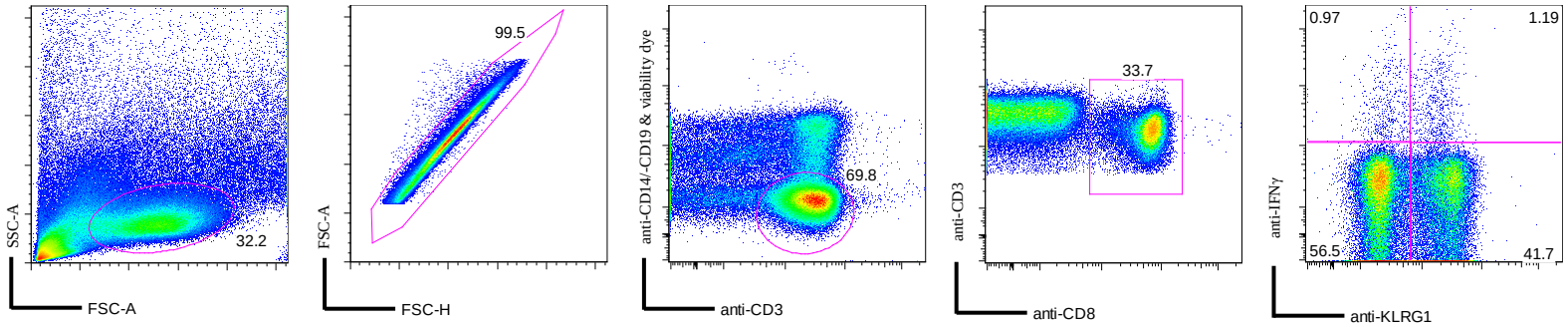
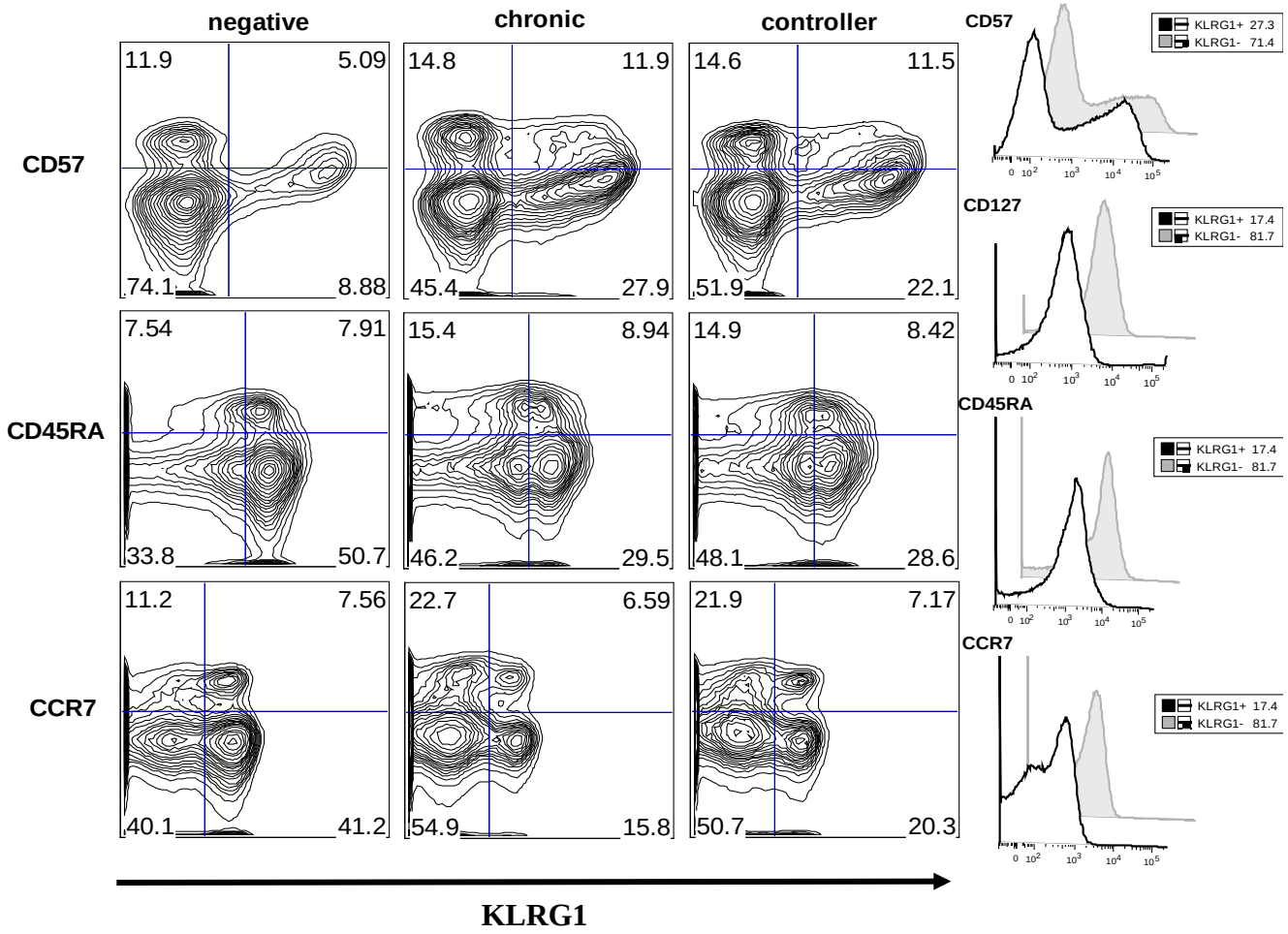


Figure S1. Representative gating strategy for IFNγ secretion of KLRG1+ or KLRG1- CD8+ T cells after Gag stimulation



- ***Figure S2. KLRG1+ HIV-1-specific CD8+ T cells have a effector/effector memory phenotype***
  Figure shows a concatenation of all tetramer+ CD8+ T cells and the expression CD45RA, CD127, CCR7 and CD57. KLRG1+ cells were predominantly CD127^{low}, CCR7^{low} and CD45RA$^{low/positive}$. The expression levels of KLRG1+ T cells were not substantially different among HIV-1 controllers, HIV-1 progressors and HIV-1 uninfected individuals.

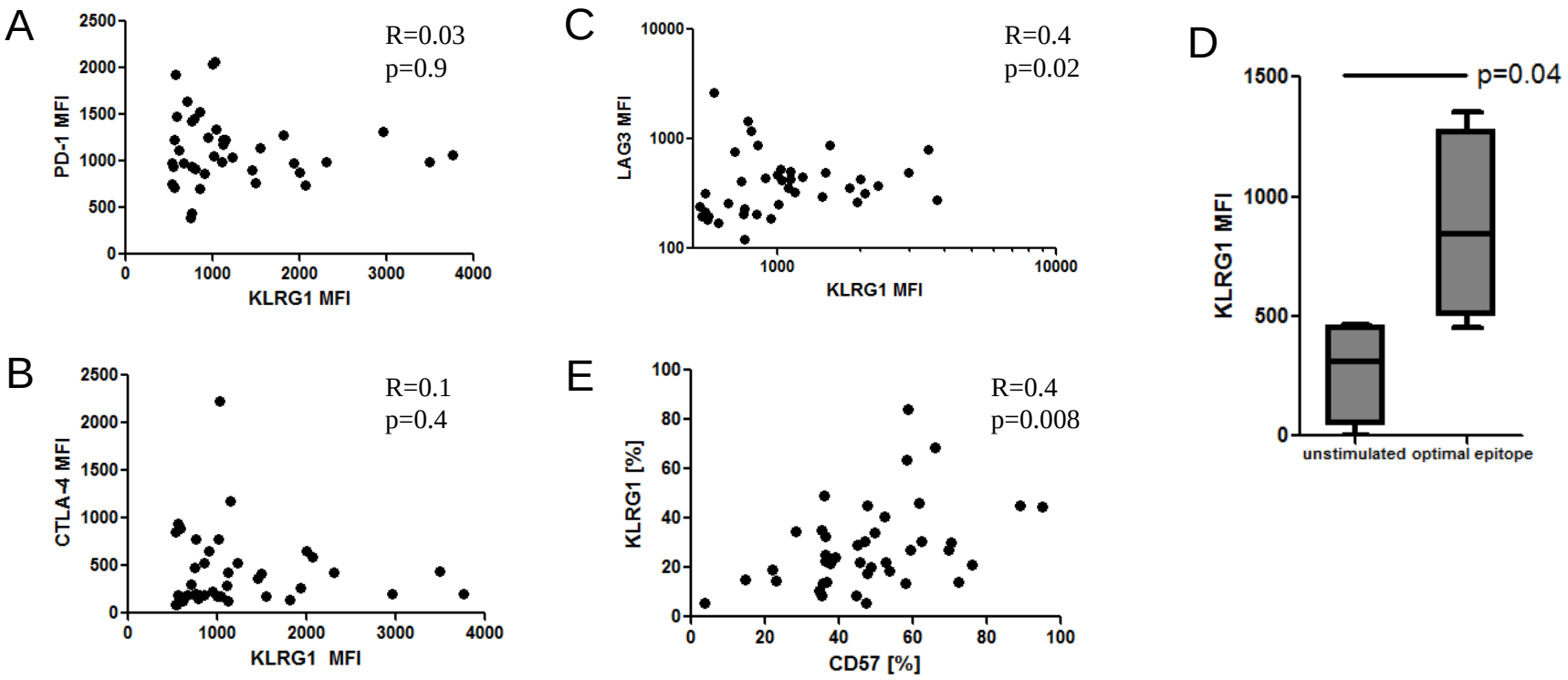


Figure S3. Co-expression of KLRG1 with other inhibitory receptors. HIV-1-specific CD8+ T cells were assessed for co-expression of KLRG1 and PD-1, CTLA-4, LAG3, and CD57. KLRG1 expression did not correlate with expression of either PD-1 or CTLA-4 (figures 2A and 2B) but did positively correlate with expression of LAG3 and CD57 (figure 2C and 2D).

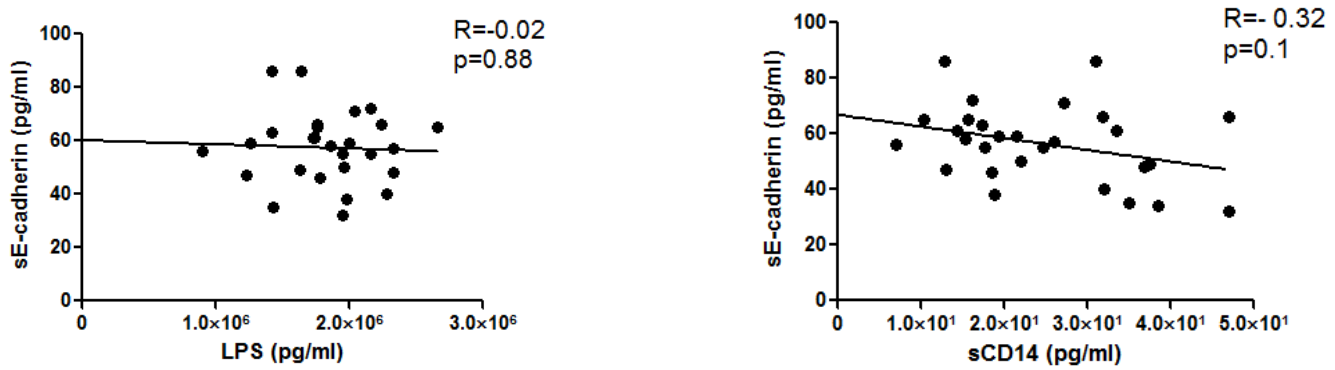


Figure S4. Correlation between LPS or sCD14 levels and soluble E-cadherin in plasma. LPS and sCD14 have been both described to be increased in blood with increased permeability of the intestinal membrane. However, only a weak correlation between sCD14 and sE-cadherin was observed suggesting different pathways of upregulation of these factors.

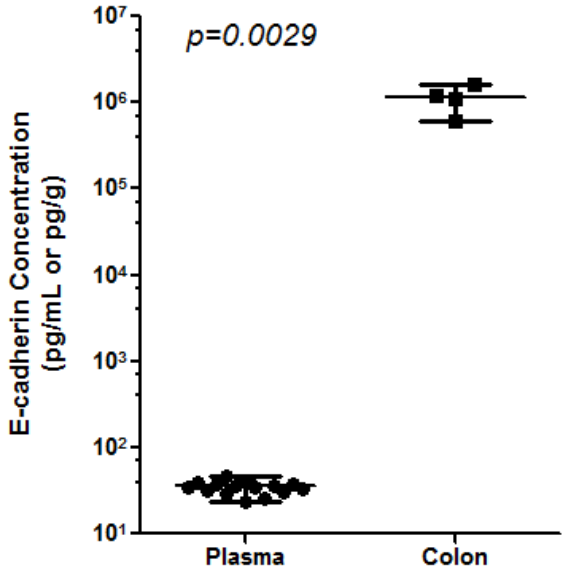


Figure S5. Distribution of Ecadherin leves in plasma and gastrointestinal tissue
To determine the relative level of E-cadherin in plasma and tissue, an ELISA was used to quantitate E-cadherin levels in the plasma from 15 HIV-1 uninfected donors compared to the levels found in homogenized colonic tissue biopsies samples from 4 HIV-1 negative donors. This showed significantly higher levels of E-cadherin within tissue ***(Fig. 4A, p=0.0029).***

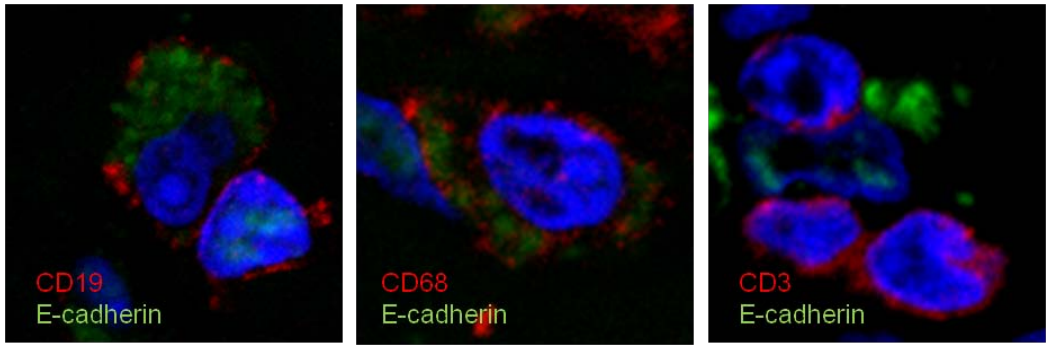


Figure S6. Confocal image analysis of localization of E-cadherin in the lamina propria. Sections of colonic mucosa were stained by immunofluorescence for E-cadherin and CD3, CD19 or CD68. E-cadherin staining was seen within CD19 and CD68 cells, but not CD3 suggesting uptake of sE-cadherin by B cells and monocytes/macrophages within the lamina propria.

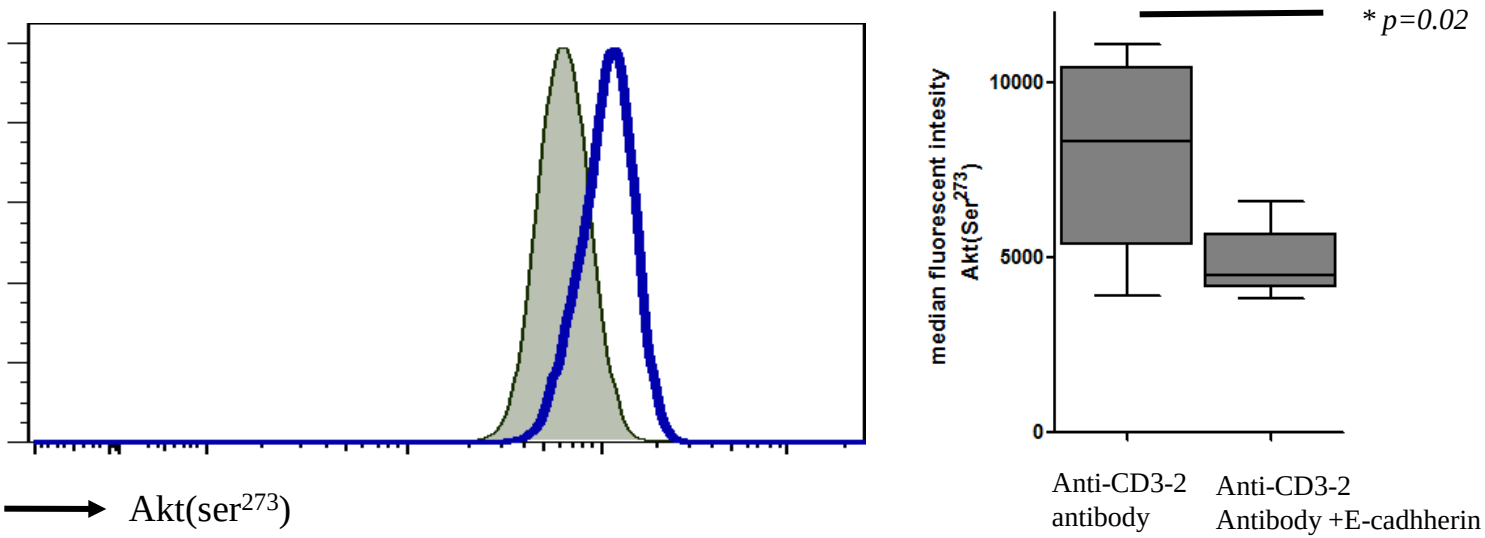


Figure S7. Signaling of sEcadherin through Akt(Ser^{273})

PBMCs of five HIV- subjects were stimulated with cross-linked anti-CD3 Ab in the presence or absence of soluble E-cadherin. Phosphorylation of Akt(Ser^{273}) was significantly downregulated in the presence of E-cadherin after stimulation as previously described.[45]