are indicators of dengue and chikungunya, point to ZIKV as the probable cause of several of the reported cases. Furthermore, laboratory-confirmed cases of infection with ZIKV were simultaneously identified in other cities within metropolitan Salvador (*6,7*) and in other states in Brazil (*8*). Low diagnosis of ZIKV infection is likely because viremia levels among infected patients appear to be low (*9*).

The spread of ZIKV represents an additional challenge for public health systems, particularly because of the risk for concurrent transmission of DENV and CHIKV by the same vectors, *Ae. aegypti* and *Ae. albopictus* mosquitoes, which are abundant throughout tropical and subtropical regions. To date, the largest outbreak of chikungunya in Brazil occurred in 2014 in Feira de Santana, Bahia, ≈100 km from Salvador, where dengue is also prevalent (*10*).

This report illustrates the potential for explosive simultaneous outbreaks of ZIKV, CHIKV, and DENV in the Western Hemisphere and the increasing public health effects of *Aedes* spp. mosquitoes as vectors. The apparent increase in reports of Guillain-Barré syndrome during the outbreak deserves further investigation to elucidate whether this syndrome is associated with ZIKV infection. Public health authorities in Brazil and neighboring countries should plan accordingly.

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Address for correspondence: Guilherme S. Ribeiro, Centro de Pesquisas Gonçalo Moniz, Fundação Oswaldo Cruz, Rua Waldemar Falcão, 121, Candeal, 40296-710 Salvador, Bahia, Brazil; email: guilherme.ribeiro@bahia.fiocruz.br

Emerging Rabbit Hemorrhagic Disease Virus 2 (RHDVb), Australia

Robyn N. Hall,[1] Jackie E. Mahar,[1] Stephanie Haboury, Vicky Stevens, Edward C. Holmes, Tanja Strive

Author affiliations: CSIRO Health and Biosecurity, Canberra, Australian Capital Territory, Australia (R.N. Hall, J.E. Mahar, S. Haboury, T. Strive); Invasive Animals CRC, Bruce, Australian Capital Territory, Australia (R.N. Hall, S. Haboury, T. Strive); The University of Sydney School of Biological Sciences Sydney, New South Wales, Australia (J.E. Mahar, E.C. Holmes); CSIRO Australian Animal Health Laboratories, Geelong, Victoria, Australia (V. Stevens)

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To the Editor: In May 2015 an isolate of the recently emerged variant of rabbit hemorrhagic disease virus (RHDV), RHDV2, was identified in an Australian wild rabbit (*Oryctolagus cuniculus*). RHDV2 (also called RHDVb) was first described in outbreaks in France in 2010 (*1*), then Italy and Spain in 2011 (*2*,*3*) and in Portugal from 2012 onwards (*4*). The virus is a genetically and antigenically distinct variant of RHDV that is able to partially overcome immunity to classical strains of RHDV (*1*,*2*). In contrast

[1]These first authors contributed equally to this article.

to case-fatality rates for other strains of RHDV, those for RHDV2 infection have been reported to be lower in mature rabbits (0%–75% in 1 study, compared with >90% for classic RHDV infection) (*3*) but higher (50% in 1 study) in rabbit kittens as young as 30 days of age, which are normally highly resistant to lethal RHDV infection (*2*). RHDV2 has been reported to spread effectively in domestic rabbits in Europe (*3*); it may be replacing existing strains of RHDV that infect wild rabbits on the Iberian Peninsula (*5*), possibly because of its ability to partially overcome immunity to these strains.

As part of ongoing opportunistic surveillance of RHDV field outbreaks, we analyzed 3 isolates from dead adult wild rabbits found in the wider Canberra region of Australia. The first virus isolate (BlMt-1) came from a rabbit found in Australian Capital Territory on May 13, 2015. The second isolate (BlueGums-2) was taken 3 days later from a rabbit in New South Wales, 50 km north of Canberra. On June 9, another dead rabbit, from which the third isolate (BlMt-2) was obtained, was found in the same location as the first. The isolates were initially typed by amplifying and sequencing the capsid gene (*6*), and the results were confirmed independently in 2 laboratories. Subsequently, full-length genome sequencing of the 3 virus isolates was performed by amplifying the viral genomes in overlapping fragments (*6*); the fragments were then sequenced by using Illumina MiSeq technology (*7*).

Phylogenetic analysis revealed that 2 isolates, BlMt-2 and BlueGums-2, were closely related to field strains currently circulating in Australia (*7*) (Figure). Strikingly, the third isolate (BlMt-1) was most closely related to an RHDV2 variant generated by recombination of the RHDV2 capsid gene (Figure, panel B) and the RHDV genogroup 1 nonstructural genes (Figure, panel A), which has recently been reported to be circulating in Portugal and the Azores (*8*,*9*). How the virus variant arrived in Australia is unclear, although our analysis indicates that it likely originated in southern Europe.

In 1991, CSIRO imported the Czech351 strain of RHDV to assess its potential as a biocontrol tool for controlling the European rabbit, which causes massive economic and ecologic damage and is declared a pest species in Australia. In 1995, after initial testing in quarantine, the virus escaped during field trials being conducted on a coastal island through passive fly transmission and subsequently spread across the continent. The RHDV2 variant reported here has not previously been investigated by CSIRO, and the organization did not possess it.

Rabbits are found in ≈70% of the 6.7 million km^2 Australian continent and Tasmania. However, natural outbreaks of RHDV infection are monitored in comparatively few locations, and their detection largely relies on opportunistic sampling. To follow the spread of this new variant and determine its current range, increased surveillance of outbreaks of RHDV infection in both wild and domestic rabbits in Australia is urgently required. The unique traits of strain RHDV2, particularly its ability to overcome immunity to classical RHDV strains (including vaccine strains) (*3*) and to infect rabbits at a younger age (*2*), may have wide-ranging implications for rabbit biocontrol in Australia. In

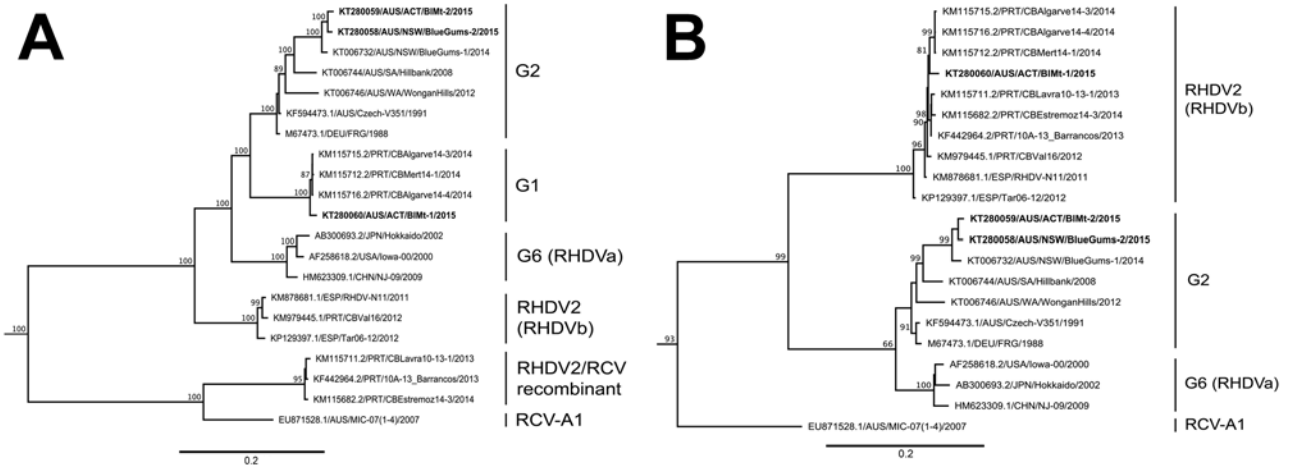

Figure. Maximum-likelihood phylogenetic analysis of the nonstructural protein genes (A) and the capsid gene (B) of rabbit hemorrhagic disease virus (RHDV) sequences. The 3 recent Australian field isolates sequenced for this study (indicated in bold) were aligned with representative RHDV and Australian rabbit calicivirus (RCV-A1) sequences from GenBank (accession numbers indicated in taxa names). Phylogenetic analysis was conducted separately for both the nonstructural genes (panel A) and the capsid gene (panel B). Phylogenies were rooted by using an early European brown hare syndrome virus strain (not shown). Statistical support for individual nodes was estimated from 1,000 bootstrap replicates with values shown for only those nodes where the bootstrap support was ≥70% (and all major nodes). Phylogenies were constructed by using the general time reversible plus gamma model of nucleotide substitution, as determined in jModelTest, by using PhyML(as available in Geneious version 8.1.5; Biomatters Limited, Auckland, New Zealand). Scale bars indicate nucleotide substitutions per site.

parallel with similar efforts in Europe, strategies need to be developed to protect commercial and pet rabbits.

Tracking the spread of RHDV2 in Australia, in competition with existing field strains, highlights the value of Australia's rabbits and their diseases as a model system for emerging infectious diseases. The point releases of both myxoma virus and RHDV into large naive host populations represent a grand experiment in disease emergence and evolution (*10*), which provides a unique opportunity to study the virulence evolution of emerging pathogens as well as their complex interactions with each other. It is notable that since the release of RHDV in Australia in 1995, strains of 1 viral lineage dominate the viral population nationwide despite hundreds of deliberate rereleases of the original virus strain for local rabbit control, which strongly suggests it has a major selective advantage (*7*). That RHDV2 appeared in a wild rabbit is therefore remarkable, particularly because Australian field strains were spreading simultaneously in the same area. Comparing the epidemiology of this strain in Australia to the epidemiology of its well-documented spread in Europe will provide valuable insights into RHDV epidemiology relevant to both continents.

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Address for correspondence: Tanja Strive, CSIRO Health and Biosecurity, Clunies Ross St, Canberra, Australian Capital Territory 2601, Australia; email: tanja.strive@csiro.au

Characteristics of Traveler with Middle East Respiratory Syndrome, China, 2015

Wen Da Guan,[1] Chris Ka Pun Mok,[1] Zi Lin Chen,[1] Li Qiang Feng, Zheng Tu Li, Ji Cheng Huang, Chang Wen Ke, Xilong Deng, Yun Ling, Shi Guan Wu, Xue Feng Niu, Ranawaka A Perera, Yuan Da Xu, Jincun Zhao, Lin Qi Zhang, Yi Min Li, Rong Chang Chen, Malik Peiris, Ling Chen, Nan Shan Zhong

Author affiliations: State Key Laboratory of Respiratory Disease, First Affiliated Hospital of Guangzhou Medical University, Guangzhou, China (W.D. Guan, Z.T. Li, S.G. Wu, X.F. Niu, Y.D. Xu, J. Zhao, Y.M. Li, R.C. Chen, L. Chen, N.S. Zhong); The University of Hong Kong, Hong Kong, China (C.K.P. Mok, R.A. Perera, M. Peiris); Huizhou Municipal Central Hospital, Huizhou, China (Z.L. Chen, Y. Ling); Guanzhou Institute of Biomedicine and Health, Guangzhou (L.Q. Feng, L. Chen); Guangdong Inspection and Quarantine Technology Center, Guangzhou (J.C. Huang); Guangdong Center for Disease Control and Prevention, Guangzhou (C.W. Ke); Guangzhou Eighth People's Hospital, Guangzhou (X. Deng); Tsinghua University School of Medicine, Beijing, China (L.Q. Zhang)

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To the Editor: A traveler returning from the Middle East initiated an outbreak of Middle East respiratory syndrome (MERS) in South Korea in 2015, which resulted in 186 cases and 36 deaths (*1–3*). We report a case of

[1]These authors contributed equally to this article.