Supporting Information

Ng et al. 10.1073/pnas.1205850109

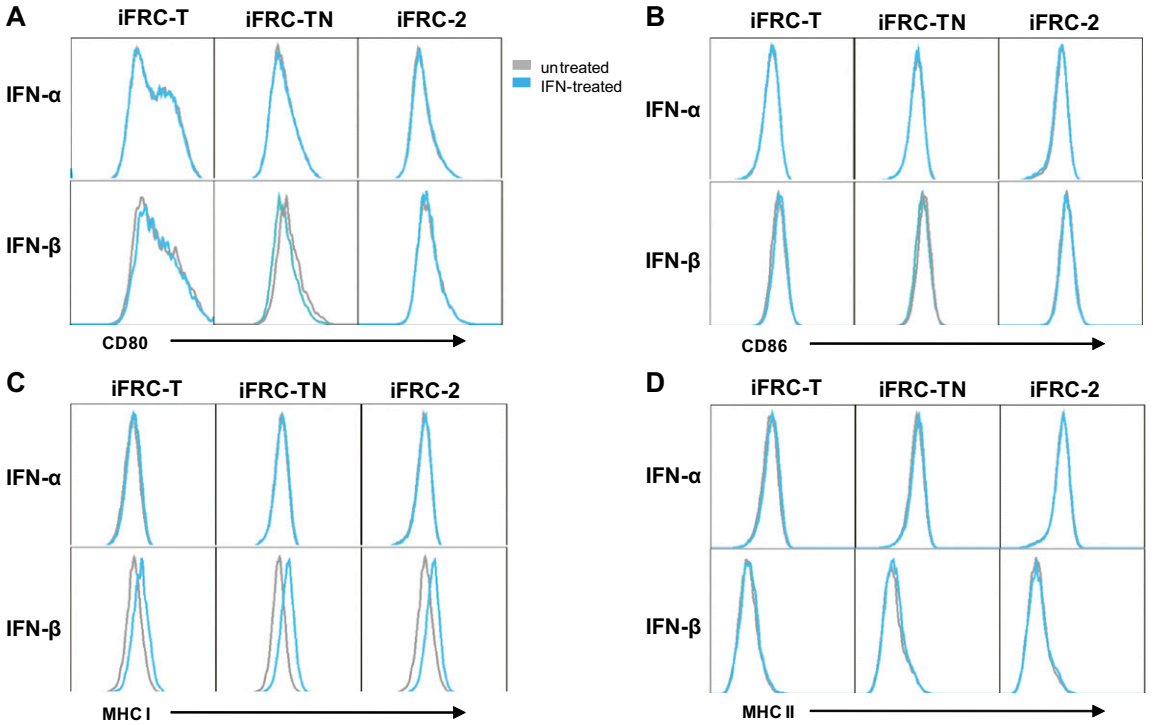

Fig. S1. iFRC expression of T-cell stimulatory ligands in the presence of type I IFN. iFRC expression of CD80 (*A*), CD86 (*B*), MHC I (*C*), and MHC II (*D*) in the presence and absence of IFN-α and IFN-β as assessed by flow cytometry.

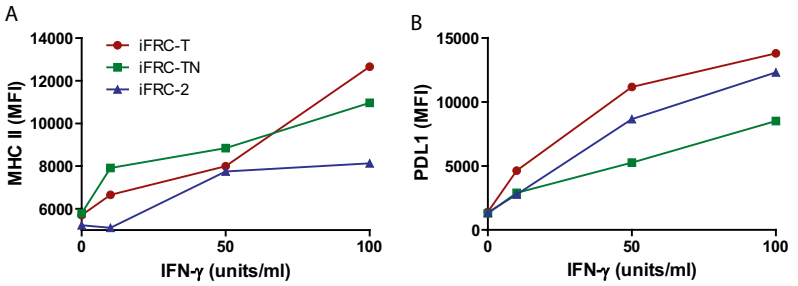

Fig. S2. MHC II and PD-L1 are up-regulated on iFRCs by IFN-γ in a dose-dependent manner. iFRCs were incubated with 0, 10, 50, or 100 U/mL of IFN-γ for 48 h and assessed for extracellular MHC II (*A*) and PD-L1 (*B*) by flow cytometry.

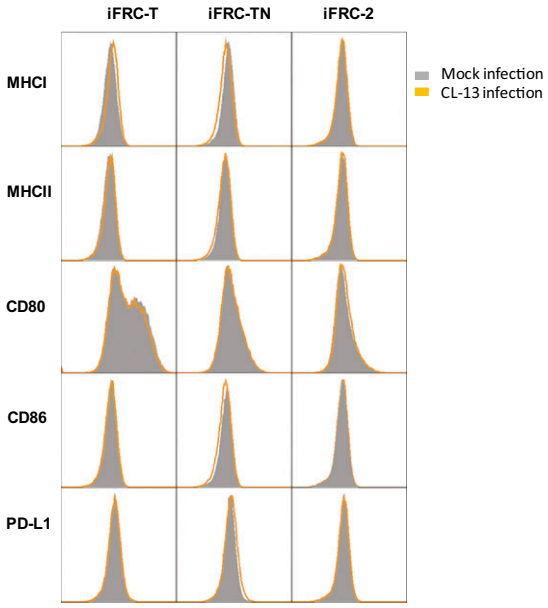

Fig. S3. CL-13 infection alone does not alter expression of T-cell stimulatory ligands on iFRCs. iFRCs were evaluated for changes in expression of T-cell ligands by flow cytometry 48 h after infection with CL-13 (MOI 1).

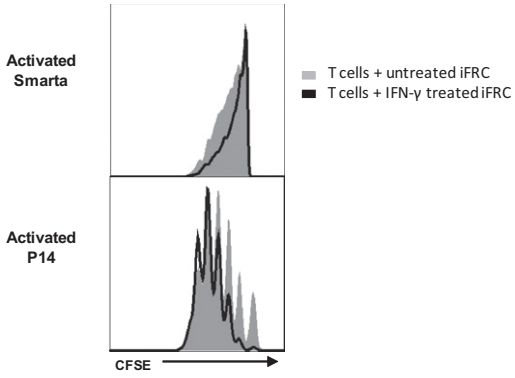

Fig. S4. IFN-γ treatment does not alter iFRC-mediated stimulation of activated CD4+ Smarta and CD8+ P14 T cells. Activated Smarta and P14 cells were stained with carboxyfluorescein succinimidyl ester (CFSE) and incubated with either mock or IFN-γ–treated iFRCs and LCMV peptide ($GP_{61–80}$ peptide for Smarta and $GP_{33–41}$ peptide for P14). IFN-γ treatment of iFRCs did not reduce the number of T-cell proliferation cycles in comparison with mock-treated iFRCs; however, mock-treated iFRCs were likely exposed to IFN-γ produced by the activated T cells.