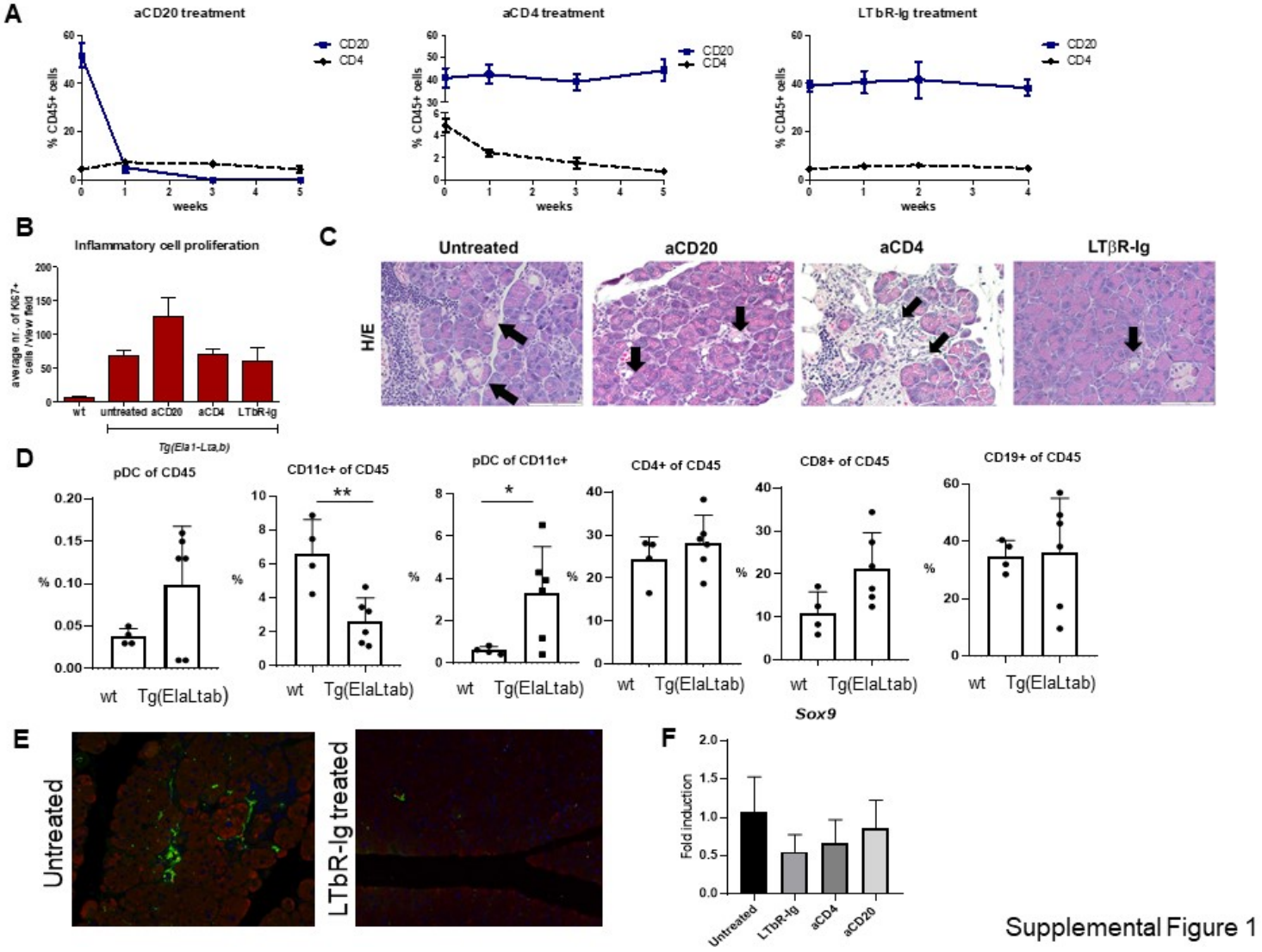
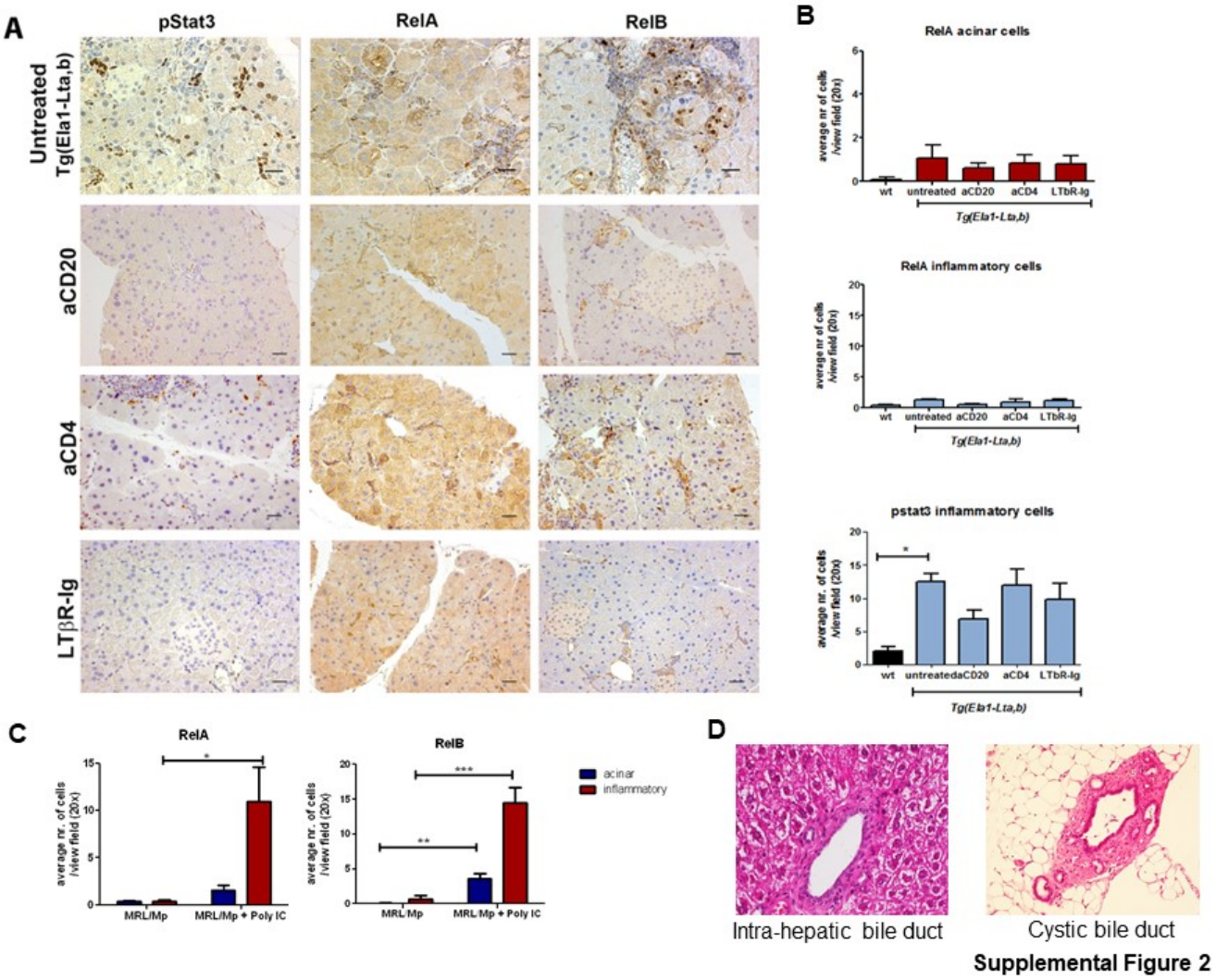


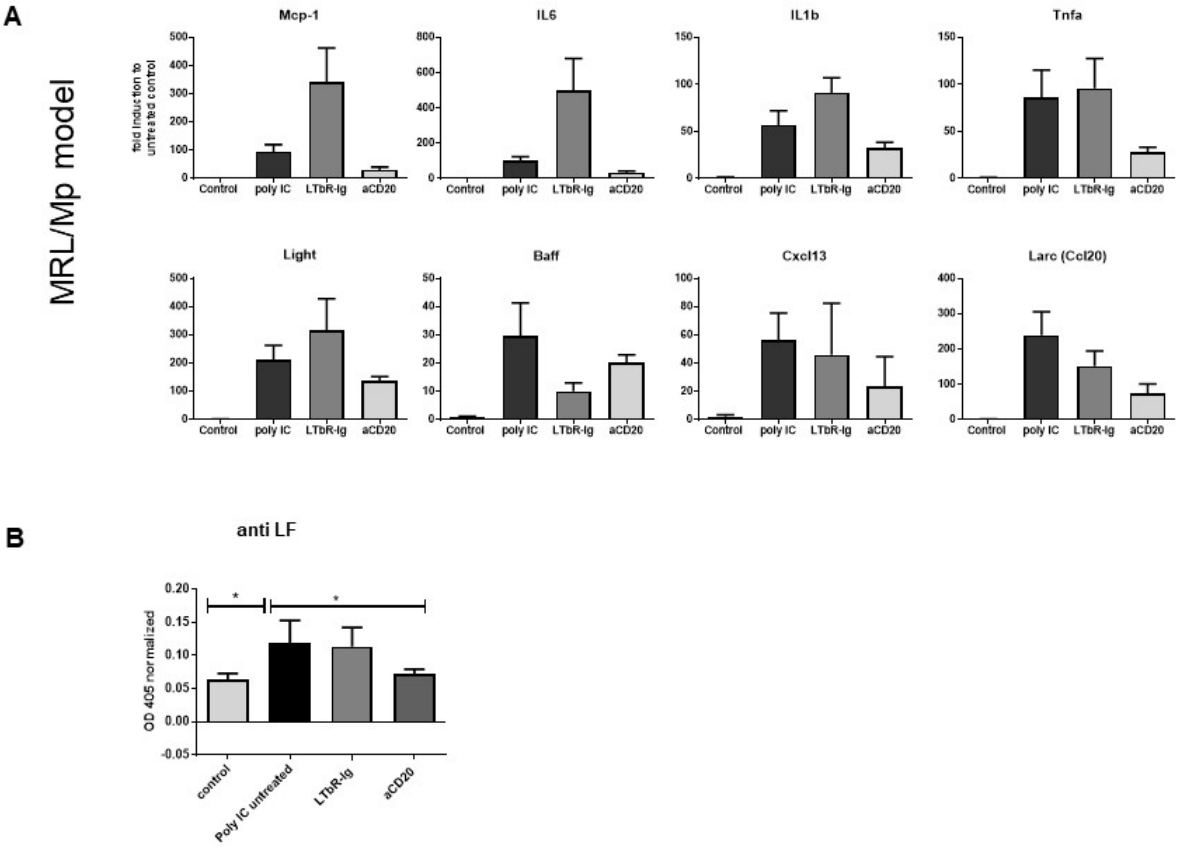
Supplemental Figure 1

Supplemental Figure 1: (A) Flow cytometry analysis of PBMCs of *Tg(Ela1-LTab)* mice after aCD20 aCD4 and LTbR-Ig treatment. The amount of CD20+ and CD4+ cells were detected in a CD45+ gated population. **(B)** Quantification of inflammatory cell proliferation based on counting of Ki67+ nuclei. **(C)** HE staining in untreated 12 months old *Tg(Ela1-LTab)* mice as well as after aCD20, aCD4 and LTbR-Ig treatment. Arrows indicate ADMs. **(D)** Distribution of infiltrating inflammatory (plasmacytoid dendritic cells, CD11+ dendritic cells, CD4 and CD8+ T-cells and CD19+ B-cells) assessed by flow cytometry analysis. The results are shown as percentage to CD45+ cells. **(E)** Co-Immunofluorescence staining of untreated 12 months old *Tg(Ela1-LTab)* with AIP and after treatment with LTbR-Ig. (Amylase red, CK19 green) Magnification 20x. **(F)** Quantitative RT-PCR of pancreatic Sox9 expression of untreated 12 months old *Tg(Ela1-LTab)* with AIP and after treatment with LTbR-Ig, aCD4 and aCD20.



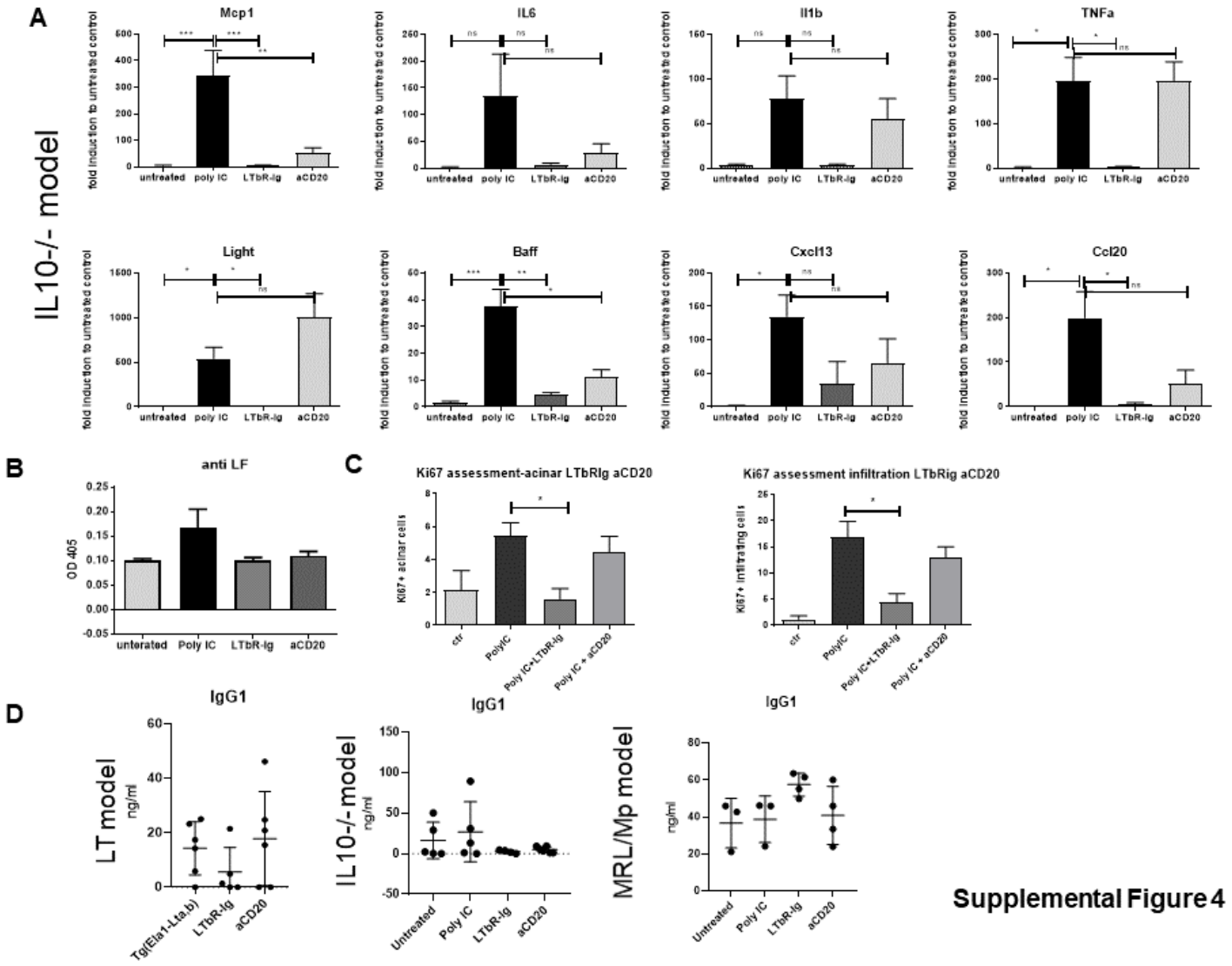
Supplemental Figure 2

Supplemental Figure 2: (A) Stat3 phosphorylation, RelA and RelB nuclear translocation was analysed by immunohistochemistry of pancreata in 12 months old Tg(ELa1-Lta,b) mice and in age matched C57BL/6 mice. Scale bar 100mm. **(B)** Quantification of RelA nuclear translocation in acinar cells and in infiltrating immune cells and Stat3+ inflammatory cells of 12 months old *Tg(Ela1-LTab)* untreated, aCD20 treated, aCD4 treated and LTbR-Ig treated mice and C57BL/6 controls. **(C)** Quantification of RelA and RelB nuclear translocation in acinar cells and in infiltrating immune cells in untreated MRL/Mp mice and in mice with induced AIP upon Poly IC treatment. **(D)** HE staining of untreated 12 months old *Tg(Ela1-LTab)* with AIP showing representative normal intra-hepatic (arrow) and cystic bile ducts.



Supplemental Figure 3

Supplemental Figure 3: MRL/Mp model. (A) Quantitative RT-PCR of pancreatic Mcp-1, Il6, Il1b, Tnfa, Light, Baff, Cxcl13, Ccl20 in untreated MRL/Mp mice (control) and in MRL/Mp mice treated with PolyIC (plus control IgG), PolyIC + LTbR-Ig and PolyIC + aCD20. **(B)** Auto-antibodies against lactoferrin were measured from the serum of untreated MRL/Mp mice and in MRL/Mp mice treated with, PolyIC + control IgG, PolyIC + LTbR-Ig and PolyIC + aCD20.



Supplemental Figure 4

Supplemental Figure 4: IL10-/- model. (A) Quantitative RT-PCR of pancreatic Mcp-1, Il6, Il1b, Tnfa, Light, Baff, Cxcl13, Ccl20 in untreated IL10-/- mice (control) and in IL10-/- mice treated with PolyIC (plus control IgG), PolyIC + LTbR-Ig and PolyIC + aCD20. **(B)** Auto-antibodies against lactoferrin were measured from the serum of untreated IL10-/- mice and in IL10-/- mice treated with PolyIC + control IgG, PolyIC + LTbR-Ig and PolyIC + aCD20. **(C)** Quantification of acinar cell and inflammatory cell proliferation based on counting of Ki67+ nuclei in IL10-/- mice treated with PolyIC + control IgG, PolyIC + LTbR-Ig and PolyIC + aCD20. **(D)** Quantification of IgG1 in 12 months old untreated, LTbR-Ig treated and aCD20 treated *Tg(Ela1-LTab)* mice as well as untreated IL10-/- mice, in IL10-/- mice treated with PolyIC + control IgG, PolyIC + LTbR-Ig and PolyIC + aCD20 and untreated MRL/Mp mice, in MRL/Mp mice treated with PolyIC (plus control IgG), PolyIC + LTbR-Ig and PolyIC + aCD20.