Supplementary Figure 1. NOD.\textit{Tnfrsf9}^-/- T cells do not have a defect to repopulate NOD.\textit{Rag1}^-/- recipients. Splenic CD4 and CD8 T cells from a subset of recipients were enumerated at diabetes onset or at the end of the incidence study. Each symbol represents one recipient mouse.
Supplementary Figure 2. CD137 expression in non-T and non-B cells does not influence T1D development. Purified NOD T cells (5x10^6) were injected intravenously into the indicated recipients. T1D development was followed for 25 weeks post-transfer. Diabetes incidence is not different between the two recipient groups.
Supplementary Figure 3. Intra-islet proliferation of CD4 T cells is comparable between NOD and NOD. Tnfrsf9⁻/⁻ strain mice. Ki-67 expression was analyzed by flow cytometry in islet infiltrating CD4 T cells from 10-14 week-old NOD and NOD. Tnfrsf9⁻/⁻ female mice. The results are summarized from 5 independent experiments. Each symbol represents islet cells pooled from 3-5 mice.
Supplementary Figure 4. Soluble CD137 suppresses CD8 T cell proliferation in a CD137 ligand dependent manner. NOD splenic CD8 T cells were cultured with CD3/CD28 beads and soluble CD137. In some wells as indicated, anti-CD137L blocking antibodies or IgG2a isotype control antibodies were added. Cell proliferation was measured by thymidine incorporation. CD137L blocking eliminated suppression of CD8 cell proliferation by soluble CD137 while isotype IgG2a did not (n=3 experiments). P values were calculated by unpaired T-test using Graphpad Prism. **: p<0.001