Middle East Respiratory Syndrome Coronavirus (MERS-CoV) causing infections in humans is genetically indistinguishable from the virus found in Arabian camels (dromedaries) in the Middle East. Although no primary human case of MERS was reported outside the Arabian Peninsula, camel populations in Africa are known to have high prevalence of antibodies against MERS-CoV. We carried out surveillance for MERS-CoV in dromedaries in Africa and Central Asia. By MERS-CoV spike pseudoparticle neutralization assay we confirmed that camel serum samples from African countries have high prevalence of MERS-CoV antibodies. Using RT-qPCR we detected MERS-CoV positives in camel nasal swabs from all different African countries from which samples were collected. However, dromedary serum and swab samples from Kazakhstan in Central Asia were negative for MERS-CoV by these assays. Phylogenetic analysis of the spike gene revealed that MERS-CoVs from Africa formed a cluster closely related to but distinct from the viruses from the Arabian Peninsula. Results from this study suggest that MERS-CoV is actively circulating in dromedary populations in Africa and the virus in Africa is phylogenetically distinct from that in the Middle East.

**A47** Origin and possible genetic recombination of the middle east respiratory syndrome coronavirus from the first imported case in china: phylgenetics and coalescence analysis

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The Middle East respiratory syndrome coronavirus (MERS-CoV) causes a severe acute respiratory tract infection with a high fatality rate in humans. Coronaviruses are capable of infecting multiple species and can evolve rapidly through recombination events. Here, we report the complete genomic sequence analysis of a MERS-CoV strain imported to China from South Korea. The imported virus, provisionally named ChinaGD01, belongs to group 3 in clade B in the whole-genome phylogenetic tree and also has a similar tree topology structure in the open reading frame 1a and -b (ORF1ab) gene segment but clusters with group 5 of clade B in the whole-genome phylogenetic tree and support the notion that natural selection is favouring the maintenance of these structures, providing compelling evidence of their likely biological relevance. This structure-first strategy for comparative analysis of genome-wide secondary structures can be broadly applied to understand the contributions of higher-order genome structures to viral replication and pathogenicity.

**A44** Molecular evolutionary dynamics of respiratory syncytial virus group A in recurrent epidemics in coastal Kenya

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The characteristic recurrent epidemics of human respiratory syncytial virus (RSV) within communities may result from the genetic variability of the virus and associated evolutionary adaptation, reducing efficiency of pre-existing immune responses. We analyzed the molecular evolutionary changes in the attachment (G) glycoprotein of RSV-A viruses collected over 13 epidemic seasons (2000–12) in Kilifi (n = 649), Kenya, and contemporary sequences (n = 1,131) collected elsewhere within Kenya and 28 other countries. Genetic diversity in the G gene in Kilifi was dynamic both within and between epidemics, characterized by frequent new variant introductions and limited variant persistence between consecutive epidemics. Four RSV-A genotypes were detected in Kilifi: ON1 (11.9%), GA2 (75.5%), GA5 (12.3%), and GA3 (0.3%), with predominant genotype replacement of GA5 by GA2, then GA2 by ON1. Within these genotypes, there was considerable variation in potential N-glycosylation sites, with GA2 and ON1 viruses showing up to 15 different patterns involving eight possible sites. Further, we identified 15 positively selected and 34 genotype-distinguishing codon sites, with six of these sites exhibiting both characteristics. The mean substitution rate of the G ectodomain for the Kilifi dataset was estimated at 3.58 × 10⁻³ [95% HPD: 3.04–4.16] nucleotide substitutions/site/year. Kilifi viruses were interspersed in the global phylogenetic tree, clustering mostly with Kenyan and European