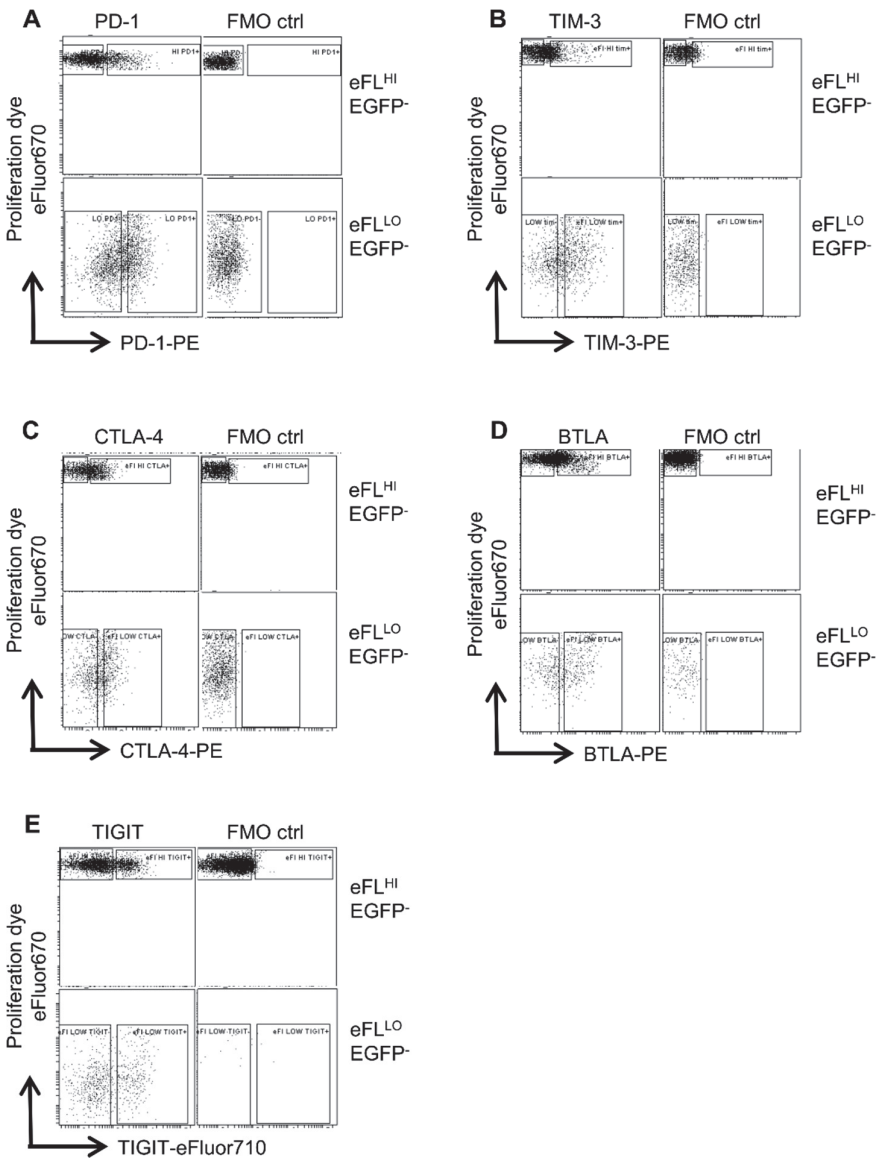

Figure S1. Sorting strategy to select for cells expressing high and low levels of IC molecules in non-proliferating and proliferating CD4$^+$ T cells. Resting CD4$^+$ T cells were cultured with syngeneic monocytes in the presence of SEB and infected with EGFP-reporter virus. At day 5 post-infection CD3$^+$HLA-DR$^-$ EGFP$^-$ (not productively infected), eFluor670^{HI} (non-proliferating) and eFluor670^{LO} (proliferating) T cells were further sorted based on IC molecule expression into low/negative (-) and high (+) expressing populations compared to FMO controls. Representative dot-plots of gating strategies for EGFP$^-$ eFluor670 high and low cells that express **A**: PD-1, **B**: TIM-3, **C**: CTLA-4, **D**: BTLA and **E**: TIGIT.

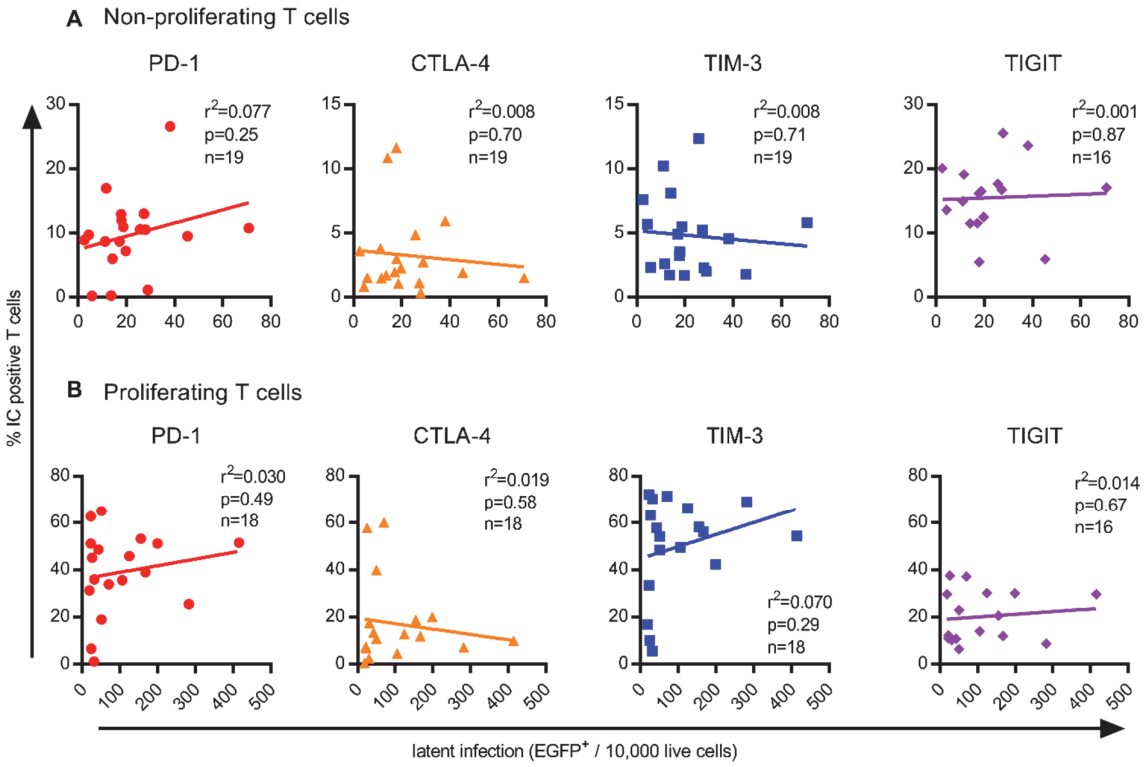

Figure S2. No correlation between IC expression and the level of latent infection in non-proliferating and proliferating T cells. Resting CD4$^+$ T cells were co-cultured with syngeneic monocytes in the presence of SEB and infected with EGFP-reporter HIV. At day 5 post-infection CD3$^+$HLA-DR$^-$ EGFP$^-$ non-proliferating (**A**) and proliferating (**B**) T cells were sorted and cultured in the presence of an integrase inhibitor with and without anti-CD3/CD28+IL-7+IL-2. On day 8, EGFP$^+$ cells were measured by flow cytometry and latent infection was calculated by subtracting the number of EGFP$^+$ cells in the unstimulated culture from the number of EGFP$^+$ cells in the stimulated culture. From the day 5 samples, an aliquot was taken prior to the sort and the CD3$^+$HLA-DR$^-$ EGFP$^-$ non-proliferating (A) and proliferating (B) T cells and were analyzed for PD-1 (red circles), CTLA-4 (orange triangles), TIM-3 (blue squares) or TIGIT (purple diamonds) expression by flow cytometry. Symbols represent individual donors. Correlation was determined using linear regression.

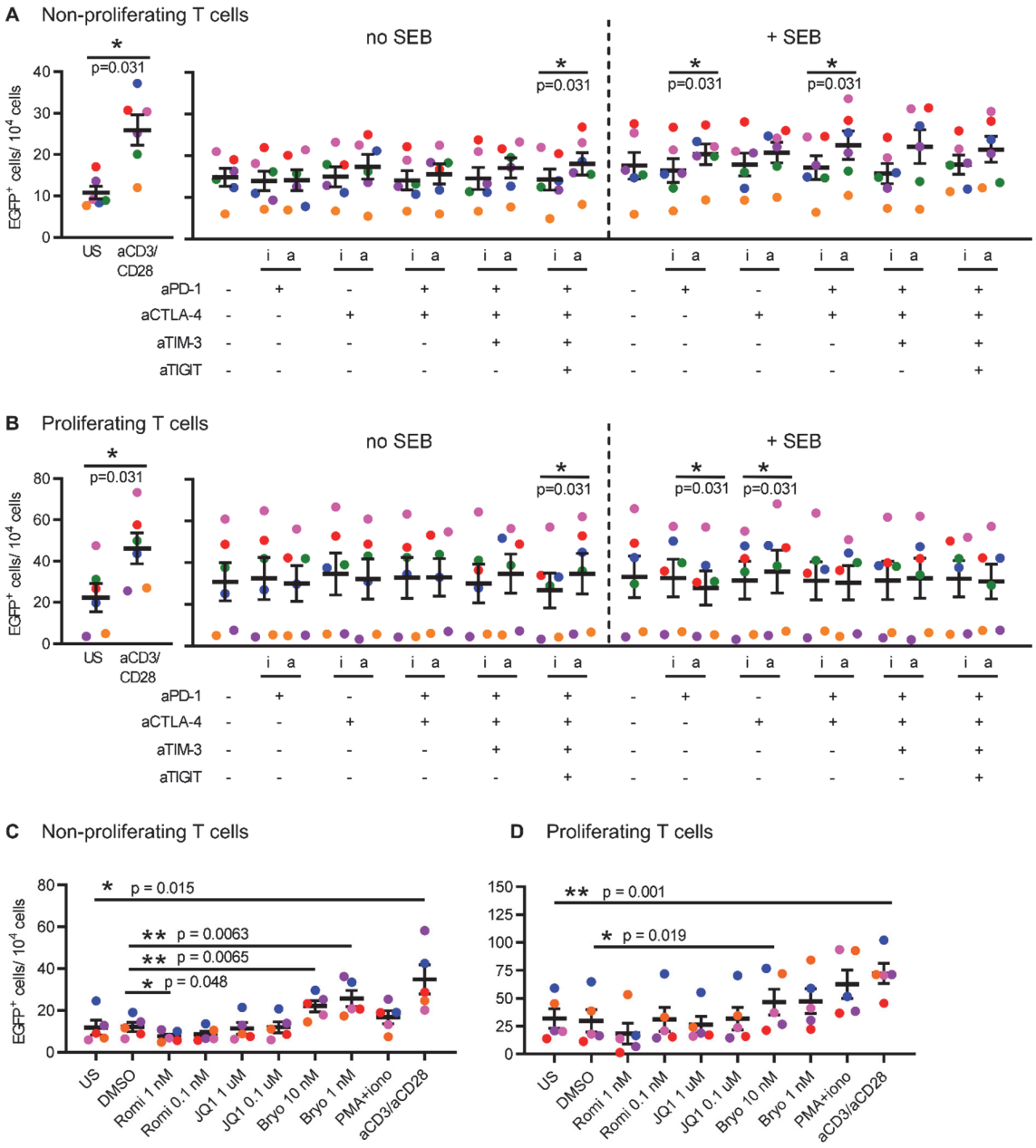

Figure S3. PD-1 blockade can reverse latent HIV but only in the presence of SEB or other IC blockers. To determine the effects of IC blockers (**A, B**) and different LRAs (**C, D**) on latency reversal in vitro, resting CD4$^+$ T cells were co-cultured with syngeneic monocytes in the presence of SEB and infected with EGFP-reporter HIV. At day 5 post-infection CD3$^+$HLA-DR$^-$ EGFP$^-$ non-proliferating (**A, C**) and proliferating (**B, D**) T cells were sorted. Sorted cells were cultured in the presence of integrase inhibitor with and without anti-CD3/CD28+IL-7+IL-2 or with an isotype (i) control or IC antibody (a) (**A, B**), or sorted cells were cultured with a panel of different LRAs (**C, D**). On day 8, EGFP$^+$ cells were measured by flow cytometry and latent infection was calculated by subtracting the number of EGFP$^+$ cells in the unstimulated culture from the number of EGFP$^+$ cells in the stimulated culture. Black lines indicate mean values ±SEM and equal colours represent equal donors across panels A-B (n=6) or C-D (n=5). *p<0.05, **p<0.01 as determined by Student's T test (n≤5) or Wilcoxon matched pairs signed rank test (n>5). US = unstimulated, aCD3/CD28 = anti-CD3/CD28+IL-7+IL-2 treated, i = isotype control, a = IC blocking antibody, Romi = romidepsin, Bryo = bryostatin.