viruses during the period of study, leading to co-circulation of variants. The frequency of circulating HA genetic groups was quite variable, with rapidly changing patterns of predominance. Evidence of reassortment events was observed which could be responsible for the rise and predominance of some clades, and might predict the emergence of other variants.

A30 Avian influenza viruses in wild birds: Virus evolution in a multi-host ecosystem

Divya Venkatesh,[1] Marjolein J. Poen,[2] Theo M. Bestebroer,[2] Rachel D. Scheuer,[2] Oanh Vuong,[2] Mzia Chkhaidze,[3] Anna Machablishvili,[3] Jimsher Mamuchadze,[4] Levan Ninua,[4] Nadia B. Fedorova,[5] Rebecca A. Halpin,[5] Xudong Lin,[5] Amy Ransier,[5] Timothy B Stockwell,[5] David E. Wentworth,[5] Divya Kriti,[6] Jayeeta Dutta,[6] Harm van Bakel,[6] Anita Puranik,[7] Marek J. Slomka,[7] Steve Essen,[7] Ian H. Brown,[7] Ron A. M. Fouchier,[2] and Nicola S. Lewis[1,7]

[1]Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, UK, [2]Department of Viroscience, Erasmus MC, P.O. Box 2040, 3000 CA Rotterdam, The Netherlands, [3]National Centre for Disease Control, Tbilisi, Georgia, [4]Institute of Ecology, Ilia State University, 3/5 Cholokashvili, Tbilisi, Georgia, [5]J. Craig Venter Institute, Rockville, MD, USA, [6]Icahn School of Medicine at Mount Sinai, New York, NY, USA and [7]Animal and Plant Health Agency-Weybridge, UK

Wild ducks and gulls are the major reservoirs for avian influenza A viruses (AIVs). The mechanisms that drive AIV evolution are complex at sites where various duck and gull species from multiple flyways breed, winter, or stage. The Republic of Georgia is located at the intersection of three migratory flyways: the Central Asian Flyway, East Asian/East African Flyway, and Black Sea/Mediterranean Flyway. For six consecutive years (2010–6), we collected AIV samples from various duck and gull species that breed, migrate, and overwinter in Georgia. We found substantial subtype diversity of viruses that varied in prevalence from year to year. Low pathogenic (LP)AIV subtypes included H1N1, H2N3, H2N5, H2N7, H3N8, H4N2, H6N2, H7N3, H7N7, H9N1, H9N3, H10N4, H10N7, H11N1, H13N2, H13N6, H13N8, and H16N3, plus two H5N5 and H5N8 highly pathogenic (HP)AIVs belonging to clade 2.3.4.4. Whole-genome phylogenetic trees showed significant host species lineage restriction for nearly all gene segments and significant differences for LPAIVs among different host species in observed reassortment rates, as defined by quantification of phylogenetic incongruence, and in nucleotide diversity. Hemagglutinin clade 2.3.4.4 H5N8 viruses, circulated in Eurasia during 2014–5 did not reassort, but analysis after its subsequent dissemination during 2016–7 revealed reassortment in all gene segments except NP and NS. Some virus lineages appeared to be unrelated to AIVs in wild bird populations in other regions with maintenance of local AIV viruses in Georgia, whereas other lineages showed considerable genetic inter-relationship with viruses circulating in other parts of Eurasia and Africa, despite relative under-sampling in the area.

A31 Diversity change of influenza A (H3N2) strains circulating in Brazil during 2017–8: What to expect in the coming winter?

P. C. Resende, B. Caetano, A. Matos, J. Lopes, C. Garcia, M. Miranda, P. Born, F. C. Motta, D. Brown, and M. Siqueira

Laboratory of Respiratory Viruses and Measles, National Influenza Centre, MoH and WHO – Oswaldo Cruz Institute – FIOCRUZ, Rio de Janeiro, Brazil

The H3N2 subtype of influenza A (H3N2) was the predominant strain during the early months of the 2017 influenza epidemic in Brazil. In Australia, it was responsible for a strong and prolonged 2017 season and reached the Northern hemisphere causing an intense 2017/8 influenza season. Several genetic and antigenic A(H3N2) variants were circulating, which made the decision about which strain to incorporate into the influenza vaccine challenging. For 2018, the WHO selected a new H3N2 strain, A/Singapore/INFIMH-16-0019/2016-like, to replace the strain A/HongKong/4801/2014-like in the Southern Hemisphere trivalent vaccine. The aim of this study was to describe the genetic diversity of influenza A (H3N2) viruses circulating in Brazil between January 2017 and January 2018, checking the match between the vaccines and worldwide circulating strains with the Brazilian influenza strains. Hemagglutinin gene sequencing of the influenza A (H3N2) was performed, followed by a phylogenetic reconstruction using additional database sequences to define genetic groups and compare with other worldwide circulating strains. We observed a large diversity of H3N2 genetic clusters, including 3C.2a, 3C.2a1, 3C.3a, and their subgroups. During the 2016–7, inter-epidemic and 2017 epidemic period the cluster most frequently detected belonged to clade 3C.2a1 (148/185; 80.0%), a distinct group related to the 2017 vaccine strain A/HongKong/4801/2014-like (3C.2a). However, the genetic profile changed during the study period and in the inter-epidemic season 2017–8 the most commonly detected genetic group was the 3C.2a cluster (43/58; 74.1%). Inside this cluster, the majority (34/43; 79.1%) of strains belonged to a single genetic 3C.2a subgroup 2 (3C.2a2), bearing antigenic substitutions T131K and R142K (site A) and R261Q (site E). The dominance of this 3C.2a2 in the 2017–8 inter-epidemic period in Brazil was similar to the 2017–8 season in Europe and Canada according their surveillance data. The new vaccine strain has five to six antigenic changes in comparison to the predominant 3C.2a2 circulating in South America since September 2017 until now. It is possible that the vaccine mismatch will not protect the population against a majority of circulating strains. Surveillance of the vaccine effectiveness supported by antigenic and serological analysis are necessary to prove this hypothesis. However, this highlights the difficulty of vaccine strain selection and highlights the need for of a universal influenza vaccine.

A32 Genomic surveillance of Zika virus transmission in the Amazonas State, Brazil

Marta Giovanetti,[1,2] Jaqueline Goes de Jesus,[1] Quick Joshua,[3] Ingra Morales Claro,[4] Joilson Xavier Junior,[1] Sarah C. Hill,[5] Souza Renato,[6] Paola Paz,[6] Jose Lourenco,[5] Simon Dellicour,[7] Esther Sabino,[4] Nicholas J. Loman,[3] Felipe Naveca,[8] Oliver G. Pybus,[5] Nuno Rodrigues Faria,[5] and Luiz Carlos Junior Alcantara[1,2]

[1]Laboratorio de Patologia Experimental Instituto Gonçalo Moniz Salvador, Bahia, Brazil, [2]Laboratório de Flavivírus, Instituto Oswaldo Cruz Fiocruz, Rio de Janeiro, Brazil, [3]Institute of Microbiology and Infection, University of Birmingham, Birmingham, UK, [4]Instituto de Medicina Tropical, Universidade de Sao Paulo, Sao Paulo, Brazil, [5]Department of Zoology, University of Oxford, Oxford OX1 3PS, UK, [6]Laboratory for Clinical and Epidemiological Virology, Institut Pasteur, Universidade Federal do Rio de Janeiro, Rio de Janeiro, Brazil, [7]Department of Microbiology and Immunology, Rega Institute, KU Leuven - University of Leuven, Leuven, Belgium and [8]Fundação Oswaldo Cruz, Fiocruz, Amazonas, Brazil

Zika virus (ZIKV) has caused an unprecedented epidemic linked to severe congenital syndromes. Transmission of ZIKV in the Americas was first confirmed in May 2015 in northeast Brazil, though the virus was likely introduced 1–2 years prior to its detection. Manaus, the capital city of the Amazonas State, the largest territory of any state in Brazil and the main economic center in the northern region, reported between 2016 and 2017 more than 2,327 suspected cases of ZIKV infection. To gain insights into the timing, source, and likely route(s) of ZIKV introduction in the Amazonas State, we tracked the virus by sequencing ZIKV genomes from infected patients. Using nanopore sequencing technology, we generated 56 Brazilian ZIKV genomes from Manaus city in the Amazonas state, sampled from human cases. On the basis of available sequences of isolates from the Americas, the Manaus sequences, we analyzed fell within a single strongly supported monophyletic clade (bootstrap support = 99%, posterior support = 1.00) that belongs to the Asian genotype. Genetic analysis suggests the outbreak most likely originated from transmission cycles not previously identified in North Brazil and not from a separate introduction into the Americas. Molecular dating analysis indicates that the outbreak was caused by a single founder strain that is estimated to have arrived in Manaus around February 2015. By analyzing surveillance and genetic data, we discovered that ZIKV moved among transmission zones in Manaus. Geographical analysis further indicates that the Northern part of the Manaus regions has a high transmission potential for ZIKV. Our work illustrates that near-real time genomics in the field can augment traditional approaches to infectious disease surveillance and control. Estimated dates for the international spread of ZIKV from the north region indicate the persistence of the virus transmission in recipient regions. Our study provides an understanding of how ZIKV initiates transmission in new regions.