

SUPPLEMENTAL FIGURE LEGENDS:

Supplemental Figure 1. NF- κ B activation response is augmented in HS mutant DCs exposed to LPS.

Cultured BMDCs harvested from mice carrying a mutation in *Ndst1* (*Ndst1^{ff} CD11cCre⁺* mutants) and *Ndst1^{ff} CD11cCre⁻* control BMDCs were exposed to LPS (100 ng/ml) for the indicated time periods. NF- κ B activation was measured as the phospho-p65 signal (indexed to total protein loading), and was quantified and graphed as the normalized signal to baseline. A minimal response seen by wildtype cells appeared to be augmented in *Ndst1* deficient mutant DCs. (Data reflects responses from 2 independent experiments.)

Supplemental Figure 2. Titration of antigen exposure for analysis of antigen presentation in BMDCs in mutant and wildtype DCs. BMDCs from mice deficient in the major HS sulfating enzyme *Ndst1* (*Ndst1^{ff} CD11cCre⁺* mutants) and wildtype *Cre⁻* control BMDCs were exposed to Ova SIINFEKL peptide and examined for SIINFEKL antigen presentation by flow cytometry using SIINFEKL/H-2Kb monoclonal antibody clone 25-D1.16 that detects SIINFEKL in the context of MHC-I on the DC surface. SIINFEKL antigen was given either overnight (o/n) or as a 2hr pulse, which boosts antigen on the cell-surface. Graphs show –fold presentation responses for SIINFEKL exposed cells normalized to that of control cells with no pre-exposure to SIINFEKL run in parallel. (LPS stimulates antigen presence on the DC surface to some extent; right graph). Nevertheless, in either non-LPS or +LPS conditions, mutation resulted in augmented antigen presentation, and to a greater degree after a 2 hr pulse exposure than after overnight exposure. (Data is from cultured DCs that were also employed for glycan compositional analysis to confirm inhibitory effect of *Ndst1* mutation on glycan disaccharide sulfation; refer to main Fig.3B)



