultures. At 48 hours after infection, a strong cytopathic effect was observed, including syncytium formation. All duck embryos experimentally infected with the isolate died within 48–72 hours after infection. The dead embryos showed swollen livers with petechial hemorrhages. Spherical, spiked virus particles, consistent with those of members of the family *Reoviridae*, were observed by electron microscopy. As reported (*7*), the diameter of the particles was ≈85 nm (online Appendix Figure, wwwnc.cdc.gov/EID/article/18/7/12-0190-FA1.htm). The RNA extracted from DRV-infected duck embryo fibroblast cultures showed 10 dsRNA segments in 3 size classes (L1–3, M1–3, and S1–4) on polyacrylamide gel electrophoresis. The isolate was designated as novel duck reovirus, DRV-TH11. The pathogenicity of DRV-TH11 was tested by infecting 10-day-old Pekin ducks subcutaneously at a dose of 4 × $10^{4.5}$ 50% tissue culture infective dose. Experimental infection caused death on day 3 after infection. The clinical signs and histopathologic examination show the same features as the naturally infected ducks.

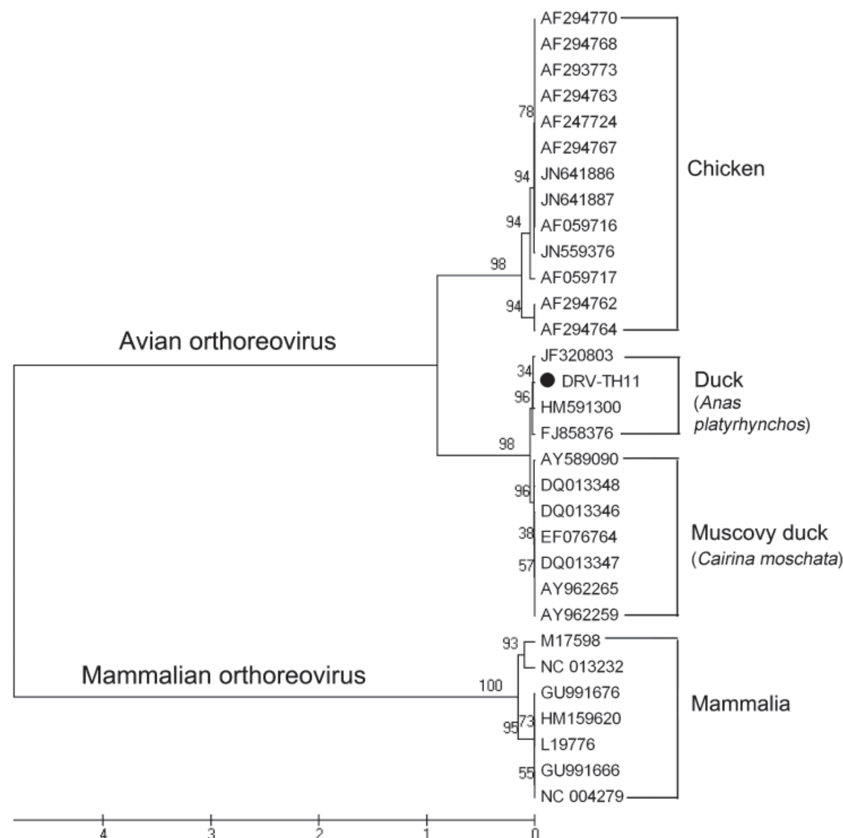

Figure. Phylogenetic relationship between DRV-TH11 isolate and orthoreovirus of the avian orthoreovirus (ARV) and mammalian orthoreovirus (MRV). ARV includes chicken reovirus, Muscovy duck reovirus, and Pekin duck reovirus. GenBank accession numbers of the sequences in the analysis are indicated in the tree. The neighbor-joining tree is based on the complete sequence of s2 gene (1,251 nt). Numbers at nodes represent the percentage of 1,000 bootstrap replicates (values <50 are not shown). Scale bar indicates a branch length corresponding to 100 character-state changes.

For phylogenetic analyses, the S2 gene was amplified by reverse transcription PCR with avian reovirus–specific primers. The complete sequence of the S2 gene (GenBank accession no. JQ664689) was aligned

with 30 published orthoreovirus sequences, including data on all 3 newly obtained sequences from Pekin duck reovirus in China in 2008 and 2011. Phylogenetic relationship was assessed by using the neighbor-joining method based on a Tamura 3-parameter model and bootstrap analysis (1,000 replicates) as implemented in MEGA5 (*8*). The phylogenetic tree shows that the complete sequence of S2 gene is distinct but clusters closely with sequences from all 3 Pekin duck isolates within the ARVs serogroup, which suggests that the novel virus is an ARV-like virus within the genus *Orthoreovirus* (Figure).

In summary, we isolated a novel duck-pathogenic orthoreovirus from the liver of affected Pekin ducks. The regression test in its natural host animal showed that the newly isolated virus caused their deaths. This finding highlights the need to prevent and control this highly transmissible infectious agent. Further study is needed to determine what role the newly isolated DRV played in the 2011 outbreaks on many of the duck farms in China.

This work was supported by the Fundamental Research Funds for the Central Institutes program (no.2011JB06, 2011JB13), Special Fund for Agro-scientific Research in the Public Interest (no. 201003012), the Chinese Natural Sciences Foundation (31101848), and the National Advanced Technology Research and Development Program of China (863 Program) (no. 2011AA10A200).

**Zongyan Chen, Yinqi Zhu, Chuanfeng Li, and Guangqing Liu**

Author affiliation: Shanghai Veterinary Research Institute–Chinese Academy of Agricultural Sciences, Shanghai, People's Republic of China

DOI: http://dx.doi.org/10.3201/eid1807.120190

## References

1. Jones RC. Avian reovirus infections. Rev Sci Tech. 2000;19:614–25.
2. Gouvea VS, Schnitzer TJ. Polymorphism of the migration of double-stranded RNA genome segments of avian reoviruses. J Virol. 1982;43:465–71.
3. Huhtamo E, Uzcátegui NY, Manni T, Munsterhjelm R, Brummer-Korvenkontio M, Vaheri A, et al. Novel orthoreovirus from diseased crow, Finland. Emerg Infect Dis. 2007;13:1967–9. http://dx.doi.org/10.3201/eid1312.070394
4. Malkinson M, Perk K, Weisman Y. Reovirus infection of young Muscovy ducks (*Cairina moschata*). Avian Pathol. 1981;10:433–40. http://dx.doi.org/10.1080/03079458108418493
5. Wu B, Chen J, Yao J. Pathogenicity of Muscovy duck reovirus isolate B3. Chin J Prev Vet Med. 2001;23:422–6.
6. Liu Q, Zhang G, Huang Y, Ren G, Chen L, Gao J, et al. Isolation and characterization of a reovirus causing spleen necrosis in Pekin ducklings. Vet Microbiol. 2011;148:200–6. http://dx.doi.org/10.1016/j.vetmic.2010.09.016
7. Zhang X, Walker SB, O'Hara D, Nibert ML, Duncan R. Structure of avian orthoreovirus virion by electron cryomicroscopy and image reconstruction. Virology. 2005;343:25–35. http://dx.doi.org/10.1016/j.virol.2005.08.002
8. Tamura K, Peterson N, Stecher G, Nei M, Kumar S. MEGA5: molecular evolutionary genetics analysis using maximum likelihood, evolutionary distance, and maximum parsimony methods. Mol Biol Evol. 2011;28:2731–9. http://dx.doi.org/10.1093/molbev/msr121

Address for correspondence: Guangqing Liu, Shanghai Veterinary Research Institute, Chinese Academy of Agricultural Sciences, No. 518 Ziyue Rd, Minhang District, Shanghai 200241, People's Republic of China; email: zychen@shvri.ac.cn

# Considerations for Oral Cholera Vaccine Use during Outbreak after Earthquake in Haiti, 2010–2011

**To the Editor:** We wish to thank Date et al. for their clear discussion of the arguments against the use of oral cholera vaccines (OCVs) in Haiti in 2010–11 (*1*). The epidemic curve in their article suggests that the control activities had an effect on mortality rates, resulting in a decrease in case-fatality rates to <1%. This finding is a remarkable success not achieved during the recent cholera outbreak in Zimbabwe that affected 98,531 persons, of whom 4,282 (4.3%) died (*2*). However, the article does not discuss the lack of effect of the control measures in Haiti on the spread of the epidemic. Considering the failure of containment, it would have been interesting to read how the authors judge the recommendation not to vaccinate, with the benefit of hindsight.

The authors list a catalog of arguments against the use of OCVs in outbreaks. These included the priority of water provision and cholera treatment measures, how modeling data provided no convincing justification for vaccination campaigns, how mobile populations cannot be trusted to take 2 doses, the time for a 2-dose vaccine to generate immunity, the logistic challenges in a setting of inadequate infrastructure and human resources, the cold chain requirements, the difficulty in transport of bulky vaccine, clean water requirements for the buffer, civil