

FIG. S1. Validation of newly generated anti-human ZSCAN4 antibodies

(A) Specificity of anti-human ZSCAN4 antibodies were assessed by western blotting. Human ES cells were transfected with or without synthetic mRNA encoding human ZSCAN4-3xFLAG-HA. Whole-cell lysates were examined by newly generated anti-human ZSCAN4 antibodies raised in rabbits. Two commercially available rabbit anti-human ZSCAN4 antibodies were examined simultaneously. For positive control, expression of ZSCAN4-3xFLAG-HA was probed by the anti-HA antibody. (B) Newly generated mouse anti-human ZSCAN4 antibodies were examined, as in A.

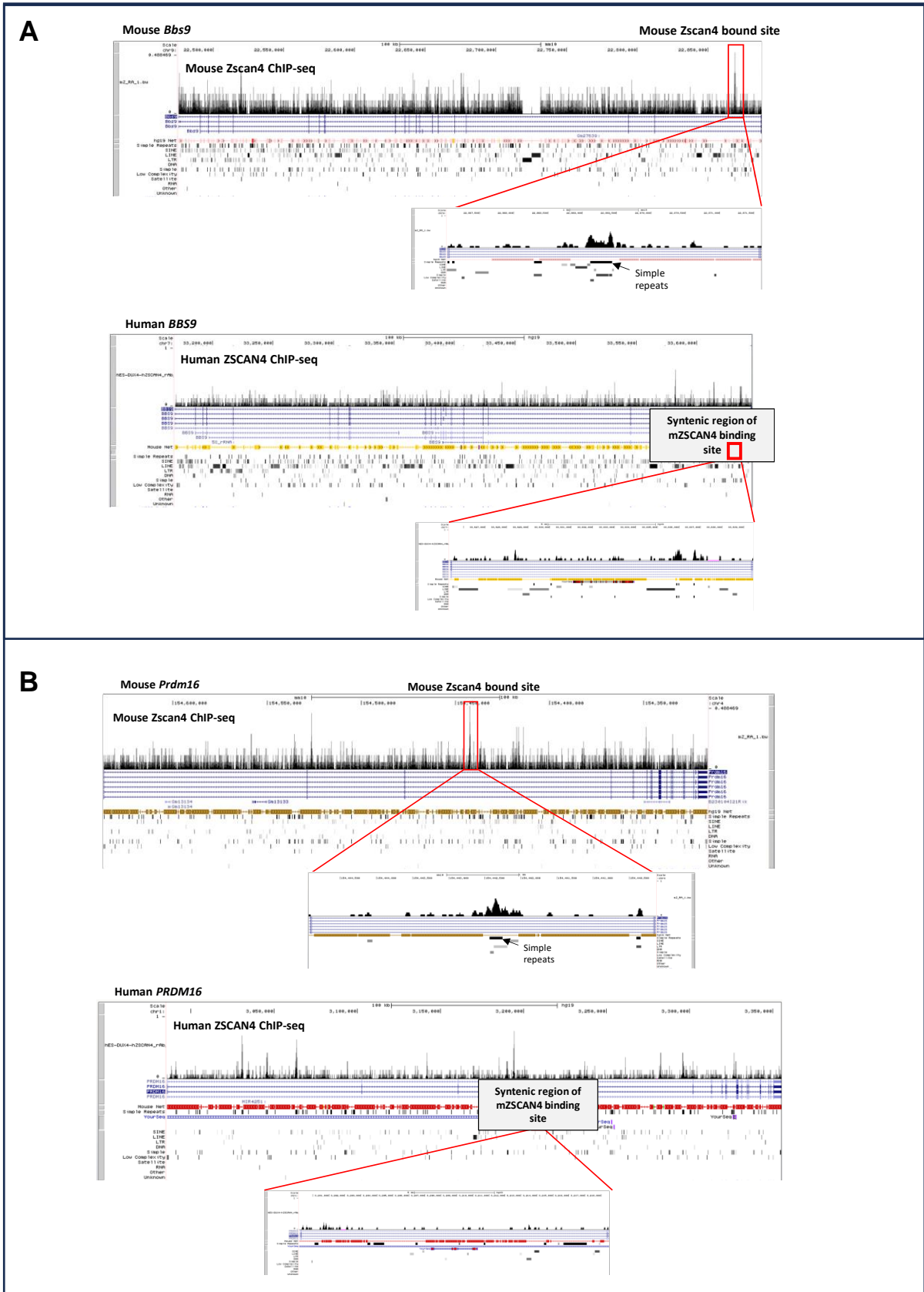


FIG S2

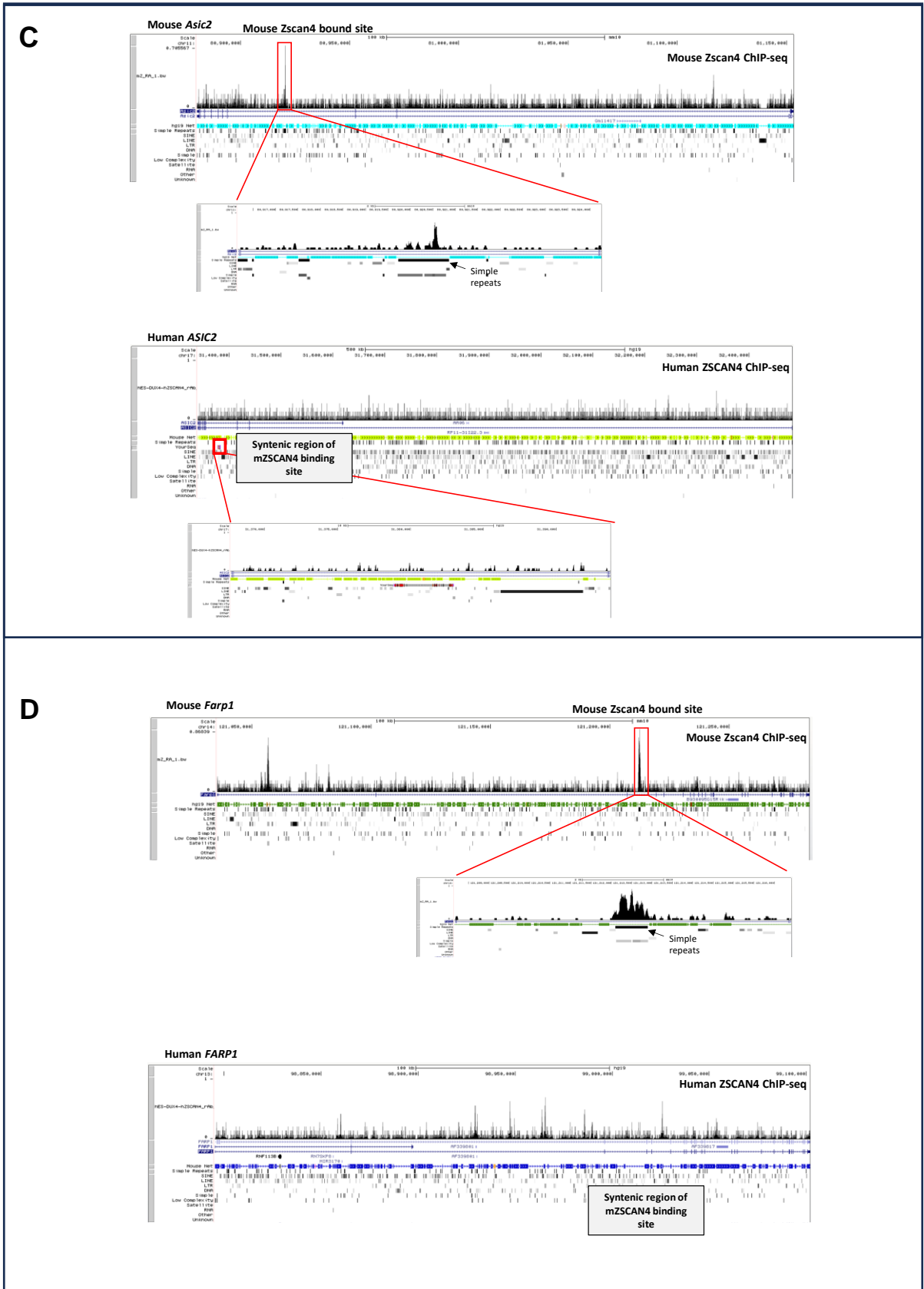


FIG S2

FIG. S2. Comparison of mZSCAN4-binding sites and hZSCAN4 binding sites. Representative regions of mouse genome (mm10) and human genome (hg19) regions where both mouse and human genes have mZSCAN4-binding sites and hZSCAN4 binding sites in their introns. (A) BBS9 gene, (B) Prdm16 gene, (C) Asci2 gene, (D) Farp1 gene. In each figure, the upper panel shows an mZscan4 binding site (ChIP-seq peaks) and the location of the microsatellites. The lower panel shows the corresponding human genome region (ChIP-seq peaks) and the location of microsatellites. The mZSCAN4-binding microsatellite locations do not align with hZSCAN4-binding microsatellite locations.

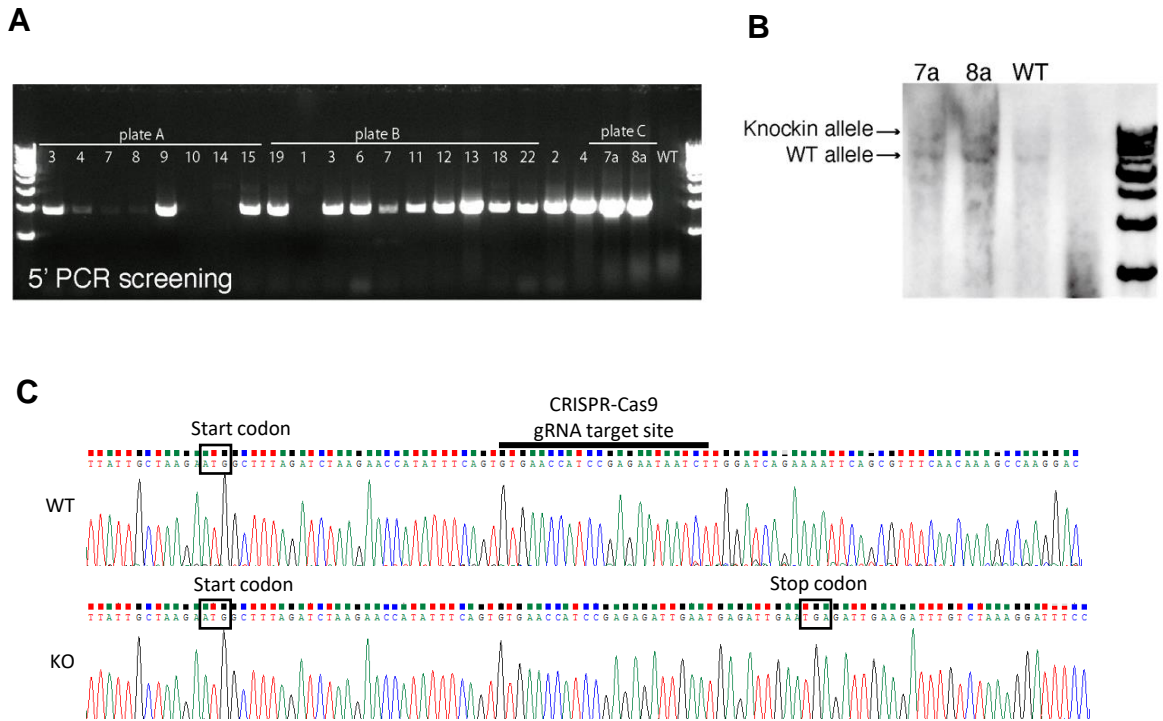


FIG. S3. Generation of hZSCAN4 knockout human ES cells.

(A) Screening for the targeted knock-in allele using 5'-side genotyping PCR.

(B) The *ZSCAN4-EGFP* knock-in allele from the two human ES clones were verified to be heterozygous by Southern blotting after HindIII digestion.

(C) Genomic DNA sequences of *ZSCAN4*-KO ES cells. Sanger sequencing analysis showing that CRISPR-Cas9 introduced an insertion mutation which results in a premature stop codon of *ZSCAN4* gene locus.