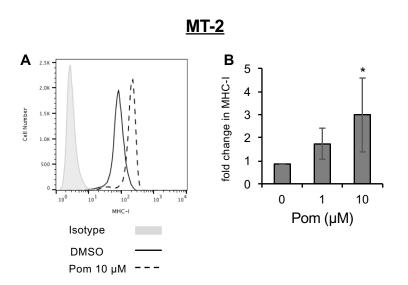
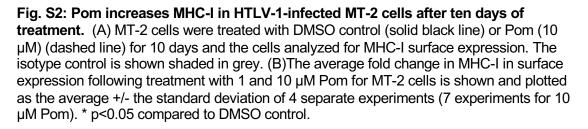


Fig. S1: Pom has little effect on MT-2 cell viability but inhibits cell growth based on live cell number after 3 and 10 days of treatment. MT-2 cells were treated with pomalidomide at 0, 0.5, 1.0 and 10  $\mu$ M. The viability (A and B) and live cell number (C and D) was then determined at 3 and 10 days after treatment. Viability (live cell number divided by total cell number x 100) shown in (A) and (B) was determined by Trypan blue cell counting. Live cell number (C and D) is expressed as percentage of the live cell number for control (DMSO) treated cells. MT-2 cell live number increased on average from 3x105 to 8x105 over a three-day period. Error bars denote the standard deviation for 3 or more experiments.





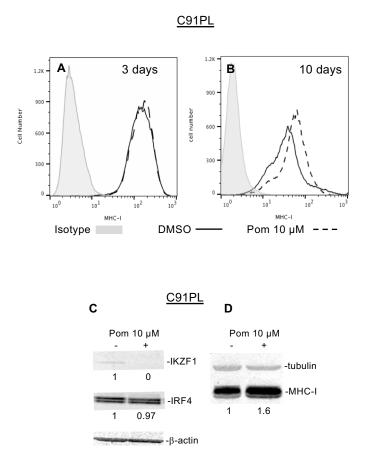
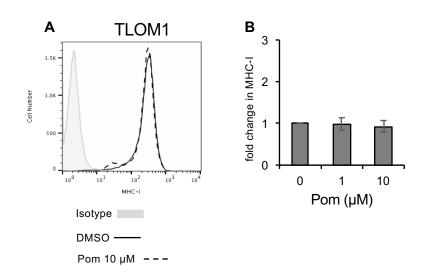
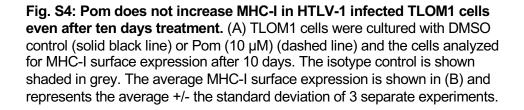


Fig. S3: 10-day treatment with Pom increases MHC-I surface and cytoplasmic expression but has little effect on IRF4 levels in C91PL cells. C91PL cells were treated with DMSO control (solid black line) or Pom (10  $\mu$ M) (dashed line) for 3 (A) and 10 (B) days and the cells analyzed for MHC-I surface expression. The isotype control is shown shaded in grey. Immunoblot for IKZF1, IRF4 and beta actin (C) and tubulin and MHC-I (D) from C91PL cells treated for 10 days without or with 10  $\mu$ M Pom. Relative protein levels were calculated based on the loading controls for IRF4 and MHC-I using the Licor system and are shown below the images.





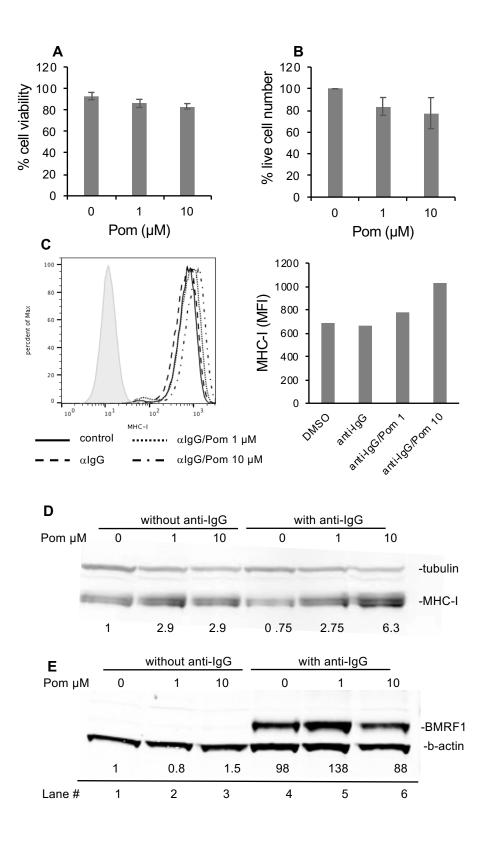
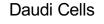
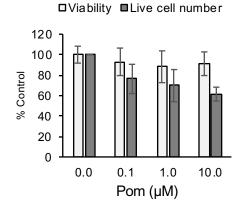


Fig. S5: Pom has little effect on ICAM-1 and B7-2 surface expression in Akata cells but increases MHC-I surface expression in lytically-induced Akata cells and cellular MHC-I in both latent and lytically-induced Akata cells. Akata cells were treated with DMSO control, or Pom at 1  $\mu$ M or 10  $\mu$ M for 48 h. Shown in (A) is the cell viability (live/total\*100) and in (B) the live cell number with values normalized to DMSO control cell number. Akata cell live number increased on average from 3x105 to 1x106 over the two-day period. Error bars denote the standard deviation for 3 experiments. (C) Cells were treated for 24 hrs with DMSO control or Pom at 1 or 10  $\mu$ M. The cells were then treated without or with anti-human IgG (50  $\mu$ g) for two days. Shown in (C) is the MHC-I surface expression in Akata cells pretreated without or with Pom for 24 hrs followed by anti-human IgG and Pom at 1 and 10  $\mu$ M for 48 h. Median values were 688, 660, 776 and 1034 for control, anti-IgG, Pom 1 $\mu$ M/anti-IgG, and Pom 10  $\mu$ M/anti-IgG, respectively. (D) Cytoplasmic lysates were prepared and the level of MHC-I was assessed by western blot with tubulin used as a loading control. The relative expression of MHC-I and BMRF1 are shown below each blot as compared to the respective loading control.





## Fig. S6: Effect of Pom on EBV-infected Daudi cells viability and live cell number. Daudi cells were treated for two days with DMSO control or Pom (0, 0.1 1, or 10 $\mu$ M). Cells were treated for two days and viability and live cell number was determined using trypan blue. Live cell number is shown as percent of DMSO control cells. Viability was calculated as live cell number over total cell number expressed as percent of DMSO control. Shown are the averages +/- the standard deviations of five independent experiments. Daudi live cell live number increased on average from 3x105 to 9x105 over the two-day period.

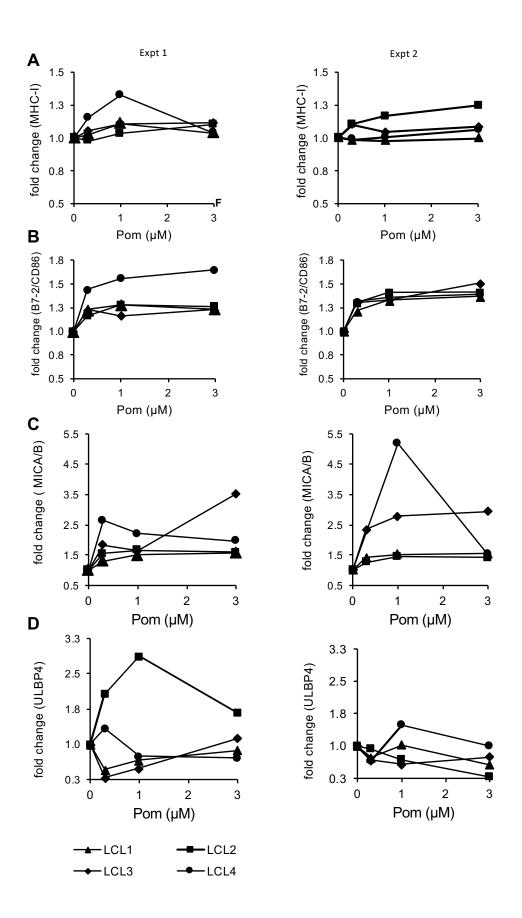
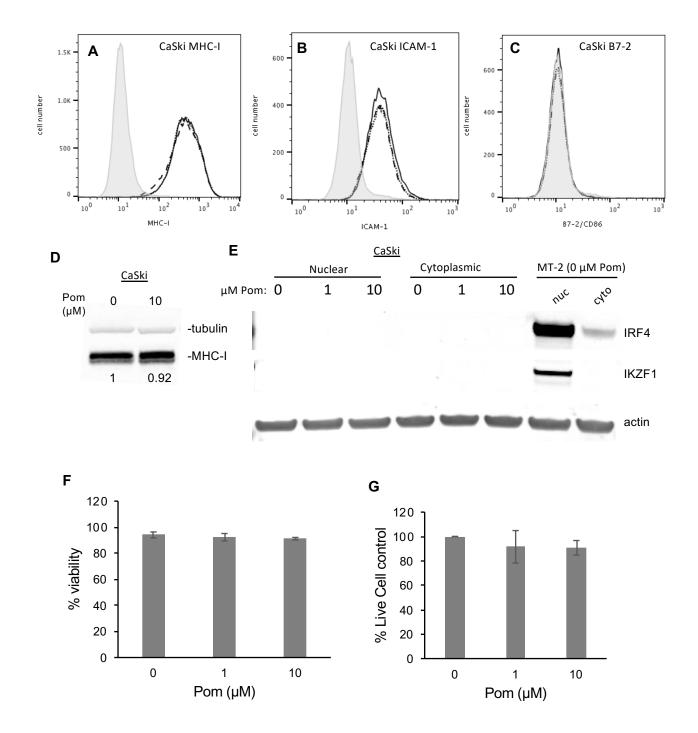


Fig. S7: Pom increases B7-2 and MICA/B in four different LCL clones. Four different LCL clones were plated at 2x105 cells per mI and treated with Pom at 0.3, 1 and 3  $\mu$ M. Three days later they were analyzed for MHC-I (A), B7-2 (B) MICA/B (C) and ULBP4 (D) expression by FACS. Analysis by FACS showed less than a 10% increase in dead cells in the presence of Pom. Shown are the results from two independent experiments for each surface marker. The 0.3  $\mu$ M data point for LCL4 was excluded in MICA/B analysis due to a technical error.



**Fig. S8: Pom does not affect MHC-I, ICAM-1, or B7-2 surface marker expression in CaSki cells.** CaSki cells were treated for two days with DMSO control or Pom (0, 1, or 10 μM). Shown are representative histograms from three independent experiments for MHC-I (A), ICAM-1 (B), and B7-2 (C) for DMSO (solid line), 1 μM Pom (dashed line), and 10 μM Pom (dotted line). The isotype control is shown in grey. (D) Western blot analysis for MHC-I expression in cytoplasmic lysates with tubulin as a protein loading control. The relative levels of MHC-I relative to DMSO-treated cells are indicated under the blot. (E) Western blot analysis of IKZF1 and IRF4 with beta actin as loading control; neither IKZF1 nor IRF4 are detectable in these cells. MT-2 cells were used as a positive control for detection of IRF4 and IKZF1. (F and G) Viability (live/total\*100) (F) and live cell number (G) was determined by Trypan blue staining. Results shown are the averages +/- standard deviations of three separate experiments. CaSki cell live number increased on average from 3x105 to 1.1x106 over a three day period.

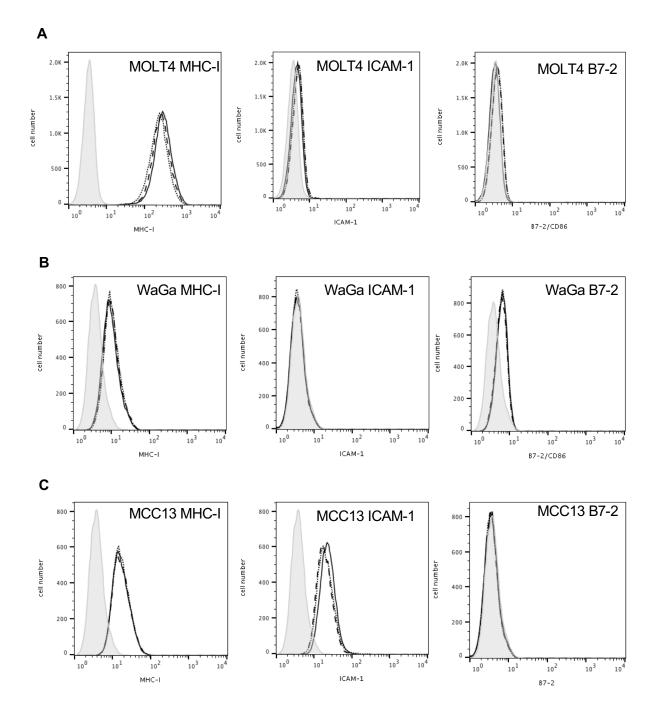


Fig. S9: Pom has little or no effect on MHC-I, ICAM-1, and B7-2/CD86 surface expression in HIV-1 and MCV- infected cells. HIV-1-infected MOLT4 T-cells (A) and MCV-infected WaGa cells (B) and MCC13 cells (C) were treated with DMSO control (solid black line), 1  $\mu$ M pom (dashed line), and 10  $\mu$ M pom (dotted line) and the cells analyzed for MHC-I, ICAM-1, and B7-2 surface expression after 10 days (MOLT4) or 3 days (WaGa and MCC13) of treatment. The isotype control is shown shaded in grey. Note that ICAM-1 expression in WaGa and B7-2 expression in MOTL4 and MCC13 cells are not detectable above isotype. Shown are representative histograms from 3 independent experiments. Cell viability was unaffected by Pom treatment and was higher than 96% under all conditions.