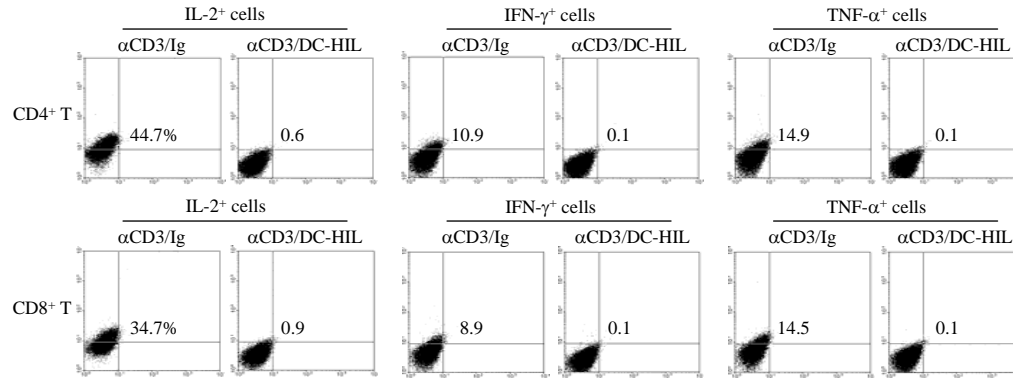
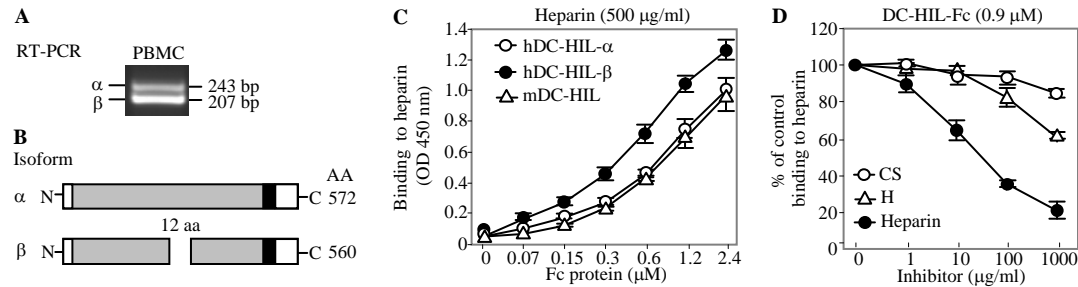


Supporting Information Figure 1



Supporting Information Figure 1. DC-HIL inhibits cytokine production by activated T cells on a per cell basis. CD4⁺ or CD8⁺ T cells (2×10^5 cells/well, in triplicate) purified from PBMCs were cultured for 3 d in microculture wells precoated with anti-CD3 Ab (0.3 μ g/ml) and DC-HIL-Fc or control Ig (10 μ g/ml). Cells were harvested from 3 wells, pooled, permeabilized, and then stained with PE-conjugated anti-IL-2, anti-IFN- γ , and anti-TNF- α . Numbers in quadrants indicate the percentage of cytokine-positive cells. Data shown are representative of 2 independent experiments.

Supporting Information Figure 2



Supporting Information Figure 2. Human DC-HIL α and β isoforms possess heparin-binding activity.

(A) mRNA expression of human DC-HIL was examined by RT-PCR of total RNA prepared from PBMCs. PCR products were size-fractionated through 0.8% agarose gel running. The upper and lower PCR bands correspond to α (full-length) and β isoforms (truncated form), respectively. (B) A full-length cDNA encoding the β isoform was cloned and sequenced, and deduced amino acid sequence (560 amino acids) was aligned with the α isoform (572 amino acids) consisting of leader sequence (open), extracellular (gray), transmembrane (black), and intracellular (open) domains. (C) Varying concentrations (μM) of mouse DC-HIL-Fc or human DC-HIL-Fc (α and β isoforms) were incubated in ELISA wells (triplicate) chemically coated with heparin (500 $\mu\text{g}/\text{ml}$). After washing, DC-HIL-Fc protein bound to heparin was assayed by ELISA. Specific binding is expressed as OD_{450} reading left after subtracting background by control mouse IgG from experimental OD_{450} . (D) Binding of human α isoform (0.9 μM) to heparin was inhibited by different doses ($\mu\text{g}/\text{ml}$) of CS (chondroitin sulfate), H (heparan sulfate), and heparin. Second set of experiments (for C and D) showed similar results.