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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 emergence 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 reassortment 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

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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

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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

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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,