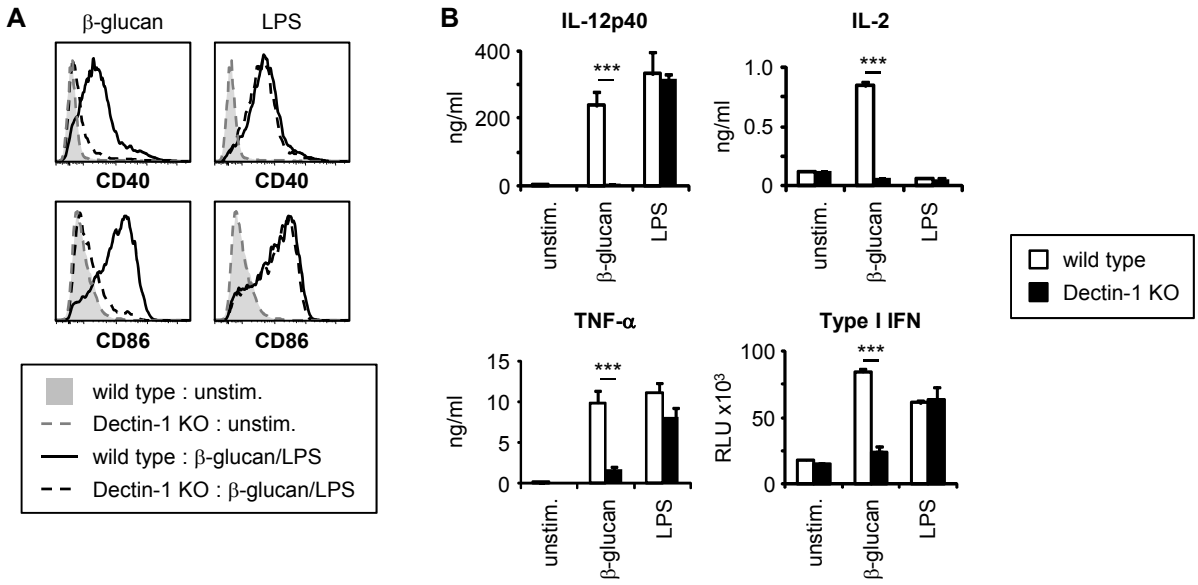
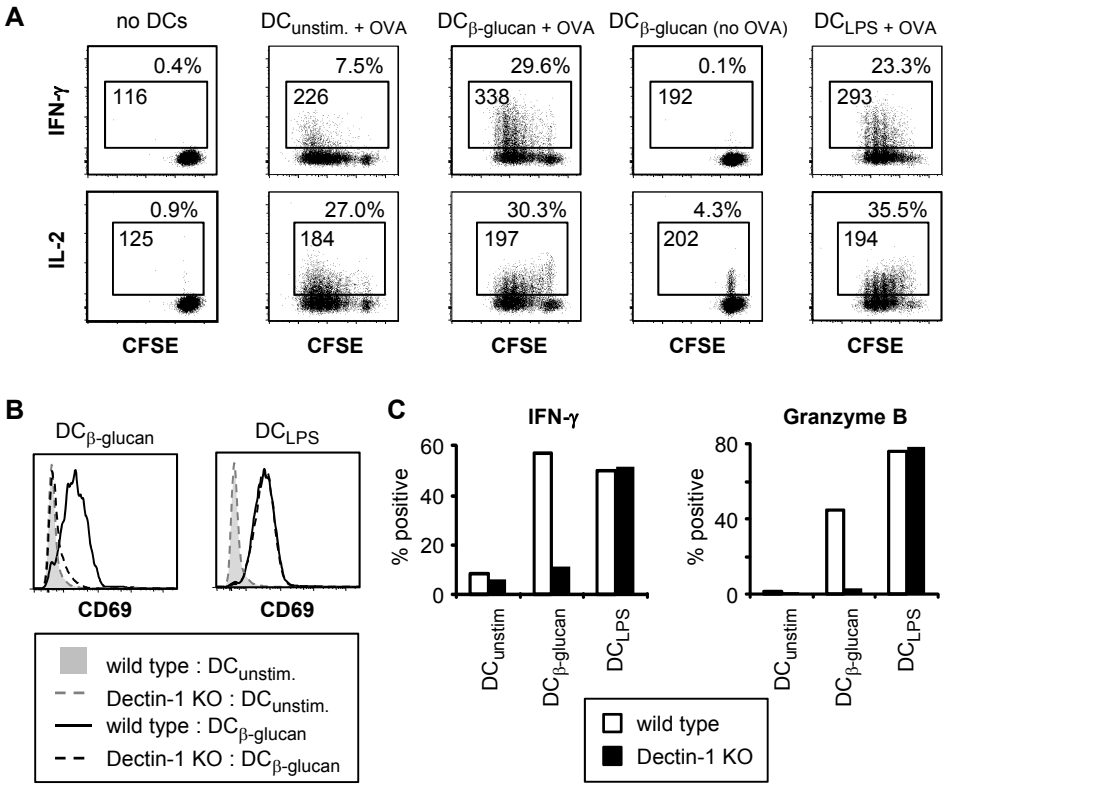
# Supplemental Figure 1



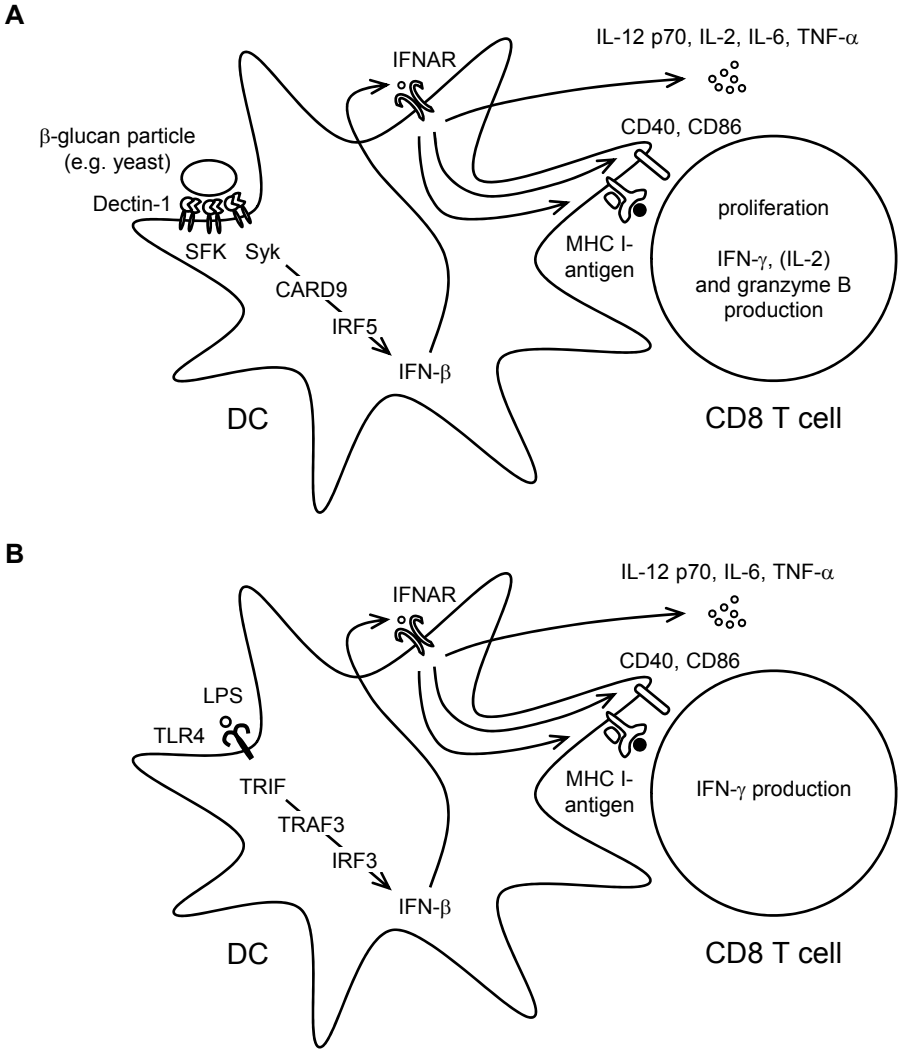

**Supplemental Figure 1. Fungal β-glucan particle-induced DC maturation is Dectin-1-dependent.** Bone marrow-derived DCs from wild type or Dectin-1-deficient mice were stimulated with 100 μg/ml β-glucan particles or 100 ng/ml LPS for 24 hours. A) Co-stimulatory molecule expression by the DCs was assessed by flow cytometry. B) Cytokine production by the DCs was assessed by ELISA (all except type I IFNs) or a luciferase reporter assay (type I IFNs). Cytokine data are mean plus standard deviation of triplicate culture. All data are representative of at least 3 independent experiments. ***p<0.001

# Supplemental Figure 2




**Supplemental Figure 2. CD8 T cell activation by β-glucan-stimulated DCs is Dectin-1-dependent.** Bone marrow-derived DCs from wild type (A-C) or Dectin-1-deficient (B-C) mice were stimulated with 100 μg/ml β-glucan particles or 100 ng/ml LPS for 24 hours. DC cultures were supplemented with 1.1 nM OVA peptide (SIINFEKL) for 1 hour prior to β-glucan/LPS stimulation (A, as indicated). After stimulation, DCs were washed with PBS prior to the addition of CFSE-labeled OT-I CD8 T cells (1:5 DC:T cell ratio), and co-cultures were incubated for 3 days. T cells were then harvested for flow cytometry to assess CD69 (B; gated on proliferating $CD8^+$ cells), and re-stimulated with PMA + ionomycin for 6 hours in the presence of protein export inhibitors for the last 4 hours to assess cytokine and granzyme B production by intracellular flow cytometry (A, C; gated on $CD8^+$ cells). % positive cells and MFI (gated on positive cells) are indicated (A) or plotted (C). All data are representative of at least 3 independent experiments.

# Supplemental Figure 3




**Supplemental Figure 3. Role of type I IFNs in promoting CD8 T cell activation by fungal β-glucan- and LPS-stimulated DCs.** Fungal β-glucan particles and LPS induce the production of type I IFNs (predominantly IFN-β in myeloid DCs) by activating IRF transcription factors. β-glucan particles are detected by Dectin-1, which signals via Src family kinases (SFK), Syk and CARD9 to activate IRF5 (A), while detection of LPS induces TLR4 signaling via TRIF and TRAF3 to activate IRF3 (B). The type I IFNs act in an autocrine manner via their receptor (IFNAR) to promote the presentation of exogenous antigen on MHC I molecules, surface expression of the co-stimulatory molecules CD40 and CD86, and the release of other cytokines, including IL-12 p70. Both fungal β-glucan- and LPS-stimulated DCs activate naïve CD8 T cells, but the role of autocrine type I IFN signaling in CD8 T cell activation by these DCs differs. IFN-γ production by CD8 T cells activated by DCs in response to both stimuli is dependent on the autocrine action of type I IFNs, but only β-glucan-stimulated DCs require autocrine type I IFN signaling to enable them to induce T cell proliferation and granzyme B production. IL-2 production by CD8 T cells activated *in vivo* (but not in *in vitro* co-cultures) by fungal β-glucan-stimulated DCs is also dependent on autocrine type I IFN signaling. Type I IFN-independent DC and T cell responses (including T cell expression of CD44 and CD69) are not shown.