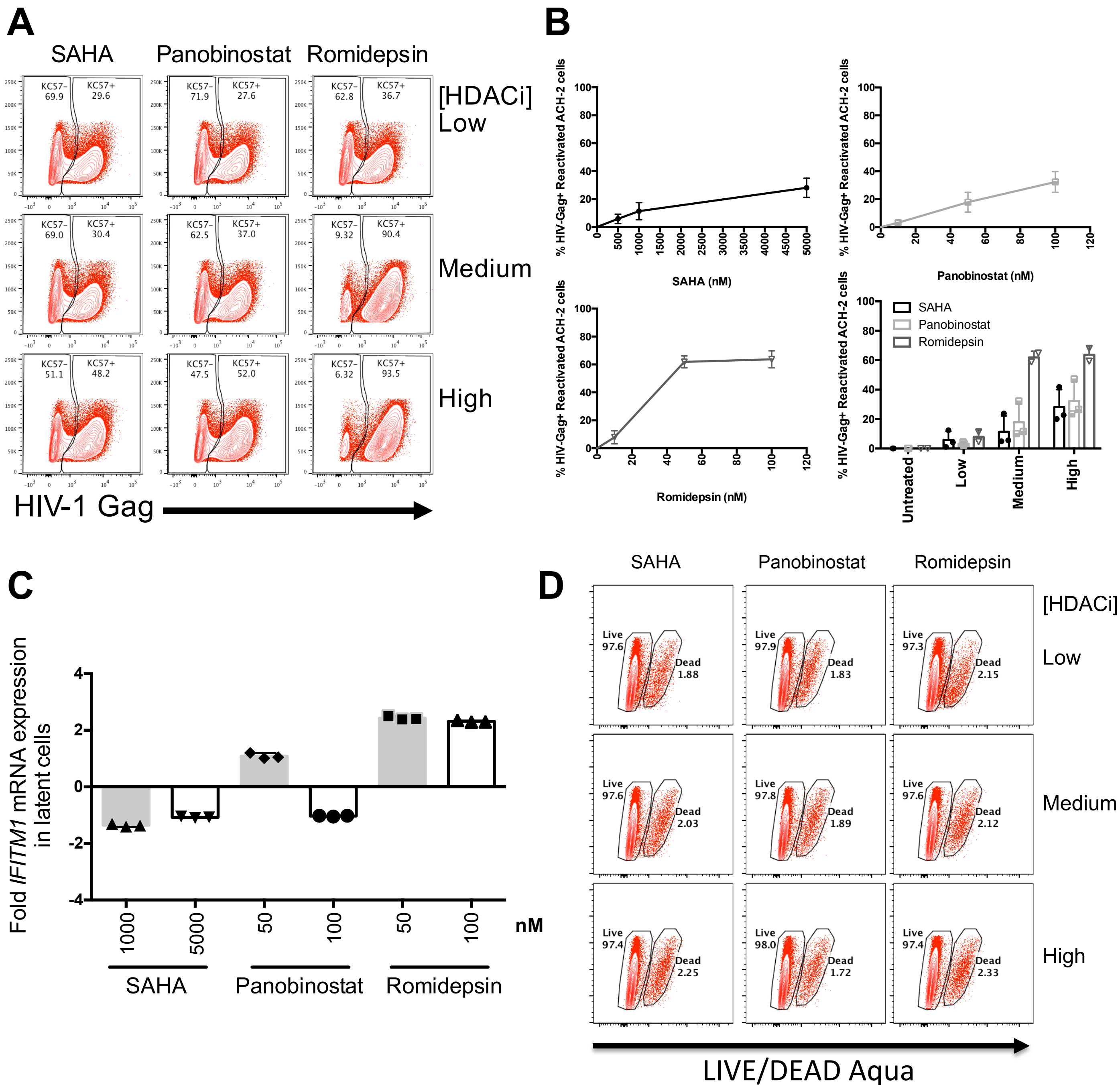

Supplemental Figure 1: Reactivation of HIV-1 in ACH-2 cells. **A)** ACH-2 cells were treated for 5h with increasing concentrations of SAHA (500, 1000 and 5000nM), panobinostat (10, 50, 100nM) and romidepsin (10, 20, 50nM). After the initial pulse, cells were washed twice in complete media and cultured for an additional 18h. The frequency of reactivated cells was determined by intracellular HIV-1 Gag staining. Representative flow plots of three independent experiments. **B)** Pooled data from three different experiments (n=3). Reactivated ACH-2 cells were calculated by subtracting the percentage of HIV-1 Gag+ cells by the percentage of HIV-1 Gag+ cells in untreated control. Data are plotted as mean ± SEM. **C)** Fold mRNA gene expression of IFITM1 in latent over reactivated cells (n=3). Data are plotted as mean + SD. Quantitative real-time PCR was performed using TaqMan probes and *UBC* was used identified as the most stably expressed gene across all conditions and was used for normalization of results by the comparative Ct method. **D)** Effect of different HDACi on ACH-2 cell viability. Representative flow plots of three independent experiments.

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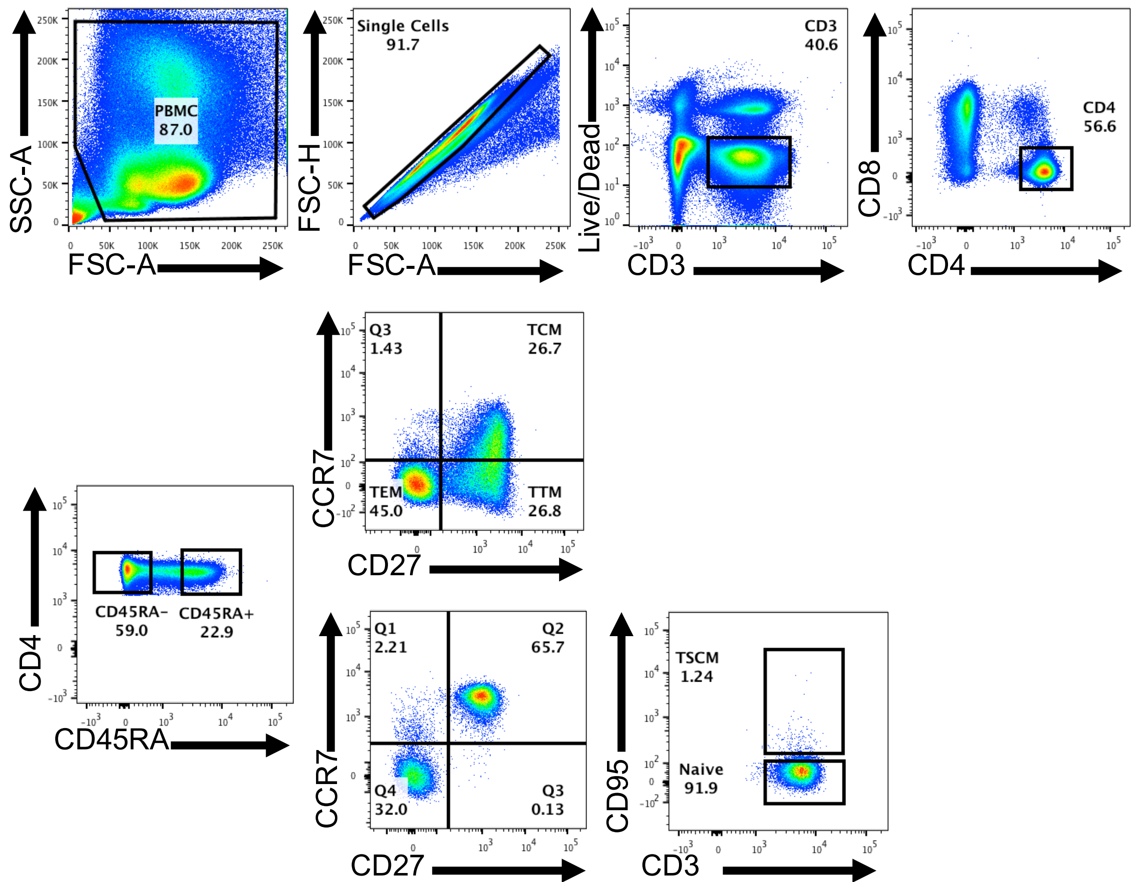

Supplemental Figure 2: Gating strategy for the detection of IFITM1 in different CD4+ T cell subsets. PBMC were stained with antibodies against CD3, CD4, CD8, CD45RA, CCR7, CD27, CD95 and IFITM1. Dead cells were excluded using a live/dead marker. Naïve cells: CD3+CD4+CD45RA+CD27+CCR7+CD95-; Stem central memory (TSCM): CD3+CD4+CD45RA+CD27+CCR7+CD95-; Central memory (TCM): CD3+CD4+CD45RA-CD27+CCR7+; Transitional memory (TTM): CD3+CD4+CD45RA-CD27+CCR7-; Effector memory (TEM): CD3+CD4+CD45RA+CD27-CCR7-.