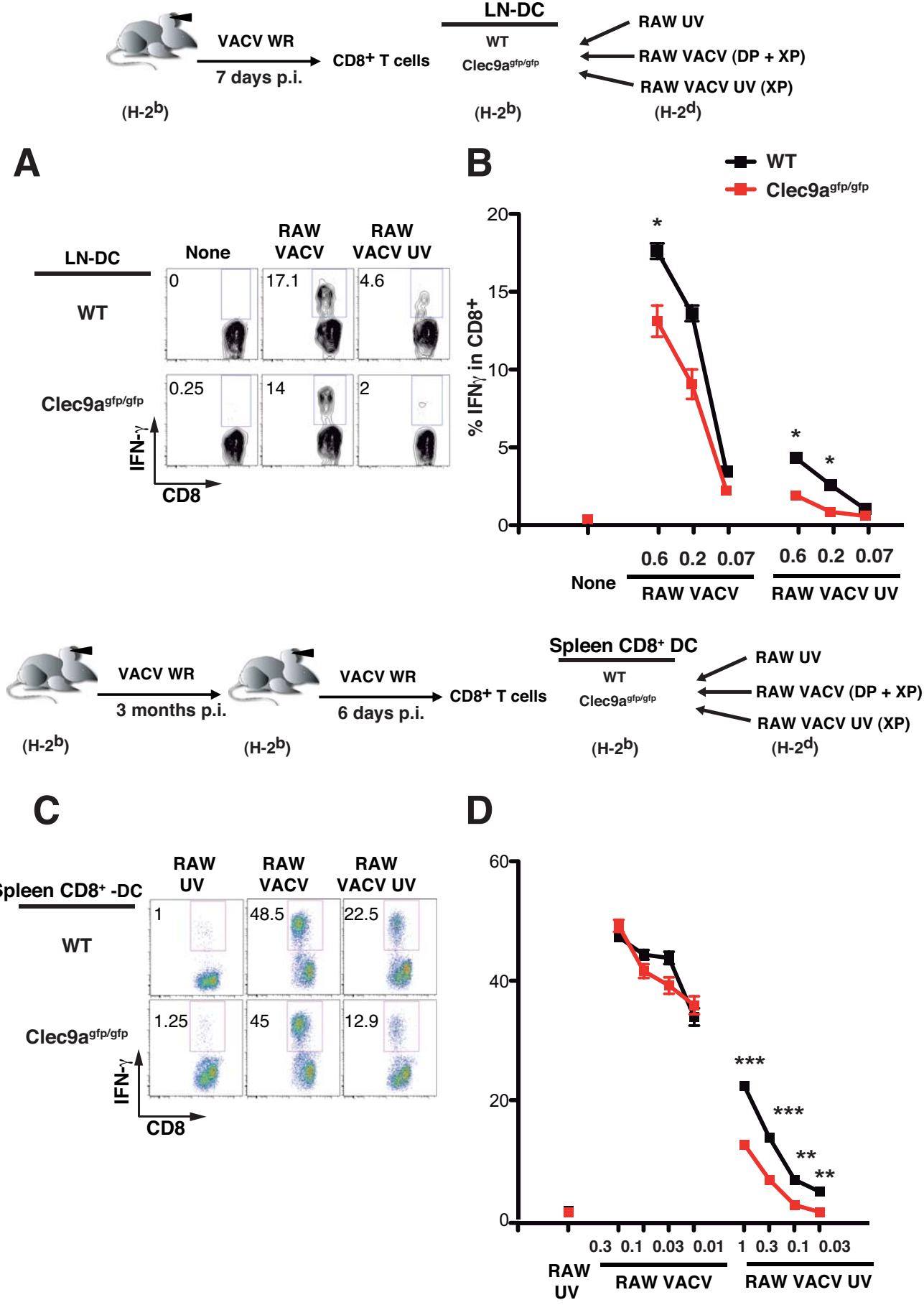
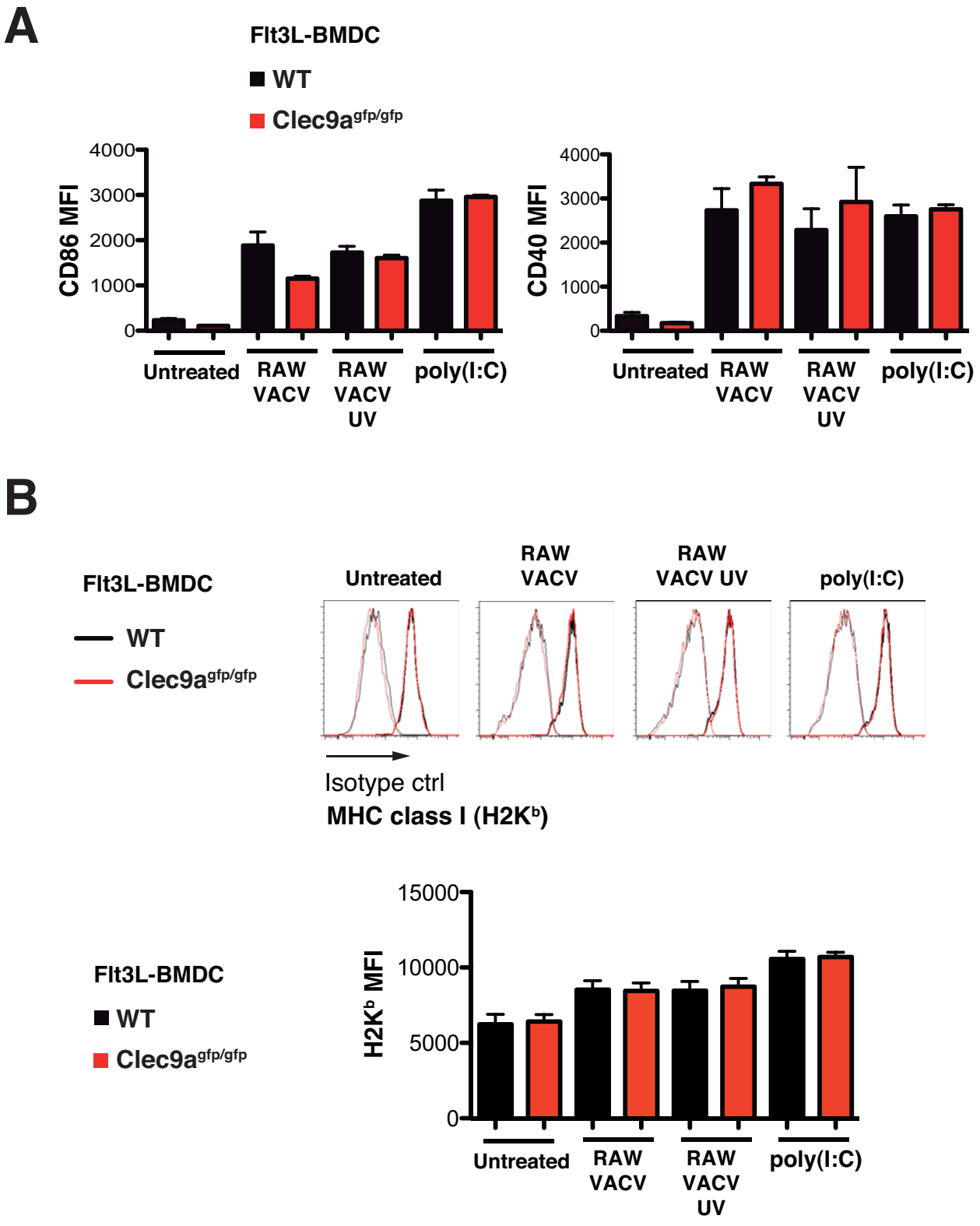
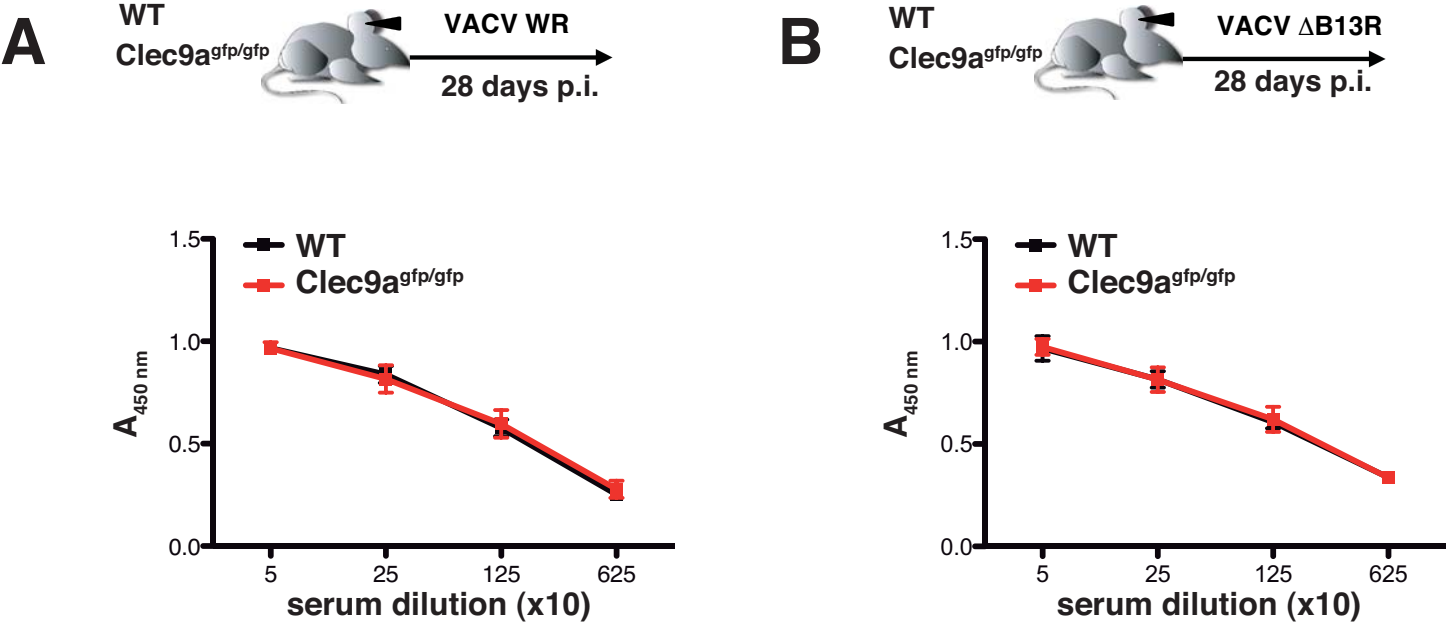


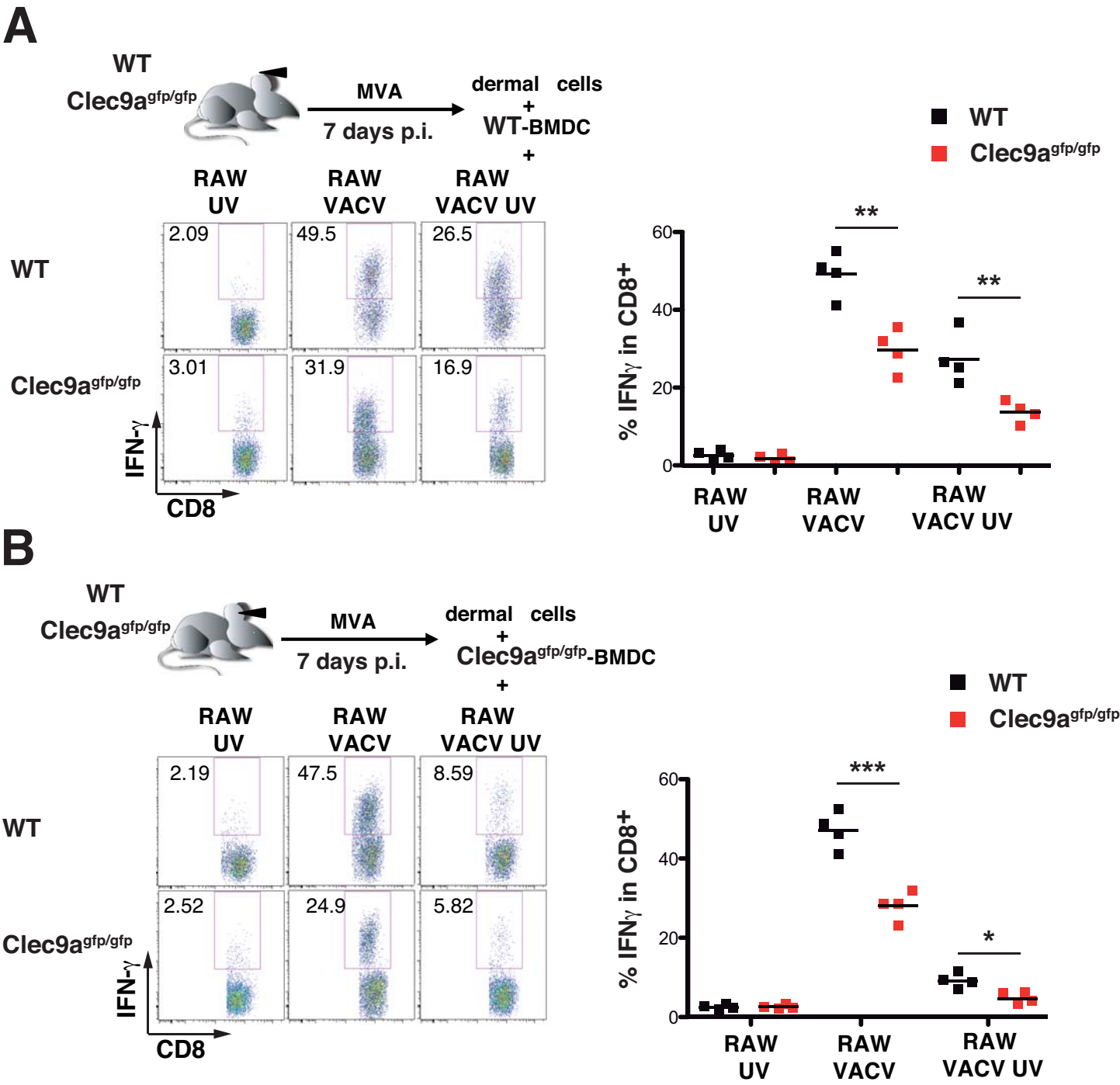
Supplemental Figure 1. Loss of DNGR-1 blocks cross-presentation of vaccinia antigens in infected cells. Impaired cross-presentation of VACV antigens by DNGR-1–deficient DCs obtained ex vivo from LN or spleen. The assay was performed as in Figure 1A, but with total DCs purified ex vivo from peripheral LNs (A, B) or with CD8α+ DCs purified from spleen (C, D). Data expression and statistics are as in Figure 1A and B. *P<0.05, **P<0.01, *** P<0.001, Student's t test.



Supplemental Figure 2. DNGR-1 deficiency does not affect the induction of co-stimulatory and class I MHC molecules in DCs upon exposure to VACV-infected cells. (A) Expression of co-stimulatory molecules is shown as mean fluorescence intensity ± SEM for a representative experiment (n=3 biological replicates) of three performed. (B) H2Kb (MHC class I) expression (bold lines) or isotype control (thin lines) are shown as a representative histogram (upper panel). MHC class I mean fluorescence intensity ± SEM for a representative experiment (n=3 biological replicates) of three performed (lower panel). (A, B) Differences between WT and DNGR-1 deficient DCs in each group are not statistically significant.



Supplemental Figure 3. DNGR-1 deficiency does not affect antibody responses to vaccinia virus infection. WT or DNGR-1–deficient mice were infected with WR (A) or ΔB13R (B) VACV strains. Serum VACV-specific IgG was determined by ELISA on day 28 post-infection (p.i). A representative independent experiment is shown of three performed, and data are expressed as mean absorbance at 450 nm ± SEM of three biological replicates per group. Differences are not statistically significant.



Supplemental Figure 4. Restimulation ex vivo with Flt3L BMDCs from DNGR-1–deficient mice further impairs restimulation by cross-presented antigens.
WT or DNGR-1–deficient mice were infected i.d. in the ear with MVA. On day 7 post-infection (p.i.), ear dermal cell suspensions containing effector T cells were re-stimulated for IFN-γ production in the presence of WT (A) or DNGR-1–deficient (B) Flt3L BMDCs pretreated with RAW-VACV or RAW-VACV-UV, as in Figures 6 and 9. Left panels show representative dot plot sets. Right panels show individual data for production of IFN-γ in $CD8^+$ T cells and $CD4^+$ T cells from a representative experiment (n=4 biological replicates) of three performed. *P<0.05, **P<0.01, unpaired two-tail Student's t test.