Simian hemorrhagic fever virus (SHFV) is an arterivirus that causes severe disease in captive macaques. We describe two new SHFV variants subclinically infecting wild African red-tailed guenons (Cercopithecus ascanius). Both variants are highly divergent from the prototype virus and variants infecting sympatric red colobus (Procolobus rufomitratus). All known SHFV variants are monophyletic and share three open reading frames not present in other arteriviruses. Our data suggest a need to modify the current arterivirus classification.
various. Across the aligned coding genomes, the viruses from individuals RT05 and RT11 were similar to each other at the nucleotide level (94.3%), as were the viruses from individuals RT09 and RT10 (93.7%). However, these two variants were only 79.4% similar to each other at the nucleotide level and less similar still to prototype variant LVR 42-0/M6941 (54.1%) or variants from Kibale red colobus (50.1%), with variable amino acid conservation across the genomes (Fig. 1).

Our Bayesian phylogeny is consistent with established relationships of arteriviruses (8, 20, 21), supporting the monophyly of SHFV variants, the sister taxon relationship of LDV and PRRSV, and the divergence of EAV (Fig. 2). Within the SHFV clade, the new variants are highly divergent from the prototype variant and from SHFV variants found in sympatric red colobus. Based on their phylogenetic positions (Fig. 2), we designate the new viruses SHFV-krtg1 and SHFV-krtg2, to indicate their origins in Kibale red-tailed guenons and to reflect nomenclature previously used to describe simian immunodeficiency virus (SIV) and SHFV variants infecting Kibale red colobus (8, 22).

In Kibale, red-tailed guenons frequently form multispecies social groups with red colobus, in which occasional direct contact occurs (23, 24). Nevertheless, the phylogenetic divergence between SHFV from red-tailed guenons and SHFV from sympatric red colobus is approximately equivalent to that between PRRSV and LDV, which are currently assigned to different viral species. This observation strongly suggests that virus-host co-evolution, rather than geography or ecological overlap, shapes the phylogeny of SHFV. Primates of the subfamilies Cercopithecinae and Colobinae diverged approximately 18 million years ago (25); cocirculating, divergent SHFV variants in both red-tailed guenons and red colobus may indicate ancient diversification of SHFV within sympatric host species or, possibly, more recent admixture of viruses due to transmission from other as-yet-unidentified hosts.

The unique 3’ genomic architecture of SHFV is conserved even across highly divergent SHFV variants. Furthermore, all SHFV variants described to date are monophyletic, even though SHFV ORFs 2a, 2b, and 3 have no homologs in the other arteriviruses and could not be included in our phylogenetic analyses of the Arteriviridae. Currently, the family Arteriviridae includes a single genus, Arterivirus (26). Given the unique and characteristic genomic architecture of all SHFV variants described to date, the monophyly of SHFV within the family Arteriviridae, and the association of SHFV with simian hosts, we suggest the reclassification of SHFV into a new genus: Simartevirus. The discovery of additional arteriviruses will help clarify the appropriateness of this proposed taxonomy, as well as any other taxonomic subdivisions within the family Arteriviridae that may be justified.

**FIG 1** (A) Genome organization of novel simian hemorrhagic fever viruses (SHFV) from Ugandan red-tailed guenons. The novel variants SHFV-krtg1 and SHFV-krtg2 are shown in comparison to the SHFV prototype variant LVR 42-0/M6941 and the recently discovered SHFV-krc1 and SHFV-krc2 variants from sympatric red colobus. Boxes represent open reading frames and are drawn to scale. (B) Sliding window similarity plots of percent amino acid identity among select SHFV variants across aligned coding genomes. The analysis was performed with a window size of 200 and a step size of 25. Dashed vertical lines indicate start positions of inferred viral proteins.
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