

Supplementary figure 1. *Irf8^{R294C}* mutation selectively abrogates cDC1 development. (A) Flowcytometric analysis of splenic DC subsets depicts the absence of CD11c⁺B220⁺SiglecH⁺ pDCs (analysed in live and CD11c⁺B220⁺gate) in IRF8^{-/-} and CD11c⁺B220⁻CD8α⁺CD4⁻ cDC1s (analysed in live and CD11c⁺B220⁻gate) in both IRF8^{-/-} and IRF8^{R294C} mice. (B) Characterisation of FLDC cultures exhibit the absence of CD11c⁺B220⁺ pDCs (analysed in live and CD11c⁺gate) in IRF8^{-/-} and IRF8^{-/-} and abrogation of CD11c⁺B220⁻CD24⁺Sirpα⁻ cDC1s (analysed in live and CD11c⁺B220⁻gate) in both IRF8^{-/-} and IRF8^{R294C} mice. (C) qRT-PCR analysis of *Irf8* transcript in splenic CD11c⁺B220⁺ pDCs and CD11c⁺B220⁻CD8α⁻CD4⁺ DCs (CD4⁺ DC) show increased level of *Irf8* transcript in IRF8^{R294C} pDCs in comparison to IRF8^{WT}. Data is representative of two independent experiments with error bar representing + SEM and *p<0.05. p value obtained from Student's t test. Data (**A**,**B**) are representative of three independent experiments.