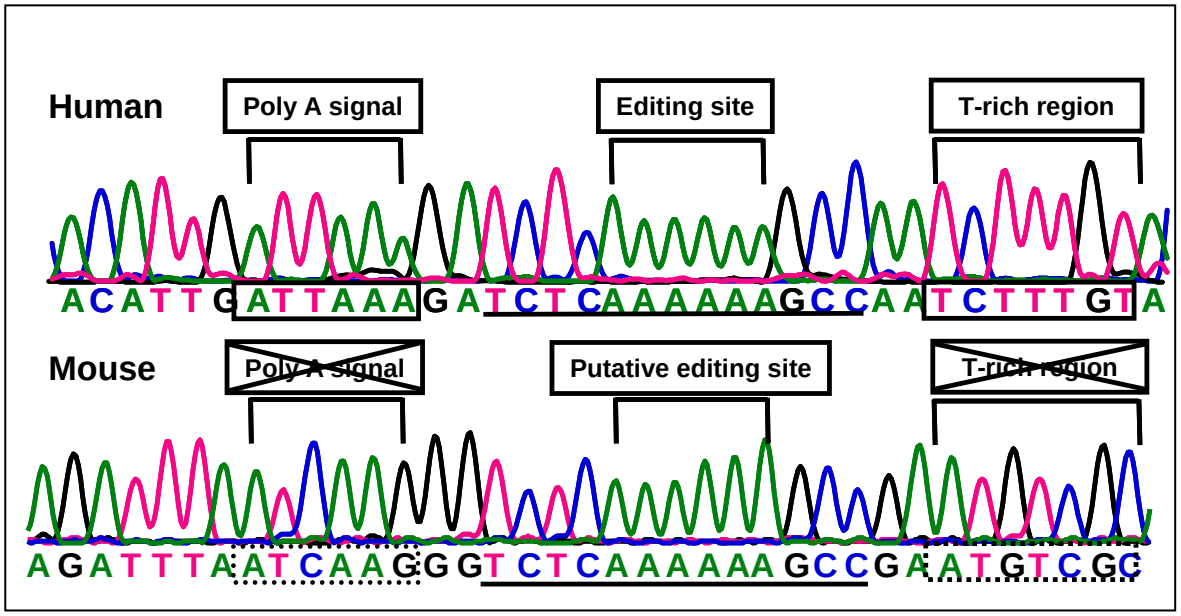
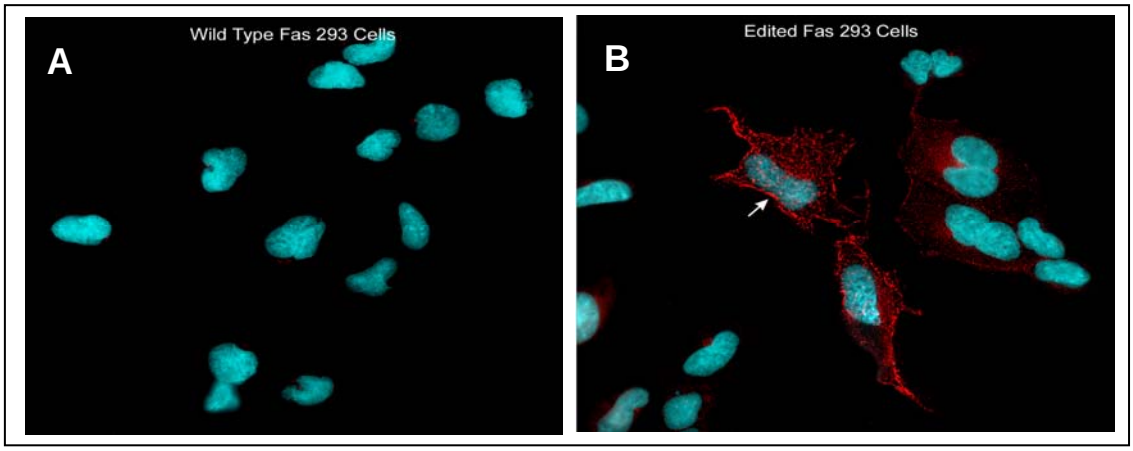
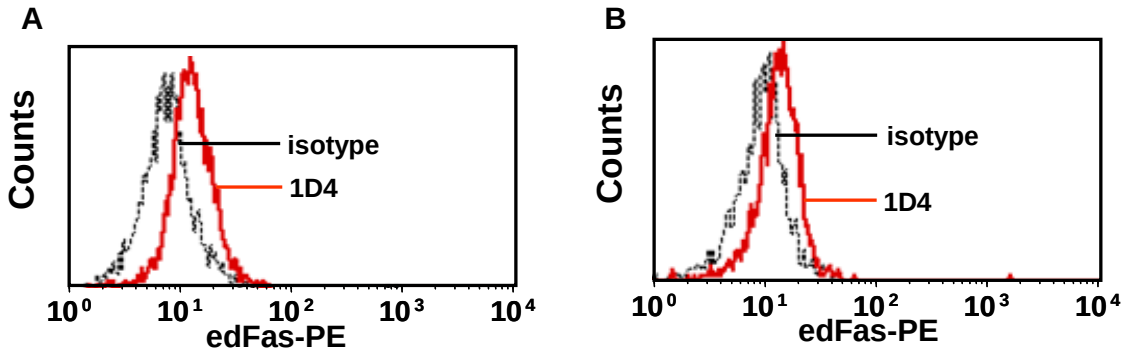


Supp. Figure S1. Poly(A) signal-dependent model for human *FAS* mRNA editing. Human *FAS* and murine *FAS* contain the same nucleotide sequence (underlined) surround the human *FAS* editing site. Upper panel shows the presence of an alternative polyadenylation signal (ATTAAA) (poly(A) signal) at the upstream of mRNA editing site and a thymidine-rich (T-rich) region at the downstream of mRNA editing site in human *FAS* gene. Poly(A) signals are required for genes encoding polyadenylated mRNAs to terminate their transcription. The poly(A) signal could direct polymerase II to pause and to release *FAS* mRNA from the template. The murine *FAS* gene lacks both the alternative poly(A) signal at the upstream of the putative mRNA editing site and the thymidine-rich (T-rich) region at the downstream of the putative mRNA editing site (lower panel), therefore, no adenosine insertion editing may occur in murine *FAS* mRNA.



Supp. Figure S2. Surface staining of edFAS in human cells. A). anti edFAS antibodies failed to stain the wild-type FAS (wtFAS) expressed in the 293 cells with immunostaining assay. B). anti edFAS antibodies stained the edFAS expressed in 293 cells and the edFAS is distributed as membrane receptor pattern in immunofluorescence staining.



Supp. Figure S3. Detection of edFAS protein in subsets of SLE T cells. A). edFAS protein was detected in $CD4^+$ T cells from a SLE patient. B). edFAS protein was detected in $CD8^+$ T cells from a SLE patient. The edFAS protein was undetectable in $CD19^+$ B cells and $CD14^+$ monocytes from SLE patients (data not shown).