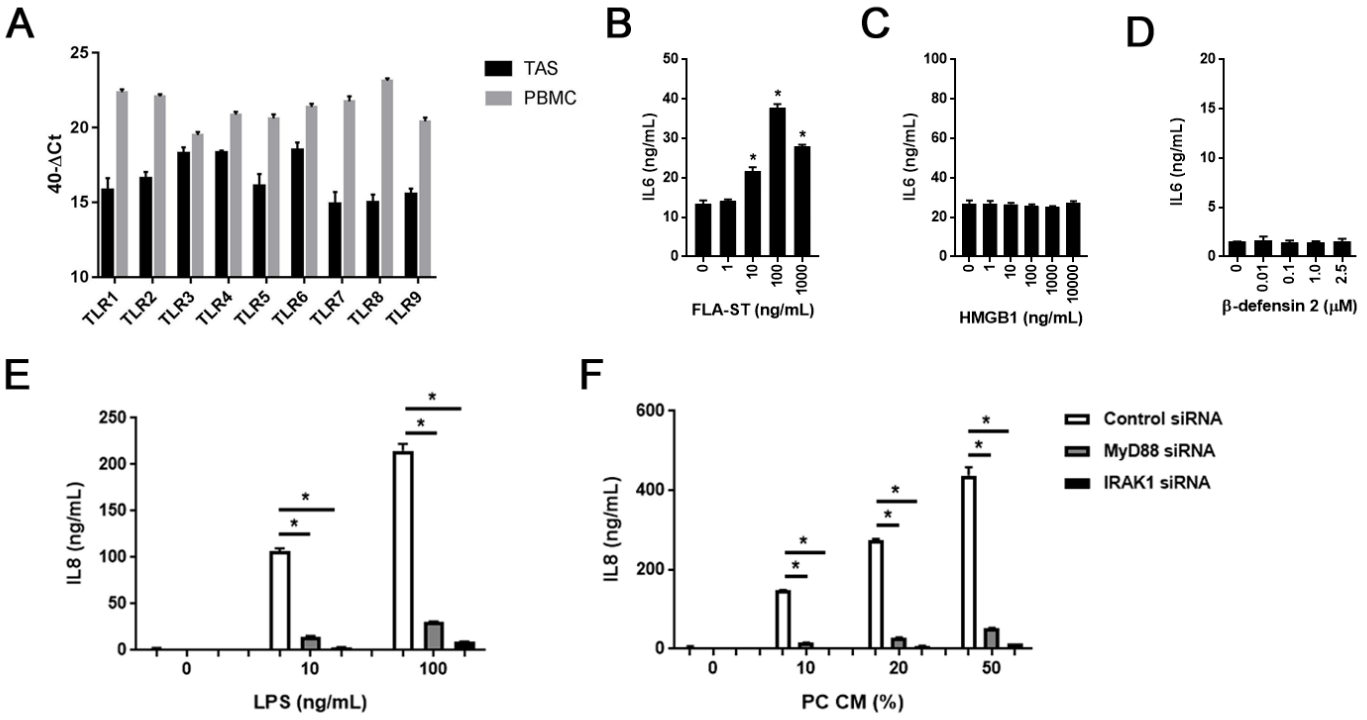
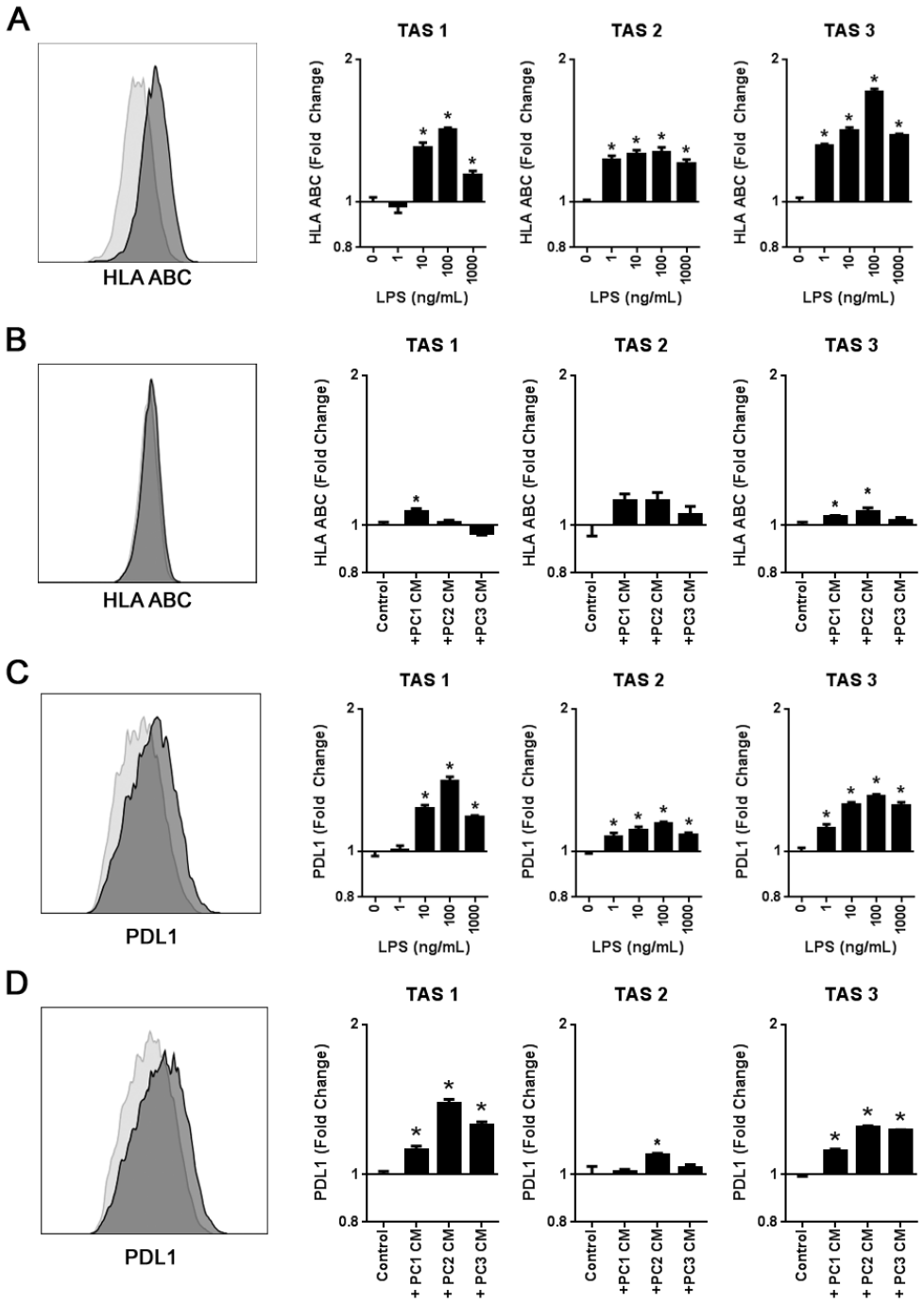
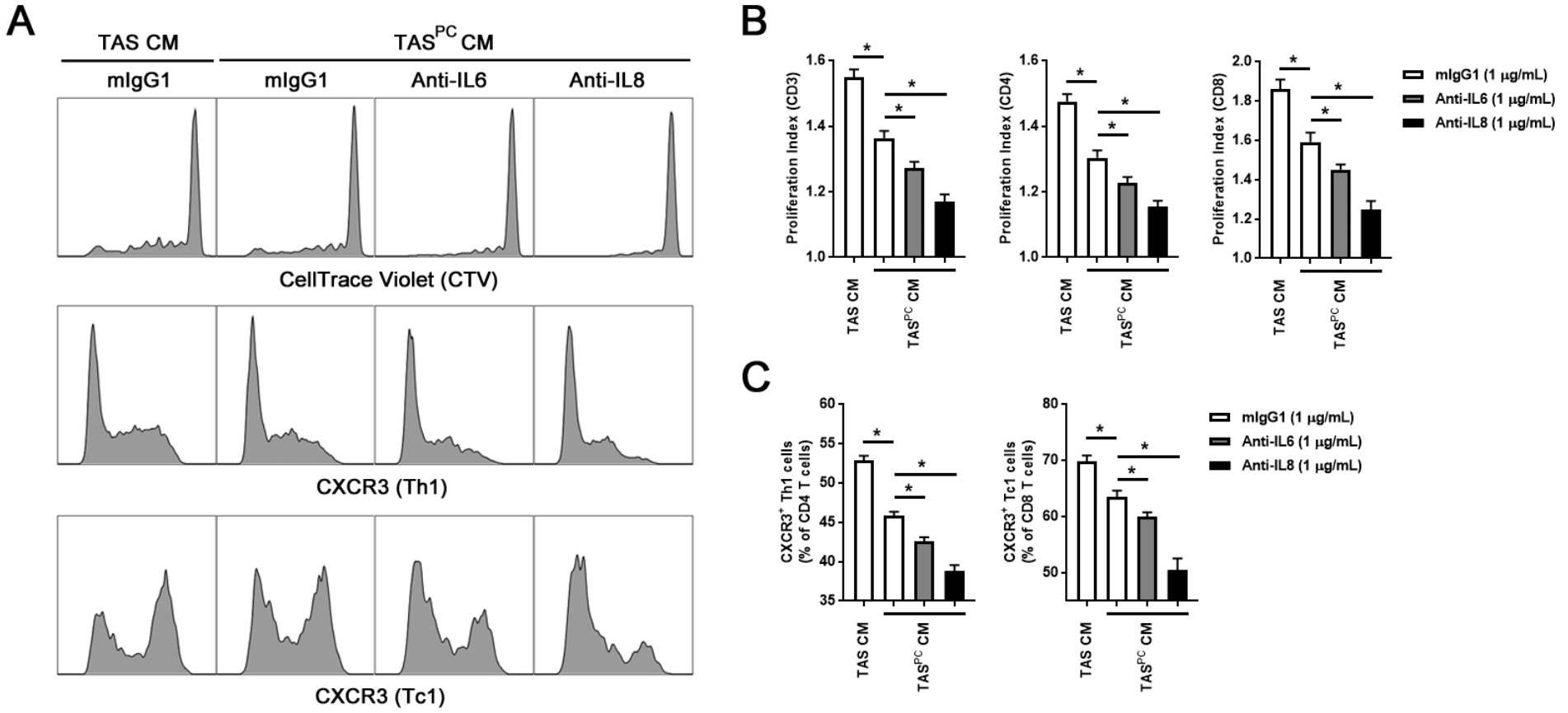


Supplemental Figure S1. Tumor-associated stromal response to pattern recognition receptor ligands. A, qPCR was performed on TAS in culture and peripheral blood mononuclear cells from a healthy donor. Data are expressed as 40 - (Ct_{TLR} - Ct_{18rRNA}). B-D, TAS cells were stimulated with (B) the TLR5 agonist, FLA-ST, previously demonstrated TLR4-binding DAMPs (C) HMGB1 or (D) β-defensin 2. IL6 secretion was then measured by ELISA. E and F, siRNA knockdown of MyD88 or IRAK1 was performed on TAS, which were then stimulated by (E) ultrapure LPS or (F) PC cell conditioned media (PC CM) as in **Fig. 4A and B**. IL8 secretion was measured by ELISA. Bars represent mean ± S.E.M. *$P < 0.05$ using the independent samples *t* test.



Supplementary Figure S2. Pancreatic cancer cell conditioned media and TLR4 ligation induces expression of antigen presentation machinery and negative co-stimulatory ligands on TAS. TAS cells were treated with either (A, C) 100 ng/ml of ultrapure LPS or (B, D) PCCM (50%) for 24 hours and surface expression of (A-B) HLA class I and (C-D) programmed death ligand 1 (PDL1) were analyzed using flow cytometry. Fold change represents the change in median fluorescent intensity of treated versus control groups. Representative histograms are shown for both unstimulated (light gray) and stimulated (dark gray) TAS. Bars represent mean ± S.E.M. *P < 0.05 compared to control using the independent samples t test.


A
TAS CM
TAS^PC CM
mIgG1
mIgG1
Anti-IL6
Anti-IL8
CellTrace Violet (CTV)
CXCR3 (Th1)
CXCR3 (Tc1)
B
Proliferation Index (CD3)
Proliferation Index (CD4)
Proliferation Index (CD8)
TAS CM
TAS^PC CM
mIgG1 (1 μg/mL)
Anti-IL6 (1 μg/mL)
Anti-IL8 (1 μg/mL)
C
CXCR3+ Th1 cells (% of CD4 T cells)
CXCR3+ Tc1 cells (% of CD8 T cells)
TAS CM
TAS^PC CM
mIgG1 (1 μg/mL)
Anti-IL6 (1 μg/mL)
Anti-IL8 (1 μg/mL)
D
Proliferation Index (CD3)
Th1 (% of CD4)
Control siRNA
TLR4 siRNA
NS
TAS CM
TAS^PC CM
TAS^PC CM

Supplemental Figure S3. TAS-mediated T cell suppression is enhanced by neutralization of either IL6 or IL8 and unaffected by TLR4 knockdown in TAS. TAS cultures were treated for 24 hours with PCCM (50%). Cells were then washed and returned to fresh growth medium for an additional 24 hours. Conditioned media from TAS was then transferred to magnetically sorted, CellTrace^{TM} Violet (CTV)-stained T cells from a healthy volunteer at a 1:4 dilution (20%) in growth medium. T cells were stimulated with anti-CD3/CD28/CD137 beads for four days in the presence of IL2 (50 U/mL). T cells were simultaneously treated with mouse IgG1 isotype control (1 µg/mL), mouse anti-hIL6 (1 µg/mL) or mouse anti-hIL8 (1 µg/mL) as indicated. A, representative histograms evaluating T cell proliferation via CTV dilution, Th1 and Tc1 skewing for TAS conditioned media (TAS CM)-treated T cells or PCCM pretreated (50% PCCM) TAS CM-treated T cells (TAS^{PC} CM). B, proliferation indices for total T cells (CD3), CD4, and CD8 T cells were calculated for each group. C, percentage of CD4 T cells with a Th1 phenotype and CD8 T cells with a Tc1 phenotype were quantified after stimulation. D, TAS cultures were treated with control siRNA or TLR4 siRNA prior to PCCM treatment and subsequent T cell stimulations. Proliferation indices were calculated for T cells as well as Th1 polarization. Bars represent mean $\pm$ S.E.M. *$P < 0.05$ using the independent samples *t* test.