

Supplementary Figure 1. IL-15 is directly associated with IL-15R $\alpha$  in IFN- $\gamma$ -primed ECs and translocates out of the nucleus upon PRA treatment. IFN- $\gamma$ -primed ECs were treated with PRA sera or control gelatin veronal buffer (GVB) prior to fixation and permeabilization. Proximity ligation assay (PLA) was performed between intracellular IL-15 and IL-15R $\alpha$  or histone H1 and IL-15R $\alpha$  and analyzed by confocal microscopy. Scale bar, 30  $\mu$ m. Results shown are representative of two independent experiments.



Supplementary Figure 2. Densitometric analyses of immunoblots from Figure 2. Densitometric quantification of IL-15 and IL-15R $\alpha$  bands on exposed immunoblots from Figure 2C. Cytoplasmic (C), membrane (M), soluble nuclear (SN) and chromatin-bound nuclear (CN).



Supplementary Figure 3. Immunoblot analysis of whole cell lysates to confirm siRNA mediated protein knockdown and activation of ECs. A) IFN- $\gamma$ -primed ECs were transfected with control or NIK siRNA prior to PRA sera treatment. Cell lysates were assessed for NIK and cleaved caspase-1 expression by immunoblotting. B) IFN- $\gamma$ -primed ECs were treated with the cytokine LIGHT or IL-1 $\beta$ . Cell lysates were assessed for p100, p52 and p65 expression by immunoblotting. C) IFN- $\gamma$ -primed ECs were transfected with control, 100 or p65 siRNA prior to PRA sera treatment. Cell lysates were assessed for p100, p52 and p65 expression by immunoblotting. Representative of at least 3 independent experiments using 3 HUVEC donors.



Supplementary Figure 4. IFN- $\gamma$ -primed ECs activated by IL-1 $\beta$  or TNF $\alpha$  are induced to coordinately express surface IL-15/IL-15R $\alpha$ . ECs were pretreated with IFN- $\gamma$  for 48 hours prior to treatment with IL-1 $\beta$ , TNF $\alpha$  or mock treatment. Unpermeabilized cells were stained for IL-15 and IL-15R $\alpha$  and analyzed by flow cytometry. (n=3). Data represent mean  $\pm$  SEM. \*\*\*\*P<0.0001; NS, nonsignificant, 1-way ANOVA and Tukey multiple comparisons test. Representative of 2 independent experiments using 2 HUVEC donors.



Supplementary Figure 5. PRA treatment of IFN- $\gamma$ -primed human ECs augments the proliferation and activation of CD8+ T<sub>EM</sub>. Proliferation and activation of CFSE-labelled CD8+ TEM after coculture for 7 days with IFN- $\gamma$ -primed ECs treated with PRA or vehicle and in the absence of DMSO. Results shown are representative of at least three independent experiments using three different PBMC and HUVEC donors.



Supplementary Figure 6. CD8+ and CD4+  $T_{EM}$  cells do not express detectable levels of surface IL-15 and IL-15R $\alpha$ . CD8+ and CD4+ effector memory T ( $T_{EM}$ ) cells from PBMCs of a representative donor used for coculture experiments in Figure 4 were identified by gating on the CD8+CCR7- and CD4+CCR7- populations. CD8+ and CD4+  $T_{EM}$  were assessed for IL-15 and IL-15R $\alpha$  surface expression by flow cytometry. Results shown are representative of three different PBMC donors.



Supplementary Figure 7. Human ECs lining quiesced human coronary artery xenografts in graft recipients with no inoculation of allogeneic human PBMCs do no express detectable IL-15 and IL-15R $\alpha$ . Further analysis of human coronary artery grafts that were quiesced for 7 days and graft recipient mice were pretreated with either NLRP3 inhibitor MCC950 or control DMSO in PBS prior to either PRA or control sera injection (one mouse per group of four for each combination) and grafts were harvested 24 h later. Graft recipients were not inoculated with human PBMCs. PRA induced both mouse complement deposition and inflammasome assembly in the EC lining that inflammasome assembly could be blocked by MCC950. The same grafts were analyzed for IL-15 and IL-15R $\alpha$  staining by immunofluorescence. Scale bar, 50 µm. Results shown are representative of three artery grafts from three different artery donors.

Quiesced for 7 days prior to treatment No inoculation of recipient with allogenic PBMCs Retransplantation of treated grafts into recipient engrafted with allogeneic PBMCs



Supplementary Figure 8. T cell-secreted IFN- $\gamma$  levels correspond to the induction of HLA-DR expression by human ECs lining human coronary artery grafts in vivo. Human ECs lining artery xenografts from Figure 5 and Supplemental Figure 7 were analyzed for HLA-DR expression by confocal immunofluorescence. Scale bar, 30  $\mu$ m. Results shown are representative of three artery grafts from three different artery donors.



Supplementary Figure 9. Human IFN- $\gamma$  induces IL-15 and IL-15R $\alpha$  upregulation by human ECs lining human artery xenografts. A) Human coronary artery grafts from one donor were transplanted into immunodeficient mice and quiesced for 10 days prior to challenge with human IFN- $\gamma$ . Grafts were recovered after 72h. (n=3) B) Immunofluorescence detection of human endothelium by Ulex and HLA-DR expression in pre-transplant, IFN- $\gamma$  or PBS control treated human coronary artery grafts. Scale bar, 30 µm. C) Pre-transplant, IFN- $\gamma$  or PBS treated grafts were analyzed for IL-15 and IL-15R $\alpha$  expression by immunofluorescence microscopy. Scale bar, 30 µm. D) qRT-PCR analysis of IL-15, IL-15R $\alpha$  (normalized to CD31) and HLA-DR and caspase-1 (normalized to GAPDH) in the grafts (n=3). Data represent mean ± SEM. \*P<0.05, \*\*P<0.01, unpaired student t-test. Results shown are representative of three artery grafts from one artery donor.