full-genome sequences were obtained, originating from four different islands and dating from the start of the outbreak (December 2013) to April 2015, when the outbreak was waning. High similarity (>99%) between sequences was found; nevertheless, all genome sequences were unique with a minimum of three SNPs differentiating one sequence from another. Thirty-three unique single nucleotide polymorphisms (SNPs) were identified, of which 29 were located in the coding regions of the genome. Eight SNPs were informative, and ten SNPs led to amino acid changes. Of the amino acid changes, nine were located in the non-structural proteins (1× nsP1, 5× nsP2, and 3× nsP3), and one was located in E2. In conclusion, we report the first whole-genome sequences of CHIKV isolates from the 2013 to 2015 outbreak that originated from the Dutch Caribbean islands. Sequencing of the remaining samples is still in progress.

A37 Transmission success of dengue virus type 1 lineages in a dynamic virus population: An evolutionary view

Carmen Koo,[1] Helen Xu,[1] Jayanthi Rajarethinam,[1] Yee Ling Lai,[1] Lee-Ching Ng,[1,2] and Hapuarachchige Chanditha Hapuarachchi[1]

[1]Environmental Health Institute, National Environment Agency, 11, Biopolis Way, #06-05-08, Singapore 138667, Singapore and [2]School of Biological Sciences, Nanyang Technological University, Singapore, Singapore

Arbovirus transmission involves an interplay between host, virus, and environmental factors. Because of the complexity of interactions, the transmission success of arboviruses could either be a function of viral fitness or be stochastic. In the present study, using 1,963 envelope (E) gene sequences and 239 whole genomes, we conducted a large-scale molecular epidemiological analysis of a dengue virus type 1 (DENV-1) population to understand the transmission success, evolution, and dispersal patterns of different lineages of DENV-1 circulating in Singapore from 2011 to 2016. The study population was highly dynamic and heterogeneous. However, only a handful of genetically distinct strains ($n = 6$) established sustained transmission, but at variable levels of dominance. Phylogeographic analysis revealed a weak spatial clustering and 35 well-supported diffusion pathways, implying widespread and complex dispersal of these strains in local settings. Yet, the dominant strains were neither evolving faster than less dominant ones nor under positive selection. These observations suggested that lineage dominance was likely to be stochastic and opportunistically driven by non-viral factors such as host immune pressure and vector abundance. Our findings, therefore, emphasize the implications of understanding the vector and human factors in parallel to virus dynamics on continuing efforts to control the arbovirus disease transmission in endemic regions.

A38 Genomic epidemiology quantifies gaps in Aedes-borne virus transmission in the Americas

Darlan da Silva Cândido,[1,2] Oliver G. Pybus,[2] and Nuno Rodrigues Faria[2]

[1]Radcliffe Department of Medicine, University of Oxford, Oxford, UK and [2]Department of Zoology, University of Oxford, Oxford OX1 3SY, UK

The rapid spread and severity of pathogens, such as Zika (ZIKV) and Chikungunya (CHIKV) viruses in the Americas, demonstrate the need for a better understanding of when and where outbreaks emerge. Sequence evolution of these viral pathogens occurs simultaneously with geographic spread, which allows phylodynamic processes to be recovered from genomic data. Here, we used time-calibrated phylogeographic analyses implemented in a Bayesian phylogenetic framework to characterize the date of introduction of ZIKV, CHIKV, dengue, and yellow fever viruses in different geographic regions of the Americas. To estimate 'surveillance gaps', we compared the estimated dates of introduction of these pathogens to the first confirmations of virus circulation in the region. Datasets included all publicly available geo-referenced and time-stamped genetic data from the Americas. A series of environmental and ecological covariates will be tested to infer what factors are associated with the delayed detection of arbovirus transmission in each geographic region. These results will provide important information on where to concentrate surveillance strengthening measures in order to prevent future mosquito-borne virus epidemics.

A39 Reconstruction of Ebola chains of transmission using sequence and epidemiological data

A. Robert,[1] J. Edmunds,[1] R. Eggo,[1] A.-M. Henao-Restrepo,[2] P.-S. Gsell,[2] C. H. Watson,[1] I. M. Longini,[3] A. Rambaut,[4,5] A. Camacho,[1,6] and S. Hué[1]

[1]Department of Infectious Disease Epidemiology, Centre for the Mathematical Modelling of Infectious Diseases, London School of Hygiene & Tropical Medicine, London, UK, [2]World Health Organization, Geneva, Switzerland, [3]Department of Biostatistics, University of Florida, Gainesville, FL, USA, [4]Institute of Evolutionary Biology, University of Edinburgh, Edinburgh, UK, [5]Ashworth Laboratories, Centre for Immunology, Infection and Evolution, University of Edinburgh, King's Buildings, Edinburgh EH9 3JT, UK and [6]Epicentre, Paris, France

Transmission trees can be established through detailed contact histories, statistical inference, phylogenetic inference, or a combination of methods. Each method has its limitations, and the extent to which they succeed in revealing a 'true' transmission history remains unclear. Moreover, the net value of pathogen sequencing in transmission tree reconstruction is yet to be assessed. We explored the accuracy and sensitivity to biases of a range of methods for transmission chain inference. We studied eight transmission chains determined by contact tracing, each one having more than a third of its cases sequenced (87 samples over 199 cases in total). We compared three inference methods on the selected transmission chains: (i) phylogenetic inference: the Ebola virus (EBOV) sequences derived from patients were mapped onto a dated EBOV phylogeny tree including 398 EBOV sequences sampled in Guinea between March 2014 and October 2015; (ii) statistical inference: we used the maximum likelihood framework developed by Wallinga and Teunis to infer the most likely transmitter-recipient relationships from the onset dates; (iii) combined method: we inferred probabilistic transmission events using both pathogen sequences and collection dates with the R package Outbreaker2. The cases coming from each transmission chain were mostly clustered together in the phylogenetic tree. The few misclassified cases were most likely allocated to the wrong chains of transmission because of the timing of their symptom onsets. Probabilistic transmission tree using only onset dates broadly matched the contact tracing data, but multiple potential infectors were identified for each case. The combined method showed that an *a priori* knowledge of the number of independent imports had an important impact on the outcome. Although cases were allocated to the correct transmission chains, discrepancies were found in identifying direct case linkage and transmission generations within a chain. Phylogenetic, epidemiological, and combined approaches for transmission chain reconstructions globally concurred in their output. Sequence data proved useful (if not necessary) to place the sampled cases in a wider context, identify transmission clusters, and misclassified cases when epidemiological chains are inferred from date of symptom onset only, and to identify links between supposedly independent chains of transmission.

A40 Estimation of Lassa virus emergence in Upper Guinea through a time-calibrated phylogeny

Elisabeth Fichet-Calvet,[1] N'Faly Magassouba,[2] and Stephan Günther[1]

[1]Bernhard-Nocht Institute of Tropical Medicine, Hamburg, Germany and [2]Laboratoire des Fièvres Hémorragiques Virales, Conakry, Guinea

Lassa fever is a hemorrhagic fever caused by an arenavirus, the Lassa virus (LASV), and can affect 150–200,000 persons per year in West Africa. The virus is hosted by several rodents, *Mastomys natalensis* and *M. erythroleucus*, *Hylomyscus pamfi*, and *Mus baoulei*. People can be contaminated at home or in the farms, by touching contaminated surfaces, eating contaminated food, or breathing aerosolized viral particles. Human-to-human transmission is occurring as well through infected bodily fluids. In Upper Guinea in particular, *M. natalensis* is the main host, with LASV prevalence of 14 per cent and IgG prevalence of 27 per cent. In humans, IgG prevalence is 40 per cent. This is, therefore, a hot spot for LASV transmission. In a previous phylogenetic study including 132 partial nucleoprotein (NP) sequences isolated from rodents, we showed that LASV could have emerged 90 years ago in the area. Here, we aim to revise the time of emergence upon analyzing the complete NP and polymerase genes of two strains coming from Upper Guinea: 'Bantou 366', a strain isolated from *M. natalensis* in