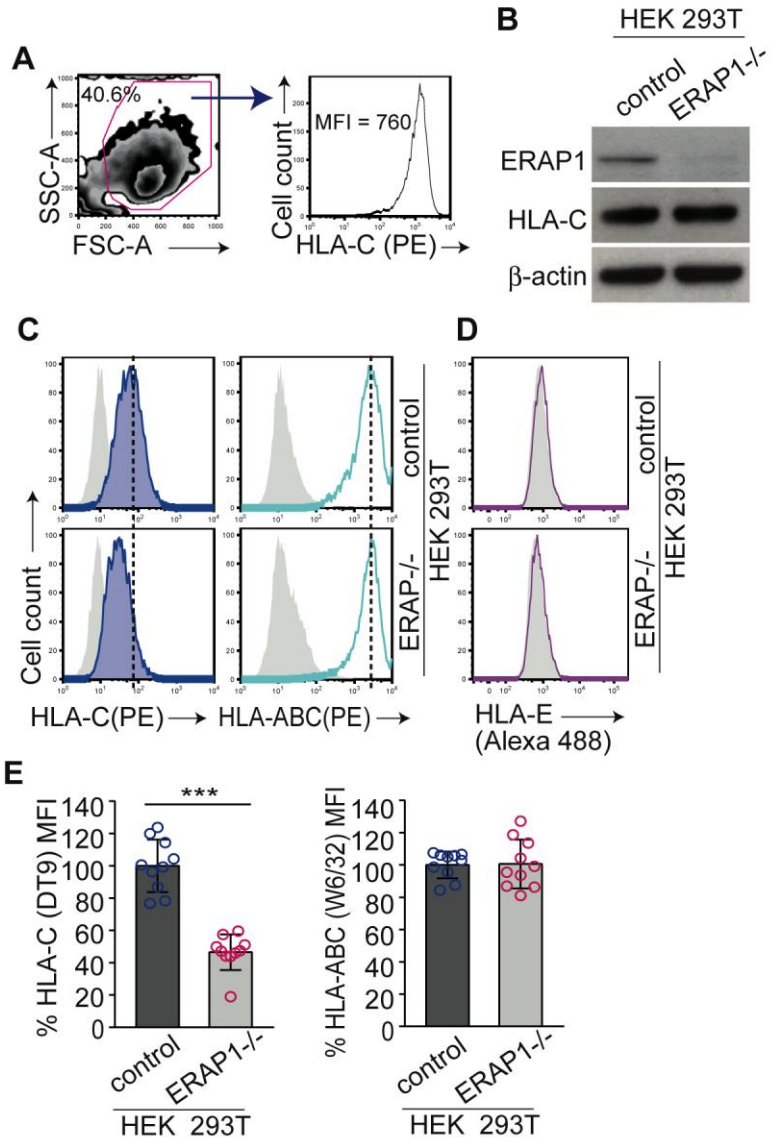

Figure S1. ERAP1 controls IFN-γ induced HLA-C surface expression of HEK293T cells. (**A**) Gating strategy to assess HLA-ABC or HLA-C staining by flowcytometry. (**B**) Western blot analyses of ERAP1, HLA-C, and β-actin from parental (control) or ERAP1$^{-/-}$ HEK293T cells cultured with IFN-γ. (**C**) Representative flowcytometric panels of HLA-C (DT-9) or HLA-ABC (W6/32) staining with isotype controls (gray) of parental and ERAP1$^{-/-}$ HEK293T cells cultured with IFN-γ from technical triplicates in two independent experiments. (**D**) Representative flowcytometric analyses of HLA-E with isotype staining (gray) for parental or ERAP1$^{-/-}$ HEK293T cells cultured with IFN-γ from two independent experiments. (**E**) Mean fluorescence intensity (MFI) of HLA-C or HLA-ABC staining of parental or ERAP1$^{-/-}$ HEK293T cells was assessed by flowcytometry and normalized to means of parental-cell data in each analysis. Data are summarized, compared by non-paired t-test (*** $P < 0.005$) and shown as mean ± s.e.m.

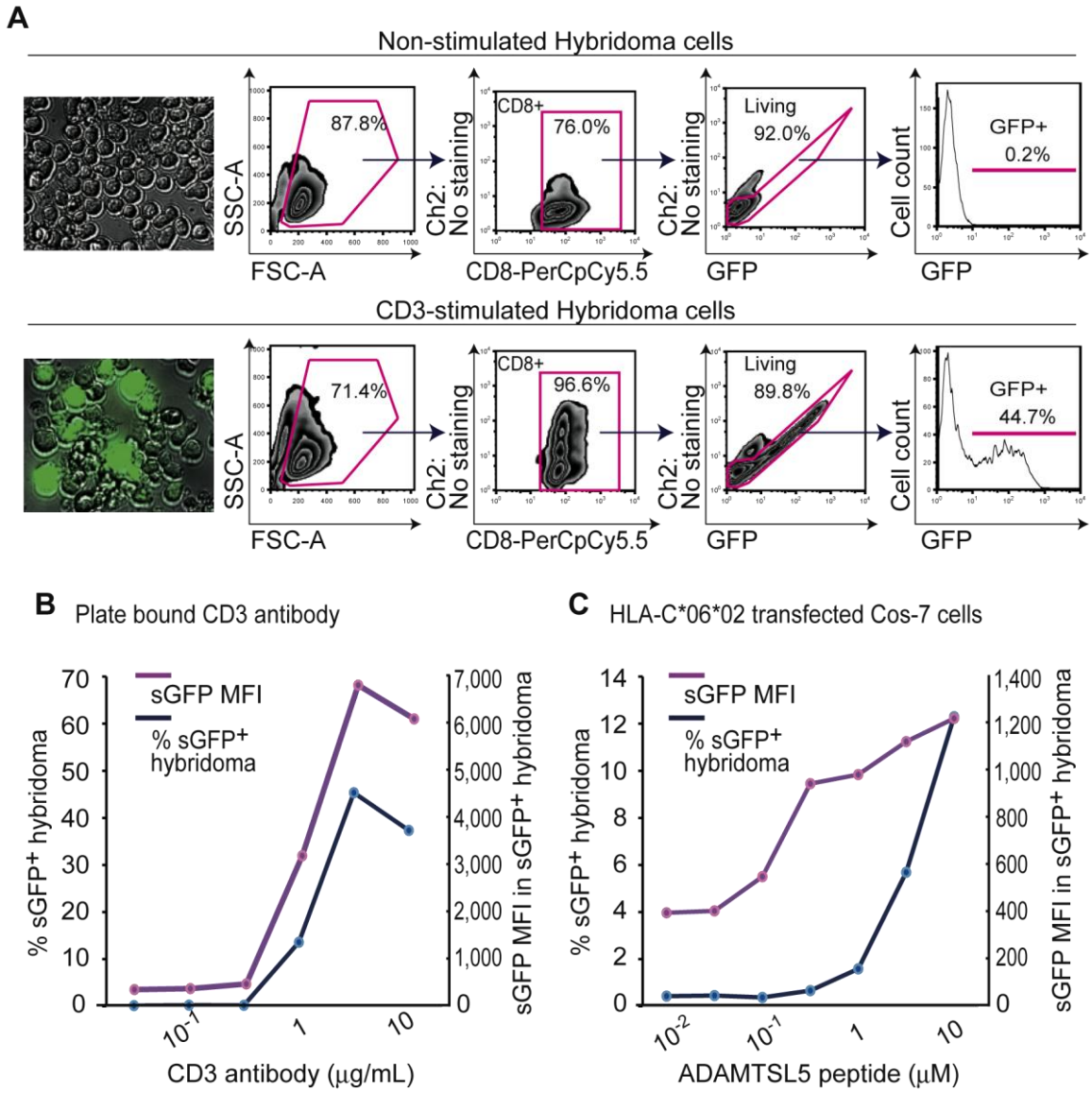

Figure S2. Quantification of sGFP induction by stimulation of the Vα3S1/Vβ13S1-TCR hybridoma cells by TCR ligation or autoantigen. (**A**) Gating strategy to assess sGFP induction in TCR-hybridoma stimulation experiments. Vα3S1/Vβ13S1-TCR hybridoma cells with high autofluorescence in Ch1 and Ch2 were excluded from analyses. In co-culture experiments with HEK293T lineage cells, hybridoma cells were stained with CD8 antibody to discriminate TCR-hybridoma and HEK293T lineage cells. GFP^+ gate is set to 0.2% of GFP^+ cells in non-stimulated hybridoma cells**.** Mean fluorescence intensity (MFI) of sGFP is assessed in $sGFP^+$ hybridoma cells. (**B)** Frequency of $sGFP^+$ hybridoma cells and MFI of sGFP was assessed for stimulation of the Vα3S1/Vβ13S1-TCR hybridoma by increasing concentrations of plate-bound CD3 antibody. Data represent duplicates from two independent experiments. (**C**) Frequency of $sGFP^+$ hybridoma cells and MFI of sGFP expression following stimulation of the Vα3S1/Vβ13S1 TCR hybridoma by co-culture with HLA-C*06:02 stably transfected Cos-7 cells loaded with serially diluted synthetic ADAMTSL peptide (9mer).

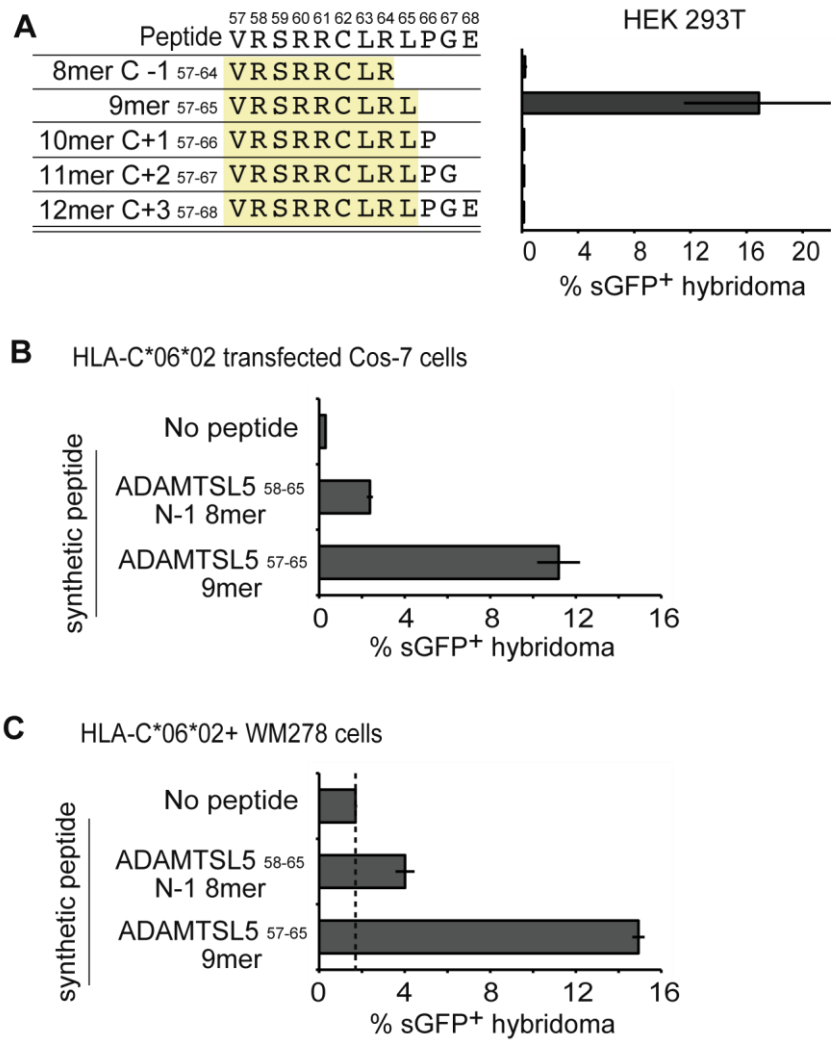

Figure S3. Stimulatory effects of C-terminal modified plasmid-encoded ADAMTSL5 or synthetic ADAMTSL5 peptides (**A**) Vα3S1/Vβ13S1-TCR hybridoma stimulation by co-culture with HEK293T cells co-transfected with HLA-C*06:02 and ADAMTSL5 peptides shortened or extended at the C-terminus according to the natural sequence of ADAMTSL5. Frequencies of sGFP$^+$ hybridoma cells were assessed by flowcytometry. Data represent technical triplicates from two independent experiments. **(B** and **C)** Vα3S1/Vβ13S1-TCR hybridoma stimulation or superstimulation by co-culture with HLA-C*06:02 stably transfected COS-7 cells (**B**) or IFN-γ treated WM278 cells (**C**) loaded with ADAMTSL5 synthetic peptides (8mer or 9mer, each 10 µg/mL). Frequencies of sGFP$^+$ hybridoma cells were assessed by flowcytometry. Data represent technical triplicates shown as mean ± s.e.m.

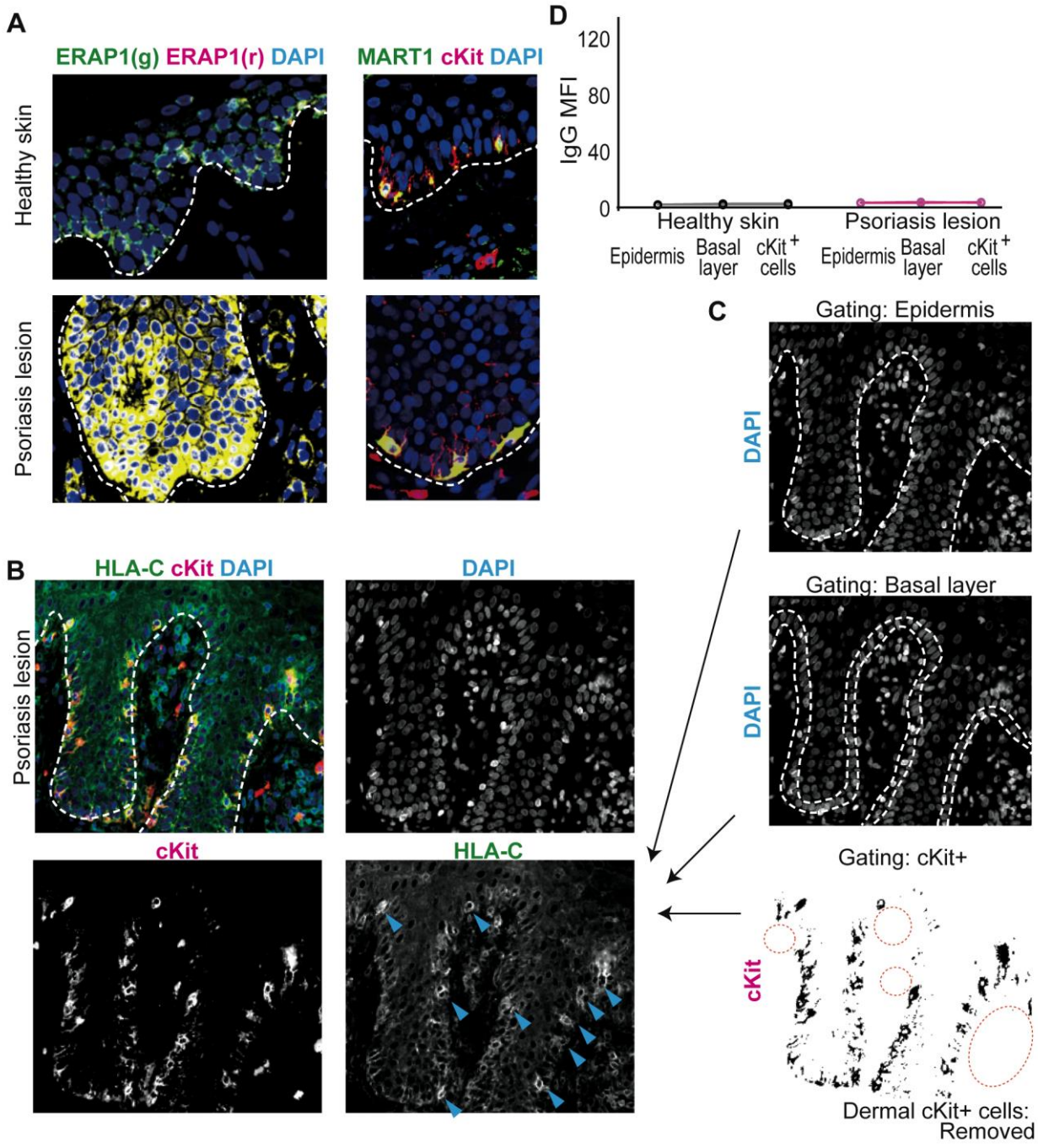

Figure S4. Validation of immunofluorescence staining conditions. (**A**) Two different ERAP1 antibodies generated in goat (green) or rabbit (red) produced indistinguishable staining of healthy or lesional psoriatic skin (overlay shown as yellow), validating the ERAP1 staining method. Two staining methods to detect epidermal melanocytes were compared by double immunofluorescence staining for MART1 (green) and cKit (red). MART1 and cKit labelled the same cell population in the epidermis of healthy skin and psoriatic lesions, validating the melanocyte detection methods. Note that c-Kit antibody also stains mast cells in the dermis. (**B**) A fraction of epidermal cKit$^+$ cells shows higher HLA-C reactivity (blue triangles). (**C**) Fluorescence intensity (MFI) analysis was gated for whole epidermis or basal layer. The basal layer was defined as one cell width in DAPI staining. cKit$^+$ or MART1$^+$ gates excluded the background staining of keratinized layer and cKit$^+$ cells in the dermis. Selection gate was transferred onto HLA-C or ERAP1 staining, and fluorescence intensity was quantified for each selection by image J software. (**D**) In double fluorescence staining we set the background levels for each staining in the presence of the isotype control of the second fluorochrome to avoid counting bias from overlapping emission spectra. cKit$^+$ cells had similar isotype staining intensity from mouse IgG compared to whole epidermis, or basal layers.