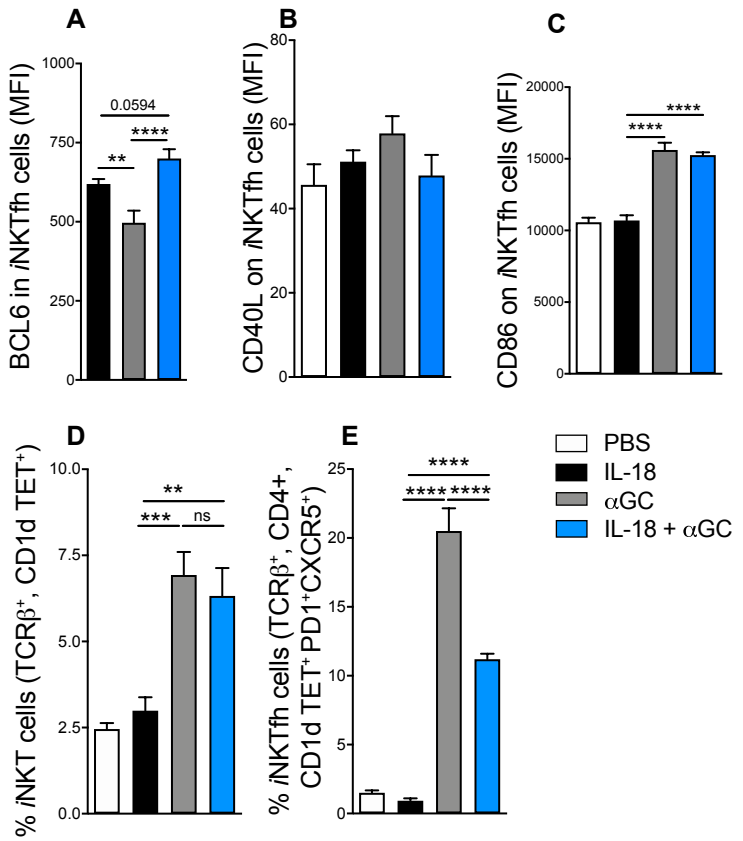
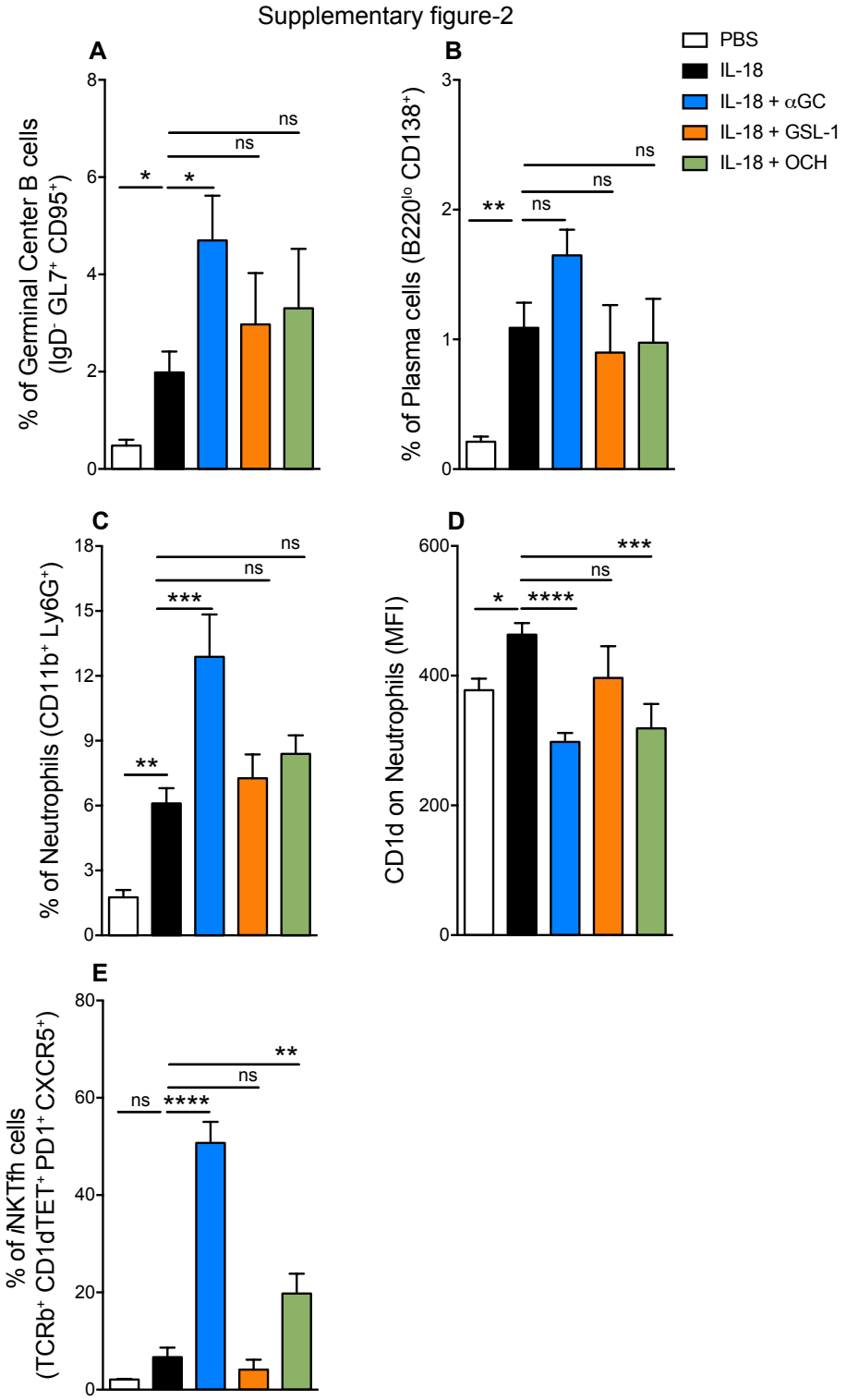
Supplementary figure-1

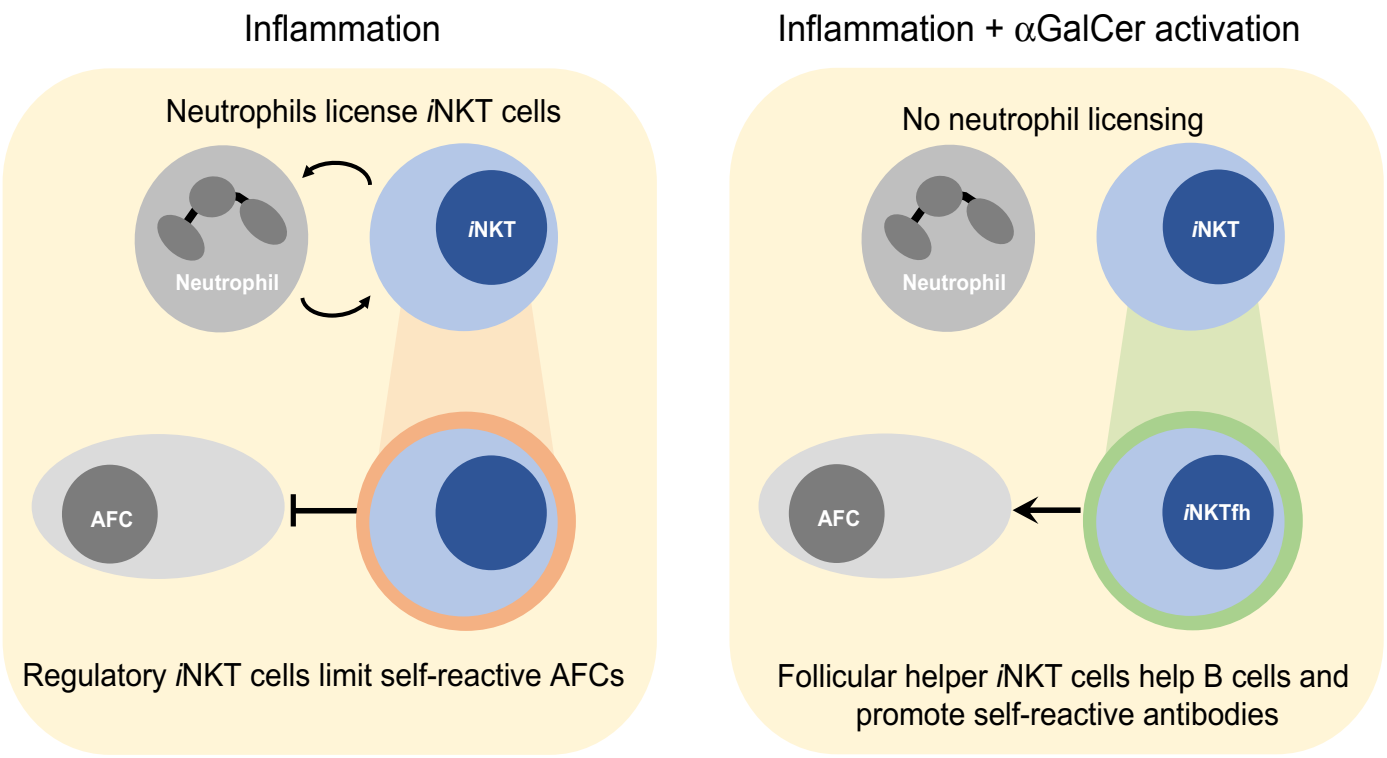


Supplementary figure-1: (A) Intracellular expression of BCL6; cell surface expression of (B) CD40L and (C) CD86 on *i*NKTfh cells; percent of (D) *i*NKT and (E) *i*NKTfh in the spleen on day 7. One experiment (n=6 mice/group).One way Anova. **p<0.005, ***p<0.001, ****p<0.0001. Graphs display group mean +/- SEM.



Supplementary figure-2: GSL-1 or OCH activated *i*NKT cells do not provide B cell help during chronic inflammation. 5μg of αGalCer Th1 polarising glycolipid GSL-1 or Th2 polarising glycolipid OCH were injected on day one in the IL-18 model as depicted in figure-1A. Single cell suspensions from spleens of these mice were analyzed by flow cytometry on day 12. Data show percentage of **(A)** Germinal center B cells, **(B)** Plasma cells, **(C)** neutrophils, **(D)** CD1d median fluorescence intensity (MFI) on neutrophils and **(E)** *i*NKT follicular helper cells. Data representative of 4 experiments with 3-5 mice/group. Statistical test: One way ANOVA. p-values: *<0.05, **<0.005, ***<0.001,****<0.0001, ns: not significant. Graphs display group mean +/- SEM.

Supplementary figure-3



Supplementary figure-3: (A) Under inflammatory conditions, *i*NKT cells are licensed by neutrophils to regulate autoreactive antibody forming cells (AFC). (B) When inflammation is combined with αGalCer, *i*NKT cells promote B cell response, including those that are self-reactive by providing B cell help.