study for detecting and assembling complete HPV genomes in cervical samples from a cohort of young women attending cervical screening with access to HPV vaccination in Luxembourg. DNA extracts of eighty-one cervical swabs from women (mean age 23 years) positive for HPV by AnyplexIIHPV28® (Seegene) were enriched by rolling circle amplification and sequenced on Illumina Miseq. Reads were mapped to 182 PaVE reference sequences of known HPV types using BBMap and assembled using VELVET. Complete HPV genomes obtained were aligned with genomes published in Genbank using MEGA6. Overall, an average of 1 per cent of reads mapped to HPV. Among the eighty-one positive samples, NGS-RCA detected 186 different HPV types spanning thirty-six of the fifty-one known mucosal types. HPV types 42, 53, 51, 56, 90, and 31 were most frequently detected in twenty-two, fifteen, ten, ten, nine, and seven samples, respectively. Detection of HPV types by NGS-RCA was highly correlated with viral load of Anyplex. About sixty-seven consensus sequences of complete HPV genomes were assembled including two novel lineages of HPV66 and HPV90 and two novel sublineage of HPV67 and HPV73, respectively. NGS-RCA is a powerful method for obtaining complete HPV genomes from cervical samples with a high viral load ($Ct < 30$). After eight years of the vaccination programme in Luxembourg, vaccine-related types 6, 11, 16, and 18 were infrequently detected in the targeted age group.

A45 Merkel-cell polyomavirus and human polyomavirus 6 in Argentina, Uruguay, and Spain: Deep characterization of the South American types

C. Torres,[1,2] M. Barrios,[1] R. Cammarata,[1,2] M. Victoria,[3] X. Fernandez-Cassi,[4] S. Bofill-Mas,[4] R. Colina,[3] M. Blanco Fernández,[1,2] and V. Mbayed[1,2]

[1]Virology Chair, School of Pharmacy and Biochemistry, University of Buenos Aires, Buenos Aires, Argentina, [2]CONICET, Buenos Aires, Argentina, [3]University of the Republic, Salto, Uruguay and [4]University of Barcelona, Barcelona, Spain

New human polyomaviruses have been discovered in the last years, including the Merkel-cell (MCPyV) and the human polyomavirus 6 (HPyV6). Although their infection is usually asymptomatic, in immunocompromised hosts they can cause life-threatening pathologies. In particular, MCPyV has been associated with Merkel cell carcinoma, an aggressive skin cancer arising in the elderly and in chronically immunosuppressed individuals. Despite being prevalent viruses, epidemiological data from South America are scarce, as too are the viral types circulating and their origin. The aims of this work were to characterize MCPyV and HPyV6 from environmental samples with different geographical origins and to analyze the phylogenetic and phylogeographic profiles to study their spatio-temporal dispersion patterns, particularly for MCPyV. Partial and complete genome sequences were obtained from sewage samples from Argentina, Uruguay, and Spain. Phylogenetic analysis showed that MCPyV sequences distributed according to their geographic origin in Europe/North America, Africa, Asia, South America, and Oceania groups, suggesting that viral diversification might have followed human migrations across the globe. In fact, the analysis of the viruses reported here emphasized this behavior, given that they reflected the origin of the current population in each country. The South American group presented a high level of clustering, showing subgroups exclusively formed by sequences from southern South America, possibly associated with local diversification events related to early migratory movements in the region. Regarding HPyV6, sequences from South America grouped with high support and were separated from all other sequences available, from USA, France, Australia, and China. The analysis of viruses from the environment allowed us to characterize prevalent infections in different geographic regions, revealing that viruses circulating in each population reflected its origin and that there are specific lineages associated with South America.

A46 Partial genetic characterization of a Brazilian strain of yellow fever virus from an epizootic outbreak in 2009

A. L. P. Mosimann,[1] T. R. Jorge,[1] L. Noronha,[2] A. Maron,[3] and C. N. Duarte dos Santos[1]

[1]Laboratório de Virologia Molecular, Instituto Carlos Chagas, FIOCRUZ, Curitiba, PR, Brazil, [2]Pontifícia Universidade Católica do Paraná, Curitiba, Paraná, Brazil and [3]Secretaria de Saúde do Estado do Paraná, Brazil

Yellow fever is the prototype virus of the *Flavivirus* genus in the Flaviviridae family. Its genome consists of a single-stranded positive-sense RNA molecule of ~10 kb presenting a single open reading frame. It is translated into a polyprotein which is processed by viral and host proteases into three structural (C, prM, and E) and seven non-structural proteins (NS1, NS2A, NS2B, NS3, NS4A, NS4B e NS5). As an arbovirus it is maintained in nature through a cycle in which the virus alternates between vertebrate (human/non-human primates) and invertebrate hosts (*Haemagogus* sp., *Sabethes* sp., and *Aedes* sp.). The species involved in the sylvatic and urban transmission cycles differ and since 1942 no cases related to urban transmission have been reported in Brazil. During a series of epizootics caused by Yellow fever virus in Brazil between 2007 and 2009, a monkey (*Alouatta* sp.) was found dead (May 2009) in a sylvatic area in the State of Paraná (southern Brazil). Brain samples from this animal were used for virus isolation. RNA was extracted from the cell culture supernatant and used to amplify an 861-bp segment, comprising the coding sequence of the C and prM proteins (nucleotides 119–979, according to GenBank accession number U17066). Sequence analyses demonstrated that it is closely related with two isolates from Venezuela (99.5 per cent identity), one isolated in 2005 from a human case and the other isolated in 2007 from *Alouatta seniculus*. Phylogenetic analysis groups them all within South American genotype I. This sequence was also compared to other sequences available in GenBank that presented only partial coverage but 100 per cent nucleotide identity (nucleotides 641 to 981, according to GenBank accession number U17066) and were not included in the phylogenetic analysis. This data indicates that this new Brazilian isolate may be part of the same epizootic that affected monkeys in the state of São Paulo (2008) and the Missiones province (2008 and 2009) and stresses the importance of yellow fever surveillance through sentinel monkeys.

A47 Whole-genome analysis of rotaviruses isolated from humans and domestic animals in Uganda, 2012–2014 reveals possible anthroponosis and multiple rotavirus reasortment events between species

J. Bwogi,[1,2] K. C. Jere,[3,4] C. Karamagi,[2] D. K. Byarugaba,[5] P. Namuwulya,[1] U. Desselberger,[6] and M. Iturriza-Gomara[3]

[1]EPI Laboratory, Uganda Virus Research Institute, 51-59 Nakiwogo Road, P.O. Box 49, Entebbe, Uganda, [2]Department of Paediatrics and Child Health, College of Health Sciences, Makerere University, P.O. Box 7072, Kampala, Uganda, [3]Department of Clinical Infection, Microbiology and Immunology, Institute of Infection and Global Health, University of Liverpool, Liverpool, UK, [4]Malawi-Liverpool-Wellcome Trust Clinical Research Programme, Department of Medical Laboratory Sciences, University of Malawi, College of Medicine, Blantyre, Malawi, [5]Department of Microbiology, College of Veterinary Medicine and Biosecurity, Makerere University, P.O.

Box 7072, Kampala, Uganda and [6]Department of Medicine, University of Cambridge, Cambridge, UK

Rotaviruses of species A (RVA) are a common cause of diarrhea in children and the young of various other mammals worldwide. Interspecies transmission of RVA may lead to the emergency of novel RVA strains which may potentially affect rotavirus vaccine efficacy. The aim of this study was to investigate for possible interspecies transmission of RVAs in Uganda. Whole-genome sequencing of eighteen human (under-fives with diarrhea) and six animal (one bovine, one caprine, and four porcine) RVA strains identified in Uganda in the same geographical region, between 2012 and 2014 was undertaken using the Illumina HiSeq platform. RotaC version 2, a classification tool for RVAs was used to assign genotypes to all eleven genome segments of each isolate. Phylogenetic analysis was carried out using the maximum likelihood method in MEGA 6.06. Human RVA strains had either a Wa- or a DS-1-like genetic constellation. One human strain was a Wa-like mono-reassortant containing a DS-1-like VP2 gene of possible animal origin. In addition, three human RVA strains had one or two genes with possible zoonotic origin. All eleven genes of the bovine RVA strain were closely related to those of human RVAs. The caprine strain had a mixed genotype backbone, suggesting that it emerged from multiple re-assortment events involving different host species. Porcine RVA strains had mixed genotype backbones with possible multiple reassortant events with strains of human and bovine origin. Interspecies transmission of RVA strains occurred in this setting. RVA strains causing diarrhea in children are primarily transmitted from person to person. Rotavirus vaccination in children in Uganda will control rotavirus transmission. It is recommended to continue molecular surveillance of RVAs in humans and animals living in the same geographical region to understand the molecular epidemiology and evolution of RVAs in Uganda and other countries.

A48 Evolutionary history constrains adaptation in vesicular stomatitis virus

Andrea Gloria-Soria,[1] Sandra Mendiola,[2] Valerie J. Morley,[3] and Paul E. Turner[1]

[1]Ecology and Evolutionary Biology, Yale University, New Haven, CT, USA, [2]College of Veterinary Medicine, University of Georgia, Athens, GA, USA and [3]Department of Biology, Pennsylvania State University, University Park, PA, USA

It is unclear how evolutionary history affects the ability of a population to adapt to novel environmental conditions. To explore this question, we use vesicular stomatitis virus (VSV) populations either evolved at a constant temperature of 37 C, or with temperatures randomly changing between 29 C and 37 C. Fitness was subsequently measured at 29 C and 37 C and gains were detected in all constant treatment replicates but the random treatment showed no fitness changes. Consensus genome sequencing revealed that populations in the random treatment had accumulated more mutations than the populations in the constant treatment. In order to determine whether elevated genetic diversity in the randomly evolved populations could facilitate adaptation to a novel environment, we pooled all five replicates of the constant and the random treatments to generate two parental populations with distinct evolutionary histories. Five replicates of each group were then exposed to 40 C for forty generations. Populations derived from the random treatment evolved higher fitness than those derived from the constant treatment when grown at 40 C. The majority of the mutations observed evolved *de novo*, although some alleles that became fixed in the evolved populations were already present at low frequency in the ancestors. Two novel convergent mutations were found in the populations derived from the constant treatment ancestor, while there was no evidence of convergence in the populations derived from the random ancestor. These results suggest that a constant environment could constrain a population to a specific evolutionary pathway when confronted with a novel environment and prevent it from achieving maximum fitness.

A49 Phylogenetic evaluation of the Zika virus emergence in the Americas: 2015–2016

G. A. Santiago and J. L. Muñoz-Jordan

Division of Vector Borne Diseases, Centers for Disease Control and Prevention, National Centers for Emerging and Zoonotic Infectious Diseases, Dengue Branch, San Juan, Puerto Rico

The introduction of Zika virus (ZIKV) to the Americas caused an unprecedented epidemic with over half a million suspected cases in over forty-eight countries reported to the Pan-American Health Organization to date. Recent phylogenetic studies have proposed that the Asian genotype of ZIKV was introduced into the Americas causing the epidemic, and the most recent ancestor to the American strains originates from a French Polynesian strain circulating in the South Pacific. We evaluated the genetic diversity of ZIKV in the Americas at the population level during the epidemic period using 198 complete genome sequences (including 157 American strains and 41 Asian strains) obtained from GenBank. Our Bayesian maximum clade credibility phylogeny and molecular clock analyses on our dataset confirm that ZIKV was initially introduced into the Americas from the South Pacific but suggest emergence initiated in Haiti prior to Brazil. Analysis of the time of the most recent common ancestor (tMRCA) of the earliest American isolates, including Haiti and Brazil, estimates that this introduction occurred in 2013 (2.011, 4.467 years 95 per cent HPD). The estimated evolutionary rate of the American ZIKV strain compares with other flaviviruses transmitted in the region but on the slower end of the range with a rate of 4.64E-04 nucleotide substitutions per site per year. A preliminary sequence analysis within American isolates did not identify significant mutations or genomic patterns that differentiate viruses isolated from mosquitoes or from humans, or from viruses isolated from different human specimen types including serum, urine, semen, and saliva. Further analyses on sequences and more recent virus isolates will be conducted to provide a better understanding on the evolution and transmission dynamics during early, epidemic, and post-epidemic periods.

A50 Genotypic distribution of HHV-8 in aids individuals without and with Kaposi sarcoma

T. R. Tozetto-Mendoza,[1] K. Y. Ibrahim,[2] A. F. Tateno,[1,3] C. M. Oliveira,[1] L. M. Sumita,[1] M. C. Sanchez,[1] E. J. Luna,[1] L. C. Pierrotti,[1] J. F. Drexler,[3] P. H. Braz-Silva,[4] C. S. Pannuti,[1] and C. M. Romano[1]

[1]Laboratory of Virology, Institute of Tropical Medicine, Universidade de Sao Paulo, Brazil, [2]Department of Infectious Diseases, Clinics Hospital of the School of Medicine, Universidade de Sao Paulo, Brazil, [3]Institute of Virology, University of Bonn Medical Centre, Bonn, Germany Centre for Infection Research (DZIF), Partner Site, BonnCologne, Germany and [4]Department of Pathology, School of Dentistry, University of São Paulo, Brazil

AIDS-associated Kaposi's sarcoma (AIDS-KS) caused by human herpes virus 8 (HHV-8) is the most severe and resistant form of KS tumor. Our aim was to verify whether there is an association between HHV-8 variability and development of AIDS-KS in Brazil by comparing the HHV-8 variability between individuals without and with KS. Saliva samples and blood, when available,