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relationships between mice and retroviruses. In particular, we establish the presence of a more ancient ERV component in the murine genome, comprised of isolated, highly degraded insertions. These sequences evidence a transition in murine evolutionary history, beginning about one million years ago, wherein the ancient ERV families that have counterparts in humans and other large mammals were overtaken by a wave of newly acquired and/or transpositionally active ERVs.

**A15 Rapid radiation of treponema pallidum pertenue in wild non-human primates**

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Bacteria of the species Treponema pallidum are causative agents of venereal syphilis (Treponema pallidum pallidum), Bejel (T. pallidum endemicum), and yaws (T. p. pertenue) in humans. We documented Treponema pallidum infections associated with disease in wild sooty mangabeys (Cercocebus atys) in Tai National Park, Côte d’Ivoire, and green monkeys ( Chlorocebus sabaeus) from Bijilo Forest Park, Gambia and Niokolo-Koba National Park, Senegal. To examine the evolutionary relatedness of these treponemes to those responsible for diseases in humans and for previously documented infections in baboons (Papio papio), we conducted a hybridization capture experiment to enrich Treponema pallidum DNA from samples collected from symptomatic individuals. This approach allowed us to sequence the full genomes of Treponema pallidum strains infecting sooty mangabeys (n = 2) and green monkeys (n = 4). Phylogenomic analyses revealed that all Treponema pallidum strains infecting non-human primates are most closely related to the sub-species T. p. pertenue. Strains infecting humans and non-human primates do not appear to be reciprocally monophyletic. The star-like phylogenetic branching pattern of the T. p. pertenue clade, with short basal branches receiving low statistical support, suggests a rapid initial radiation across humans and non-human primates. These results greatly broaden the known host range of T.p. pertenue and suggest the existence of a vast zoonotic reservoir that could possibly contribute to the failure of global eradication efforts.

**A17 Molecular characteristics of hepatitis B virus (HBV) isolated from chronic hepatitis B patients in South Vietnam**

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Chronic infection with hepatitis B virus (HBV) is a major health problem due to its worldwide distribution and its long-term effects. Vietnam is country with a high HBV burden and the prevalence of chronic HBV infection in general population is 8.8–12.3%. In East Asia, the most common HBV genotypes are B and C. Despite high prevalence of HBV, data on HBV (genotype and subgenotype, virulence markers, drug resistance mutations and prevalence of recombinant strains) is limited in Vietnam. There are only few reports on HBV genotypes in Vietnam, mostly based on pre-S/S gene sequences. We have analyzed whole genome sequence of 98 HBV isolates from chronic HBV patients attending at Hospital for Tropical Disease, Ho Chi Minh City, Vietnam, who were under treatment for 1–6 years. HBV genome was amplified in 4 overlapping fragments (777–1,136bp) and the amplicons were subjected to deep sequencing by using Illumina MiSeq system. Sequence assembly, genome analysis and phylogenetic analysis were performed within Geneious package. A sequence was assigned to a certain genotype and subgenotype if it was contained within a well-supported phylogenetic cluster (bootstrap value >75%) and the intra-genotypic nucleotide divergence was < 7.5 and > 4.5%, respectively. Mutations in Basal core promoter (BCP), pre-CORE, and CORE gene regions were determined by comparing with reference sequences. Finally, screening of minor (sub-consensus) variants was performed using the SNP detection tool available in Geneious. 1% frequency and 500-fold coverage were chosen as cut-off values. Among the isolates, 71.43% were genotype B, 27.55% were genotype C and one isolate was a recombinant (between B and C). Among genotype B isolates, 65 were subgenotype B4 (92.86%) and 5 were B2 (7.14%). 92.6% of subgenotype C belong to C1, 3.7% is subgenotype C2 and the remaining 3.7% to C3. Mutations G1752A, T1753C, G1757A, A1762G/T, G1764A and C1766G on BCP and CORE were found in 76 of 98