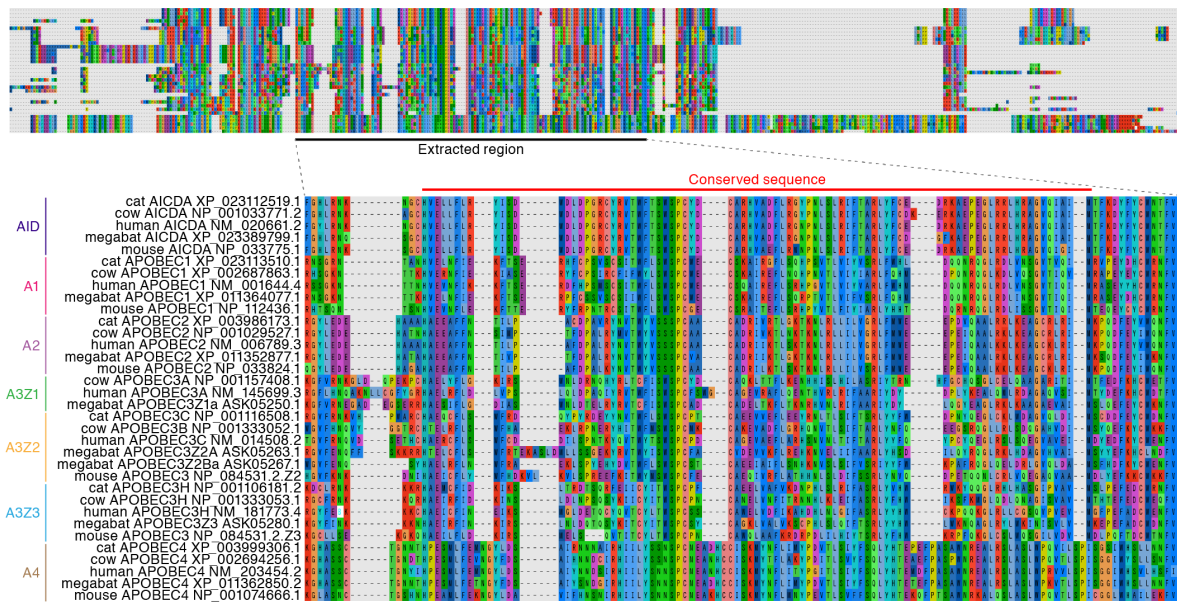


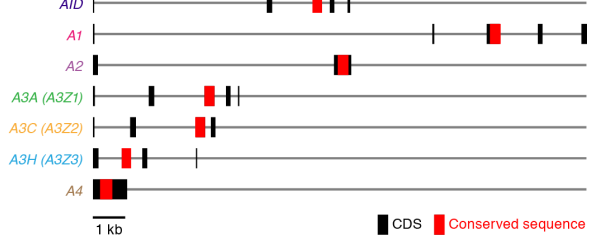
A



B



C



D

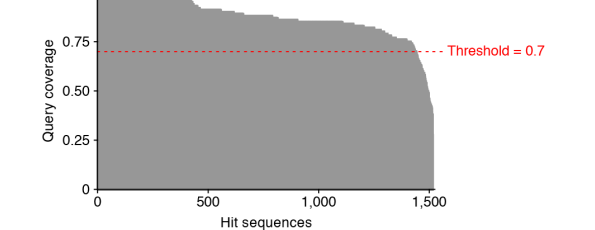


Fig. S1. Sequence conservation in *AID/APOBEC* family genes. (A) Amino acid sequences of *AID/APOBEC* family genes used as queries for *in silico* genome screening are shown. Sequences disclosing homology to the conserved sequences of *AID/APOBEC* family genes (“Conserved region”) were extracted. (B) Phylogenetic tree of *AID/APOBEC* family genes. The tree was constructed using maximum likelihood (ML). Bootstrap values are indicated at the nodes. (C) Gene structure of *AID/APOBEC* family genes in the human genome. Black and red boxes indicate regions corresponding to the coding domain sequence (CDS) and the conserved sequence, respectively. Note that the conserved sequence is located on a single exon in all types of *AID/APOBEC* family genes. (D) Coverage of sequence alignment among sequences recovered via *in silico* genome screening. Y-axis indicates the proportion of the length of the query sequence covered by the alignment (referred to as coverage). Sequences that covered < 70% of the conserved region were discarded.

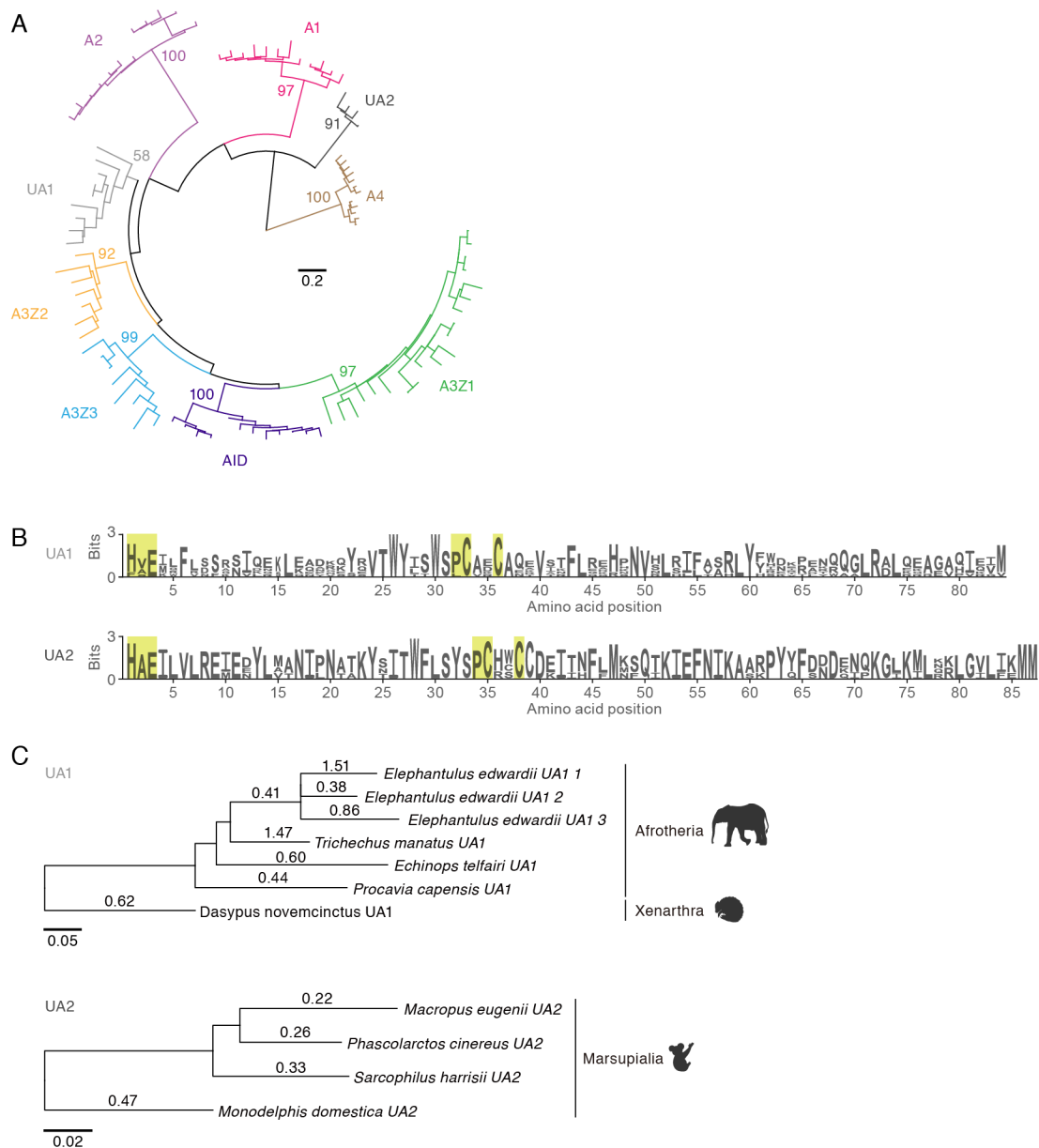


Fig. S2. Characterization of lineage-specific *AID/APOBEC* genes (*UA1* and *UA2*) found in basal eutherian mammals. (A) Phylogenetic tree of *AID/APOBEC* Z domains identified in Afrotheria, Xenarthra, and Marsupialia. The tree was reconstructed using a maximum likelihood (ML) approach, and is based on an alignment of nucleic acid sequences. Bootstrap values are indicated at the nodes. (B) Logo plots of amino acid sequences of *UA1* and *UA2*. Yellow squares indicate the amino acid residues corresponding to the catalytic domain of *AID/APOBEC* proteins. (C) Phylogenetic tree of *UA1* and *UA2* Z domains. The tree was reconstructed by maximum likelihood (ML) method based on the nucleic acid sequences. Estimated dN/dS ratios are indicated on the branches. Note purifying selection (dN/dS < 1) was detected in the Z domains of both genes.

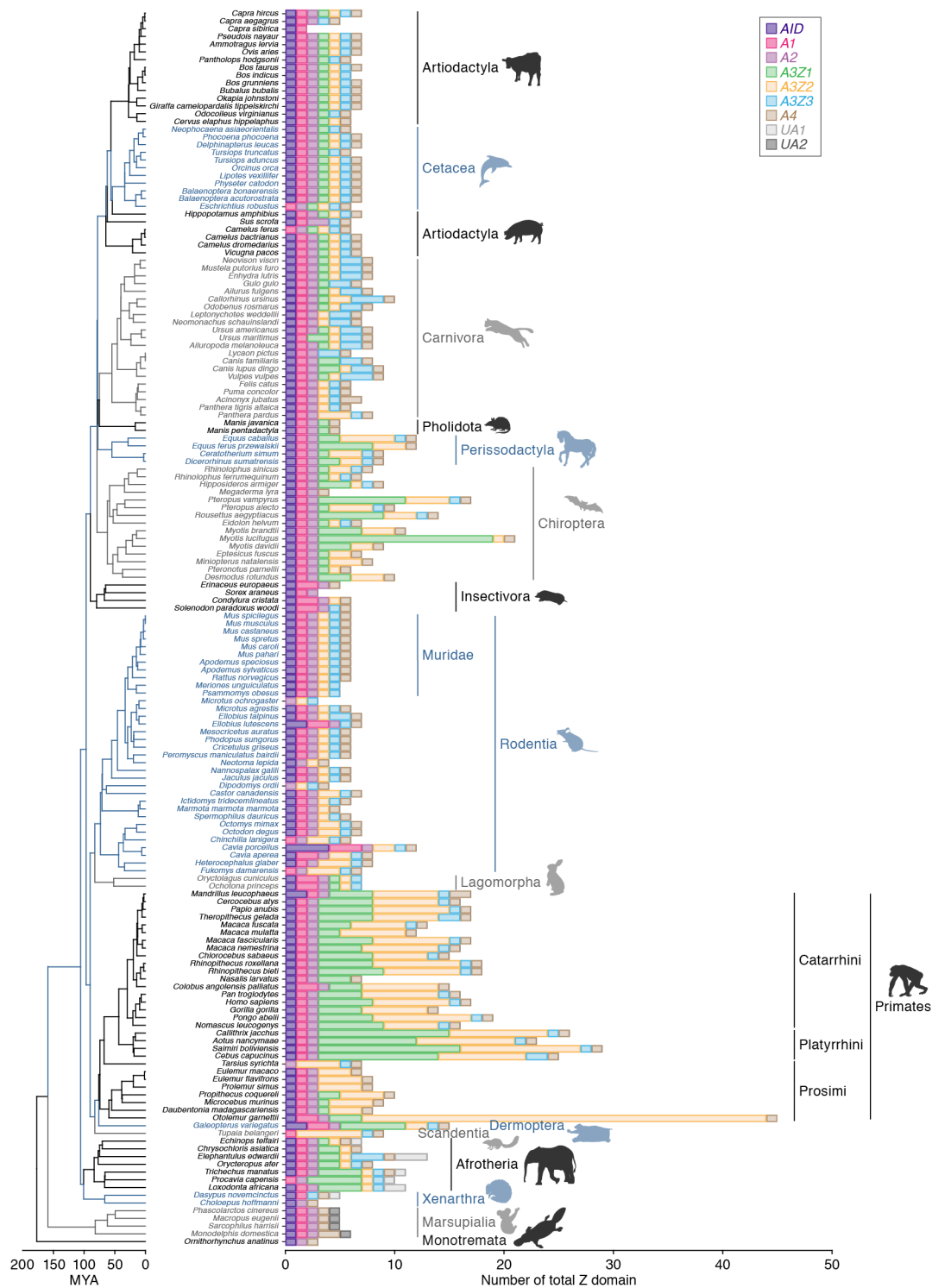


Fig. S3. Number of AID/APOBEC Z domains identified in each mammal species. Counts of both intact and pseudogenized AID/APOBEC Z domains are shown.

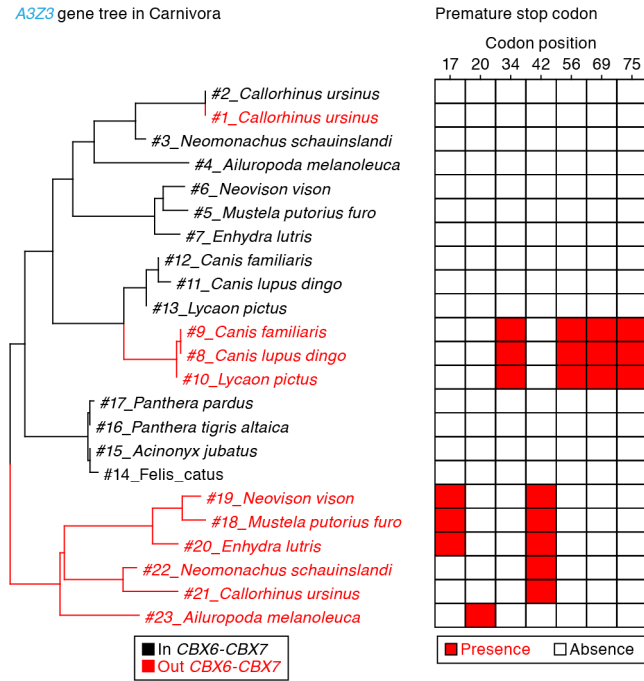


Fig. S4. Gene duplications of A3Z3 followed by pseudogenizations in Carnivora. (*Left*) Phylogenetic tree of A3Z3 in Carnivora. Red and black indicate A3Z3 located in the inside and outside of the canonical A3 gene locus. (*Right*) Presence of premature stop codons. Positions of the stop codons in the multiple sequence alignment are indicated. The phylogenetic relationships and shared patterns of premature stop codons indicate that fixation of duplicated A3Z3 pseudogenes has occurred at least twice in carnivore evolution.

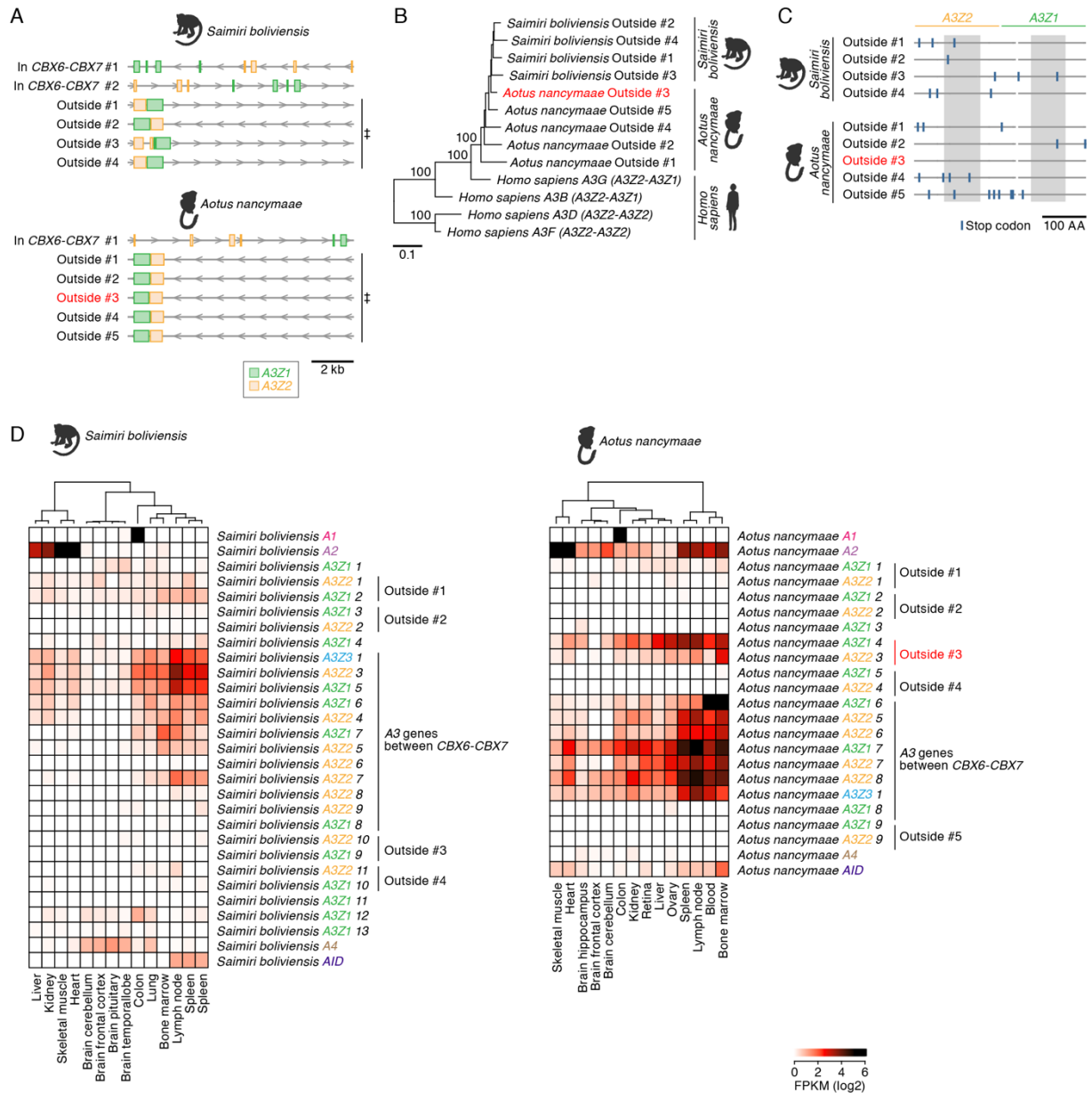


Fig. S5. Retrotransposition of A3G-like genes in New World monkeys. (A) Gene structures of A3Z2-A3Z1 type genes in the genomes of New World monkeys. Genes within and outside the canonical A3 gene locus (“in CBX6-CBX7”) are shown. Arrowheads indicate the direction of the respective loci. The sequences indicated by double daggers are intron-less sequences and correspond to those in Fig. 3C. (B) A phylogenetic tree of retrotransposed A3Z2-A3Z1 type genes identified in New World monkeys. Note that the retrotransposed genes form a cluster with the human A3G (A3Z2-A3Z1 type) gene. (C) Presence or absence of premature stop codons in the retrotransposed A3Z2-A3Z1 type genes in the two New World monkeys. Regions corresponding to the conserved sequence of AID/APOBEC Z domains are boxed. (D) RNA expression of the retrotransposed A3 genes in the tissues of New World monkeys. Rows indicate the sequences of AID/APOBEC family genes, and columns indicate the tissues of New World monkeys. Color indicates RNA expression level (log₂ FPKM). Note that the “outside #1-4” genes in *Saimiri boliviensis* and “outside #1-5” genes in *Aotus nancymaae* are identical to those in panels A-C.

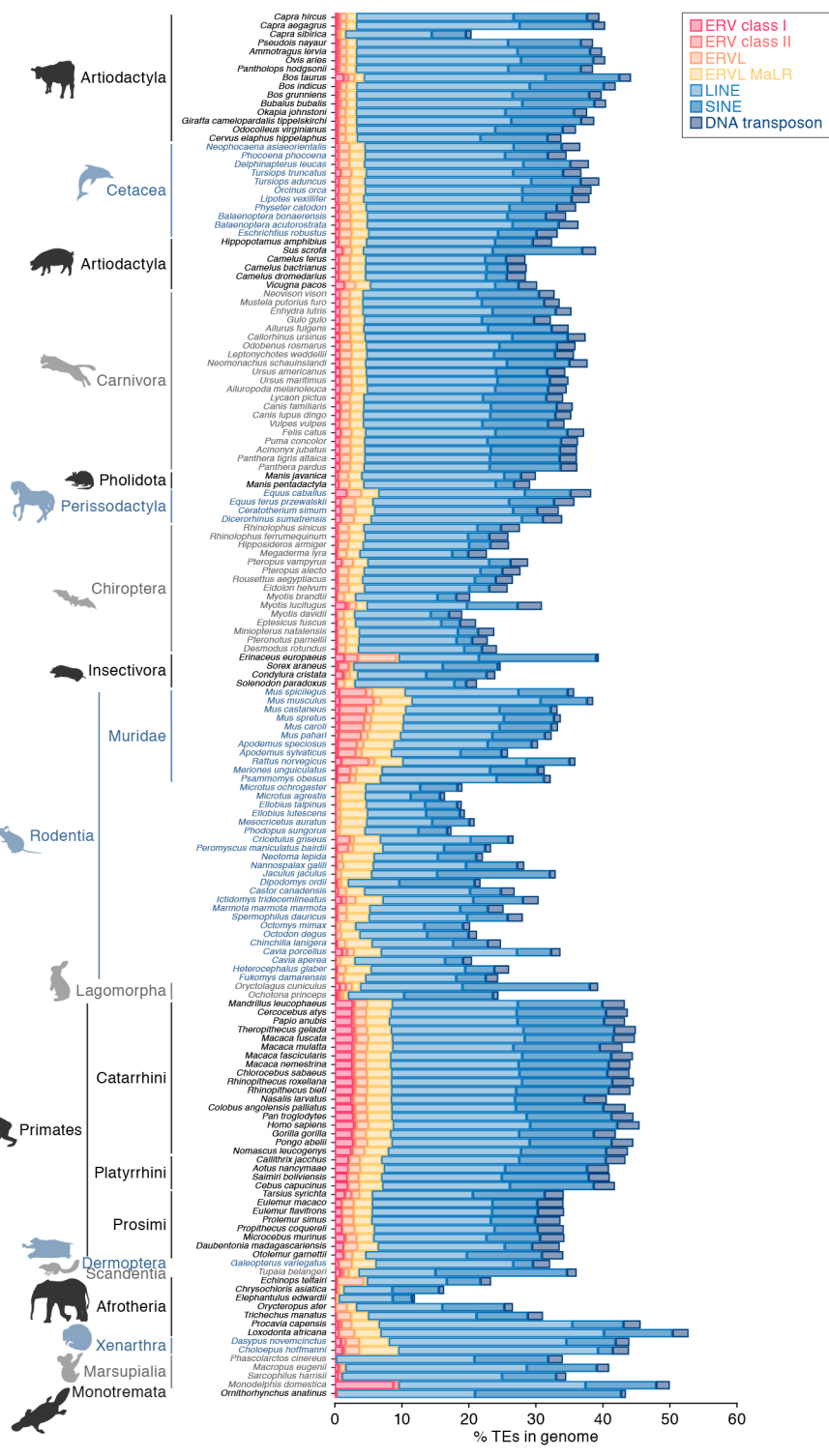


Fig. S6. Proportions of TE sequences in the genomes of the respective mammalian species. Proportions of ERV sequences in the genomes of the respective mammalian species are also shown in Fig. 4A.

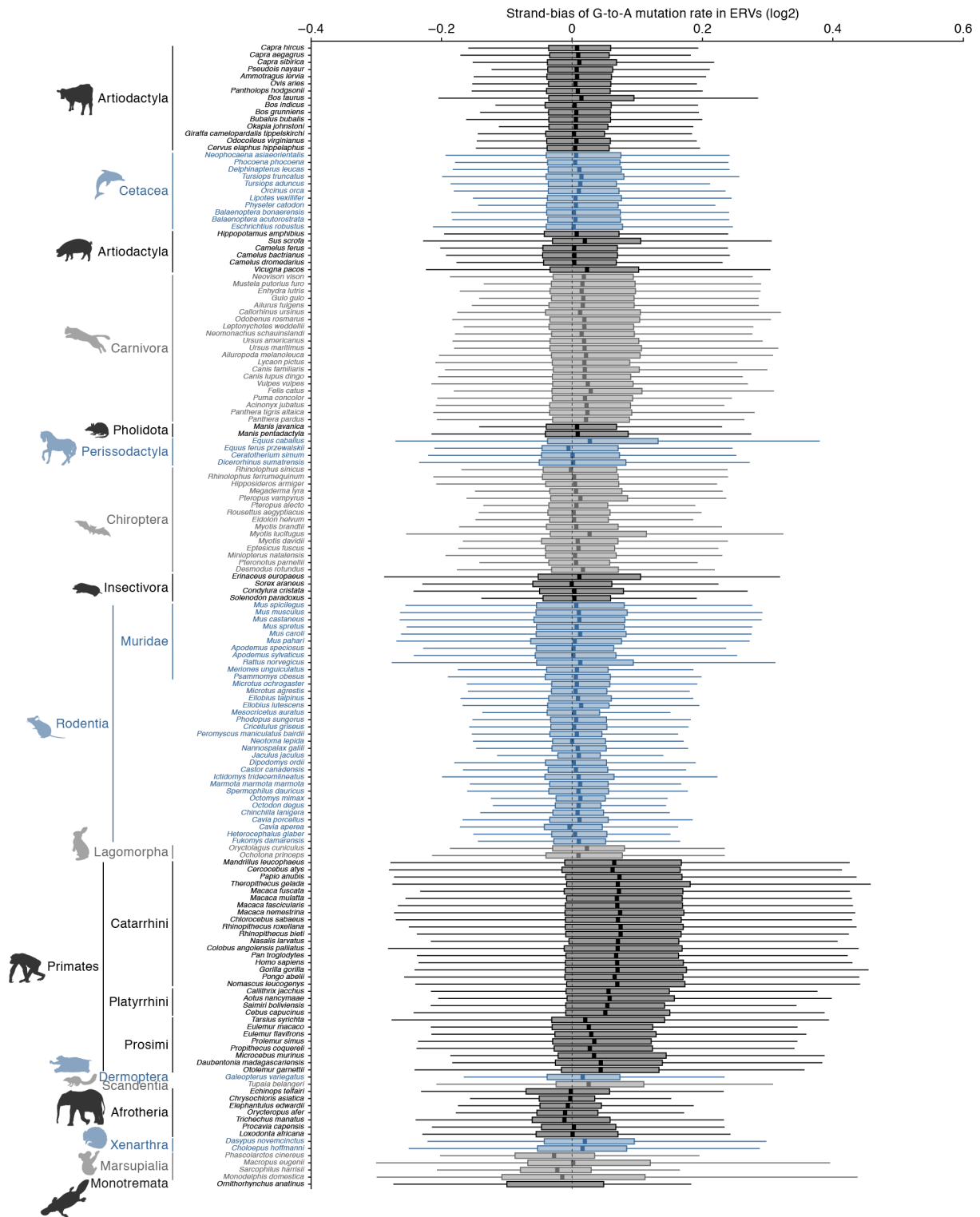


Fig. S7. Difference in the strand biases of G-to-A mutation rates in ERVs among mammals. The log₂-transformed strand bias score of G-to-A mutations is shown (i.e., the mutation rate in the positive strand divided by the one in the negative strand).

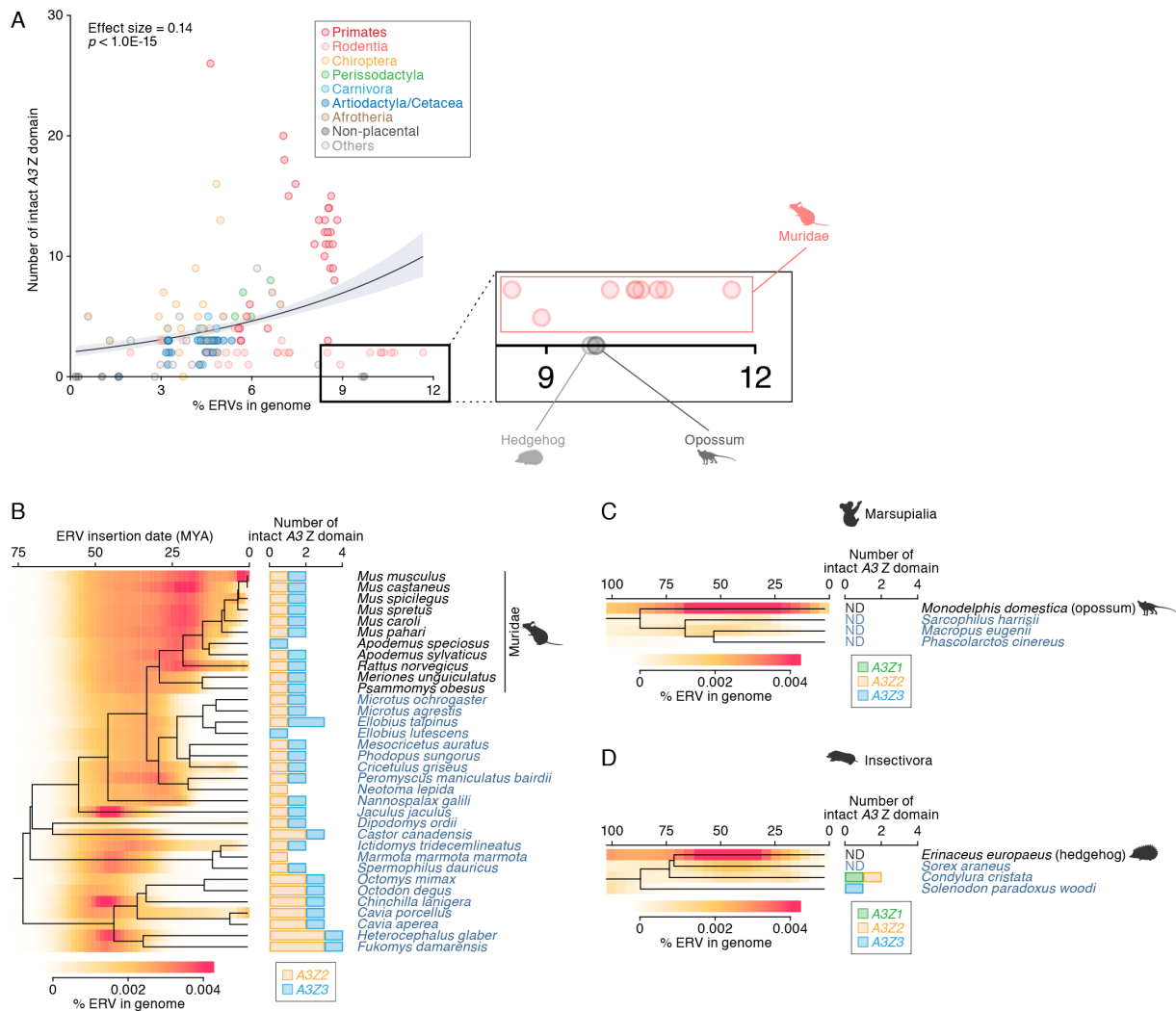


Fig. S8. “Outlier” species with a high number of ERV invasions but only few or no A3 genes. (A) Relationship between the number of A3 Z domains and the amount of ERV insertions in the genome. “Outlier” species (Muridae, hedgehog, and opossum), are indicated by enlarged dots. (B-D) Insertion dates of ERVs and the numbers of A3 genes in mammals. The insertion dates of ERVs and numbers of A3 genes in Rodents (B), Marsupialia (C), and Insectivora (D) are shown. (Left) Amounts of ERV insertions at each age in the respective mammalian species. ERV insertion date was estimated according to the genetic distance of each ERV integrant from the consensus sequence under the molecular clock assumptions (B, 7×10^{-9} mutation/site/year; C and D, 2.2×10^{-9} mutation/site/year). (Right) Number of intact A3 Z domains.

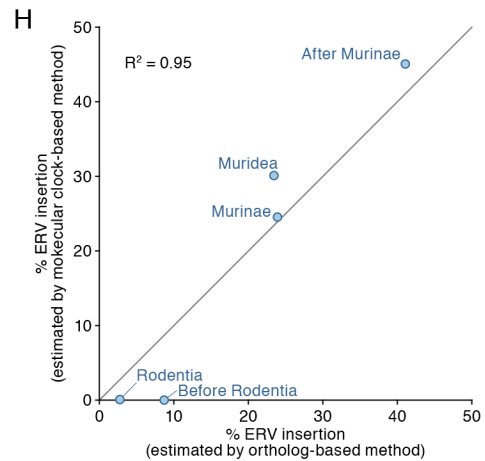
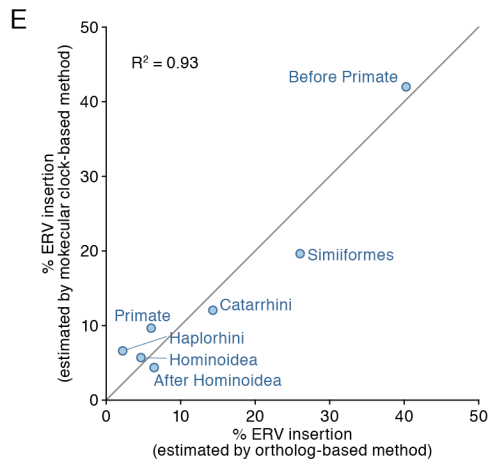
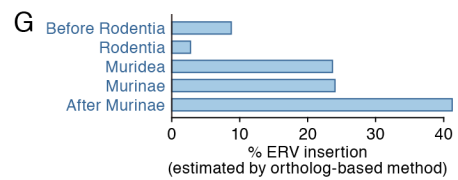
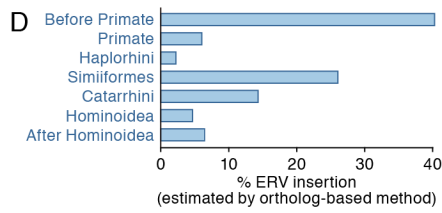
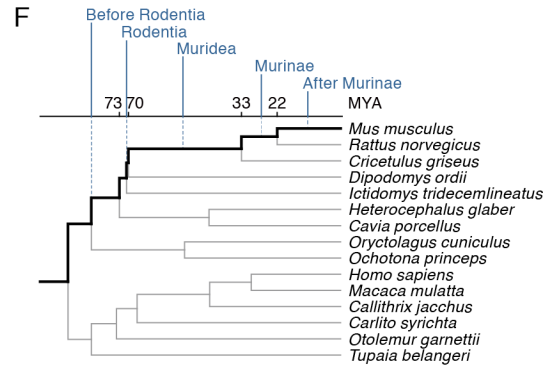
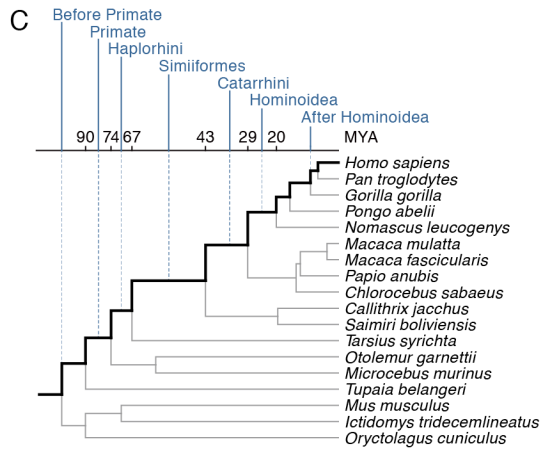
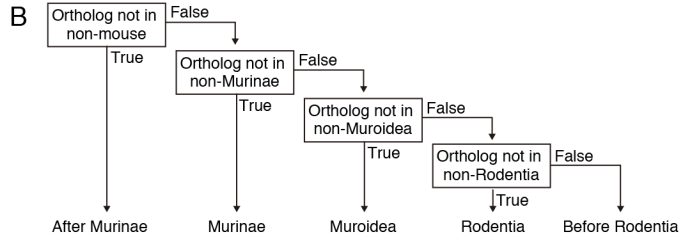
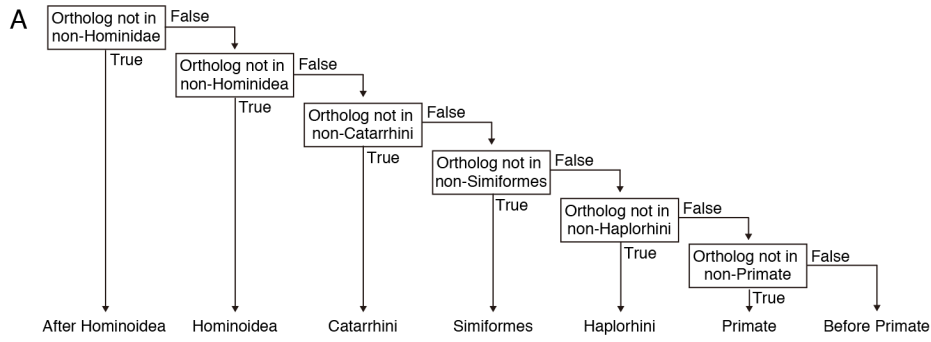


Fig. S9. Estimation of insertion dates of ERVs using the ortholog distribution-based and genetic distance-based methods. (A and B). Flowchart showing the procedure used to estimate the insertion dates of ERVs based on ortholog distributions. The procedures used for ERVs in the genomes of humans (A) and mice (B) are shown. (C-E) Comparison of ERV insertion dates estimated by the ortholog distribution-based and genetic distance-based methods (human ERVs). (C) Stratification of human ERVs according to estimated insertion dates. (D) Amount of ERV insertions acquired in distinct time periods as estimated using an ortholog distribution-based approach. (E) Comparison of ERV insertion amounts estimated by the two methods. In the genetic distance-based method, 2.2×10^{-9} mutation/site/year was used as the mutation rate. The line $y = x$ and coefficient of determination (R^2) are shown. (F-H) Comparison of ERV insertion dates estimated by ortholog distribution and genetic distance-based methods in mouse ERVs. (F) Stratification of mouse ERVs according to the estimated insertion dates. (G) Amount of ERV insertions acquired in distinct time periods as estimated using the ortholog distribution-based methods. (H) Comparison of ERV insertion amounts estimated by the two methods. In the genetic distance-based method, 7×10^{-9} mutation/site/year was used as the mutation rate. The line $y = x$ and coefficient of determination (R^2) are shown.