

high-throughput sequencing to observe how SAMHD1 expression alters the mutational profile (frequency and spectra) of integrated proviruses. We will explore how mutation rates of HIV-2 can be manipulated through the use of nucleoside analogs and RNRI drugs to explore what effects these compounds have on the HIV-2 mutation profile. Using single-cycle infectivity assays as well as long-term spreading experiments, we will be able to correlate mutagenesis with viral evolution and infectivity data to explore how sensitive these two viruses are to changes in viral mutation. This work will serve to understand how HIV-2 operates at a lower mutation frequency than HIV-1, elucidate the relationship between mutagenesis and infectivity for the two viruses, and provide insights into the contrasting phenotypes observed between the viruses.

A28 Phylogeographic analysis of HIV-2 ANRS CO5 cohort reveals new trends in HIV-2 epidemic patterns in West Africa

B. Visseaux,¹ M. Bertine,¹ A. Storto,¹ F. Damond,¹ F. Collin,² G. Chêne,² S. Matheron,³ C. Charpentier,¹ S. Hué,⁴ and D. Descamps,¹ the French ANRS CO5 HIV-2 Cohort

¹IAME, UMR 1137, Univ Paris Diderot, Sorbonne Paris Cité, INSERM; AP-HP, Hôpital Bichat, Virologie, Paris, France, ²Université Bordeaux, ISPED, Centre INSERM U897-Epidémiologie-Biostatistique, Bordeaux, France, ³IAME, UMR 1137, INSERM, Maladies Infectieuses et Tropicales, Univ Paris Diderot, Hôpital Bichat, Sorbonne Paris Cité, AP-HP, Paris, France and ⁴LSHTM, London, UK and Hôpital Bichat-Claude Bernard, AP-HP, Paris, France

The early spread of HIV-2 in Western Africa is imperfectly described for group B and the recently identified subtype A2. Recent HIV-2 epidemiological data are also scarce outside of Guinea-Bissau. The sequence database of the HIV-2 ANRS CO5-cohort, one of the largest to date, was used to explore the early migration patterns of these strains by phylodynamic's means. All publicly available (forty-nine and eight for A and B, respectively) and ANRS CO5-cohort (125 and 68 for A and B, respectively) pol sequences with available time of sampling and patient's country of birth were included. Bayesian phylogeographic reconstructions and effective population size estimations were performed under the best fitting combination of evolutionary, demographic, and molecular clock models using BEAST 1.8. The tree topology was assessed with maximum likelihood trees using RAxML 8.0.0. The estimated introduction of group A in humans was 1945 [95 per cent HPD: 1935–1953], as previously reported. Subtype A1, present in Senegal, Gambia, Guinea-Bissau, and Guinea, experienced an early diversification around 1946 [1936–1954] with two distinct early epidemics in Guinea-Bissau and Senegal. Subtype A2, present in Ivory Coast and Mali, experienced a latter diversification (1956 [1947–1963]) in Ivory Coast with two introduction events in Mali (1963 [1957–1969] and 1967 [1960–1974]). Group B was originally introduced in Ivory Coast in 1962 [1953–1913]. Changes in effective population size over time revealed initial exponential growth phases occurring sequentially for the three HIV-2 strains and followed by a population decline starting in the 2000s for all HIV-2 strains. The rate of this decline was slower for A2 and B subtypes (Ivory Coast, Mali) than for A1 (Guinea-Bissau, Senegal). This phylogeographic study is the first to reconstruct the early dispersal of A2 and B HIV-2 clades in Western Africa. Our results suggest that subtype A1 was circulating in Guinea-Bissau and Senegal before the independence war of the former, believed to have contributed to the dispersal of HIV-2. Both A2 and B clades emerged in Ivory Coast and experienced latter diversification and population expansion (starting in 1980 and 1990, respectively) than A1. There is indication of slow decreasing incidence rates of HIV-2 in Ivory Coast or Mali where recent data are scarce.

A29 Development of a full-genome sequencing platform to study norovirus diversity

C. J. Lepore,¹ K. Tohma,^{1,2} L. A. Ford-Siltz,¹ G. Sánchez,³ H. Mayta,³ R. H. Gilman,⁴ M. Saito,² and G. I. Parra¹

¹Division of Viral Products, Food and Drug Administration, Silver Spring, MD, USA, ²Department of Virology, Tohoku University School of Medicine, Sendai, Japan, ³Infectious Diseases Research Laboratory, Department of Cellular and Molecular Sciences, Universidad Peruana Cayetano Heredia, Lima, Peru and ⁴Department of International Health, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, MD, USA

Norovirus is a major cause of acute gastroenteritis worldwide. Noroviruses are very infectious and highly diverse, with two different genogroups (GI and GII) and almost thirty different genotypes infecting humans. Over the last two decades a single genotype (GII.4) has been shown to be the predominant cause of viral gastroenteritis outbreaks worldwide, therefore, most of the research was focused on this genotype. However, the epidemiological picture has changed during the last three seasons, where two different genotypes GII.17 (2013–2015) and GII.2 (2015–2016) have emerged as the major causes of gastroenteritis in different countries. Thus, a better understanding of the evolution of all different norovirus genotypes is needed for vaccine development. Much of current research on norovirus evolution has been focused on the major capsid protein (VP1), the major target for vaccine development. The VP1 is encoded by the open reading frame (ORF) 2, which constitutes only about one-fourth of the whole genome. However, much is unknown about the evolution, functional, and immunological roles of ORF1, a 5,000-nucleotide segment of the genome that encodes six nonstructural proteins. Because only 0.3 per cent of the norovirus sequences deposited in public databases correspond to full-length genomes, we developed an RT-PCR assay that amplifies the full-length genome of different norovirus genotypes; the resulting amplicons are sequenced using next-generation sequencing platforms. Using this platform, we successfully sequenced and assembled over fifty norovirus genomes from eleven different genotypes. The full-length sequences of two genotypes, GII.9 and GI.7, were obtained for the first time. Using neighbor-joining phylogenetic trees, we determined that the GII.9 presented an ORF1 region very similar to the one associated to GII.6, GII.7, and GII.14 noroviruses. On the contrary, sequences of GII.17 strains circulating pre-2013 clustered in different branches, for both ORF1 and ORF2 regions, than the ones circulating during 2013–2016. In addition, ORF1 sequences from different GII genotypes showed the clustering into at least two different groups. Together, this suggests independent evolution of the two different regions of the genome. We expect this method will encourage full-genome sequencing in the norovirus field, and create an improved database to expand our genomic analyses.

A30 Norovirus epidemiology and diversity in South Africa, 2009–2016

Janet Mans,¹ Victor V. Mabasa,¹ Tanya Y. Murray,¹ Sandrama Nadan,^{1,2} Johannes C. Botha,¹ Nicola A. Page,^{1,2} and Maureen B. Taylor¹

¹Department of Medical Virology, Faculty of Health Sciences, University of Pretoria, Pretoria, South Africa and ²Virology Division, Centre for Enteric Diseases, National Institute for Communicable Diseases, Sandringham, South Africa

Norovirus is a major cause of viral gastroenteritis in all age groups. The virus is classified in the Caliciviridae family of small, icosahedral viruses with a ~7.6-kb linear positive-sense RNA genome. The genome encodes three open reading frames