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Hunter Island Group Phlebovirus in Ticks, Australia

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To the Editor: A recent article described the isolation and subsequent analysis of a tickborne phlebovirus: Hunter Island Group virus (HIGV), associated with an albatross disease event that occurred in 2002 on Albatross Island, 6 kilometers off the northwest coast of Tasmania, Australia (*1*). The authors present HIGV as a novel isolate; however, new data and historical records demonstrate that the virus was originally isolated in 1983. Provisionally named Albatross Island virus (ABIV), the virus was classified as unidentified because of its uniqueness and dissimilarity to any known virus in Australia. ABIV and HIGV were isolated from ticks of the same species, *Ixodes eudyptidis*, collected from the nests of shy albatross (*Thalassarche cauta*) on Albatross Island, the only island inhabited by albatross within the Hunter Island Group Important Bird Area. At the time of collections, many immature albatross were dying. Records from this time indicate that postmortem blood samples were collected from the birds, and subsequent virus neutralization studies conducted soon after demonstrated that 50% of these samples were ABIV positive. Ensuing testing of samples collected in the next 2 years also identified a positive sample from a black noddy in Queensland (Table). ABIV was subsequently sent for testing at the Arbovirus World Reference Laboratory and, more than a decade later, to the Australian Animal Health Laboratory, Commonwealth Scientific and Industrial Research Organisation, where it was identified as a bunyavirus but remained largely uncharacterized.

We recently sequenced the genome of ABIV by using high-throughput sequencing and have compiled near complete sequences for the large (L), medium (M), and small (S) segments (GenBank accession nos. KM198925–7). Overall, ABIV shares 99% nt identity with HIGV, and thus they can be considered isolates of the same virus. The translated nucleocapsid and S segment nonstructural proteins of both viruses are identical, and the polymerases and glycoproteins share 99% identity. There are 26 nt changes across the whole genome (1 in S, 8 in M, 17 in L), but only 7 of these translate into an amino acid change (3 in the Gn/Gc polyprotein, 4 in the polymerase protein). Predictive protein analysis indicates that at least 1 of the 3 aa changes occurs in the ectodomain of the Gn protein, which could affect virus–host interactions. Of the remaining changes, 14 are silent mutations and 5 occur in noncoding regions.

In light of the genomic similarity of these 2 viruses, we suggest that the species name Albatross Island virus encompass both isolates, ABIV and HIGV, thereby representing the name of the original 1983 isolate and the location

Table. Virus neutralization assay results for Albatross Island virus, Australia

Source (species)	Location/year sample collected	No. positive/no. tested
Magpie goose (*Anseranas semipalmata*)	Northern Territory/1974	0/2
Masked lapwing (*Vanellus miles*)	Northern Territory/1975	0/1
Little corella (*Cacatua sanguinea*)	Northern Territory/1975	0/1
Whimbrel (*Numenius phaeopus*)	Northern Territory/1975	0/8
Cattle egret (*Ardea ibis*)	Gatton, Queensland/1981	0/5
Shy albatross (*Thalassarche cauta*)	Albatross Island/1983	18/36
Black noddy (*Anous minutus*)	Heron Island, Queensland /1985	1/115
Wedge-tailed shearwater (*Ardenna pacifica*)	Heron Island, Queensland /1985	0/46
Domestic chickens (*Gallus gallus*)	Armidale, New South Wales/1979	0/10
Little red flying fox (*Pteropus scapulatus*)	Unknown/unknown	0/5
Seals (unknown)	Macquarie Island/unknown	0/35
Maruspials (various wallabies, kangaroos, possums)	Queensland /Northern Territory/unknown	0/97
Cattle (*Bos taurus*)	Queensland /Northern Territory/1974–1985	0/160
Rusa deer (*Rusa timorensis*)	SE Queensland/1984	0/30

where both viruses were isolated. These 2 viruses are closely related to 2 tickborne phleboviruses: severe fever with thrombocytopenia syndrome virus, isolated in China (*2*), and Heartland virus, isolated in the United States (*3*). Each of these recently emerged viruses causes severe febrile illness with thrombocytopenia; deaths have been reported from 4 countries. In addition, Malsoor virus (*4*), a phlebovirus recently isolated from bats in India, has been shown to be closely related to severe fever with thrombocytopenia syndrome virus and Heartland virus. At the protein level, the similarity of ABIV to these 3 viruses is as follows: L, 66%–67%; M, 52%–56%; S, 58%–62%.

Deaths of albatross chicks in the Albatross Island colony occur every year; the intensity of these events varies from year to year (*5*). The cause of these events is multifaceted, but fowlpox is believed to be a major factor (*6*). No tests exist to quantify the extent and cause of the problem, although solutions are being pursued (R. Alderman, pers. comm, 2015). Wang et al. were unable to confirm infection of albatross with the HIGV isolate (*1*); however, the results presented here suggest that ABIV does infect albatross. Although infection is not direct evidence of disease, the fact that both isolates were collected from the same albatross colony during disease events almost 2 decades apart should not be neglected. Viral challenge studies would be useful for determining if and how ABIV contributes to disease in these birds.

Birds of the albatross family tend to fly long distances over open water. The geographic range of shy albatross extends from their breeding base in Tasmania to southern Africa (*5*). White-capped albatross (*T. steadi*) reportedly migrate from their breeding base in New Zealand as far as South America and eastward into shy albatross territory (*7*). It is possible to misidentify 1 of these albatross species as the other; indeed, the phylogenetic distinction between these species, once considered the same (*Diomedea cauta*), is controversial. The ease of albatross movement between vast geographic areas could provide an opportunity for intercontinental spread of emerging infectious diseases. Consequently, phleboviruses similar to ABIV may be present in bird populations in the southern areas of Africa and South America.

The need for intensified international investigations to identify genetically related tickborne phleboviruses with zoonotic potential is evident. The opportunity for the distribution of such viruses over a large global area is of concern to public health. Surveillance and investigation on an international level are needed.

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Toxoplasma gondii in Wild Red Squirrels, the Netherlands, 2014

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To the Editor: *Toxoplasma gondii*, a zoonotic protozoan parasite for which felids are the only definitive hosts, can infect humans and other warm-blooded animals. Transmission usually occurs orally from oocysts shed by felids in water and on food, through tissue cysts in undercooked meat, or transplacentally. In particular, young cats shed oocysts that can sporulate and become infectious within a day, depending on temperature and humidity. Sporulated oocysts can survive in moist soil for months to years (*1*).

In September 2014, the number of dead squirrels reported to the Dutch Wildlife Health Centre and the Dutch Mammal Society increased suddenly. The red squirrel (*Sciurus vulgaris*) is the only species of squirrel endemic to the Netherlands. Members of the public claimed that squirrels were "dropping dead from trees." Subsequently, the public was encouraged to report and submit dead squirrels. A total of 187 animals were reported through October 2014, of which 37 were submitted for necropsy. Necropsy included macroscopic examination; cytologic analysis of liver, spleen, lungs, and intestinal contents stained with hemacolor (Merck, Darmstadt, Germany); and histologic examination of samples of various organs fixed in formalin, embedded in paraffin, cut into 4-μm sections, and stained with hematoxylin and eosin.

For 8 adult animals, body condition (based on degree of fat storage and muscle development) was good; 12 juveniles were in poor condition. Typically, the trachea contained foam, and lungs were hyperemic and edematous. The liver was enlarged and pale, and the spleen was enlarged. In 13 animals, numerous small crescent-shaped organisms, with eccentrically placed nuclei consistent with tachyzoites of *T. gondii*, were identified by cytology in lung, liver, and spleen (*2*). Main histopathologic findings were pulmonary interstitial lymphoplasmocytic and neutrophilic infiltrates with edema and numerous intra-alveolar macrophages (17/20) and multifocal lymphoplasmocytic infiltrates with necrosis in the liver (13/20). Extensive splenic necrosis was occasionally observed (4/20). Intestines contained mild plasmacytic infiltrates. Numerous tachyzoites consistent with *T. gondii* were present in alveolar macrophages and epithelial cells, splenic macrophages, and hepatocytes. Duplicate slides were stained immunohistochemically by using polyclonal antibodies against *T. gondii* following a standard ABC protocol (*3*). Organisms stained for *T. gondii* in liver, spleen, lungs, and intestine. *Toxoplasma* was not detected in any brain. DNA was isolated (DNeasy Blood and Tissue Kit; QIAGEN, Hilden, Germany) from tissues of 14 squirrels and tested by quantitative PCR (*1*); *T. gondii* DNA was detected in 13. We successfully sequenced the *T. gondii* GRA6 gene for 11 squirrels and identified sequences to clonal type II *T. gondii* previously identified in sheep from the Netherlands (GenBank accession no. GU325790) (*4*). Incidental findings in the animals tested were encephalitis (2/20), coccidiosis (5/20), trauma (6/20), myocarditis (4/20), nephritis (1/20), lymphadenitis (1/20), and intestinal (3/20) and external (5/20) parasites.

The remaining 17 animals showed ≥1 of the following pathologic conditions: hemorrhages consistent with trauma (12/17), mild to severe intestinal coccidiosis (12/17), pneumonia (3/17), splenitis (1/17), *Taenia martis* cysticerci (1/17), and external parasites (8/17). Immunohistochemistry results for all 17 were negative for *T. gondii*.

On the basis of necropsy and molecular findings, we conclude that 20 of 37 examined squirrels died of disseminated *T. gondii* type II infection. These animals included adults and juveniles and were not restricted to specific geographic areas (Figure). The remaining animals died of trauma (12/17) or other causes (5/17).

Red squirrels are susceptible to *T. gondii*, and infection can lead to death. However, in our sample, the proportion of squirrels that died of toxoplasmosis (>50%) was higher than in other studies (≈16%) (*5*–*7*). The apparent increase in squirrel deaths and unexpectedly high proportion of fatal *T. gondii* infections suggests a toxoplasmosis outbreak among red squirrels. Possible explanations for this surge in cases include increased exposure to the parasite, increased susceptibility to infection, or increased virulence of the pathogen. Clonal *T. gondii* type II, the strain most frequently involved in human cases and endemic to Europe and North America, was identified. An increased virulence of the pathogen could not be proven (*8*). On the basis of lymphoid hyperplasia in