**Supplemental Figure Legends**

**Supplementary Figure S1. Gating strategy to identify EpCAM-specific cells and their HLA-E expression.** (A) Identification of EpCAM-specific cells, by using dumping markers to exclude lymphoid-derived and myeloid-derived cells. (B) HLA-E expression (by MFI) on three different populations (EpCAMhigh tumor cells, EpCAMdim tumor cells and EpCAMdim paratumor cells) of esophageal carcinoma (n=3), gastric carcinoma (n=4) and colorectal carcinoma (n=3). \* indicates p-value<0.05, \*\* p-values<0.01, \*\*\* p-values<0.001, ns, not significant. p-values were calculated using one-way ANOVA with Tukey post hoc analysis. Data represent with medians.e.m.

**Supplementary Figure S2. Identification of different antigen-presenting cells subsets and associated surface expression from paired tumor, paratumor and PBMC.** (A) Gating strategy identifying myeloid-like population from paired tumor, paratumor and PBMC samples. (B) Gating strategy to identify each antigen-presenting cell subset populations, using several markers known to be expressed or absent in particular APC populations (for example: CD123 to identify pDC). (C) Representative histogram of MHC-Ia expression *(left panel)* and HLA-E expression *(right panel)* on CD141+ cDC from paired tumor, paratumor and PBMC from one of the colorectal cancer patients. (D) Comparative analysis of the HLA-E expression (by MFI) on CD141+ cDC, CD1c+ cDC, pDC and macrophages with the frequency of each antigen-presenting cells populations, from paired tumor, paratumor and PBMC samples. N, number of patients =22; (esophageal carcinoma, n=7, gastric carcinoma, n=7, colorectal carcinoma, n=7); two-way ANOVA with Tukey’ post hoc analysis. (E) Correlative analysis of HLA-E and MHC-1a expression on non-professional antigen-presenting cell subset, B cells from paired tumor, paratumor and PBMC. N, number of patients =22; (esophageal carcinoma, n=7, gastric carcinoma, n=8, colorectal carcinoma, n=7); correlation analysis using non-parametric Spearman test, p<0.05.

**Supplementary Figure S3. Similarities of CD94/NKG2a+ T cells and the maturation phenotype of CD94/NKG2a+ and CD94/NKG2a- CD8+ TILs *ex vivo.*** (A) The frequency of CD94/NKG2a+ CD8+ T cells from paired tumor, paratumor and PBMC from patients having either esophageal, gastric or colorectal cancers. N, number of patients=22 (esophageal carcinoma, n=7, gastric carcinoma, n=8, colorectal carcinoma, n=7). (B) The proportion of CD94/NKG2a+ CD8+ TILs *(left)*, CD94/NKG2a- CD8+ TILs *(right)* according to maturation phenotype expression of CD27, CD45RA and CCR7 in paratumor tissue *(top*) and PBMC *(bottom*). N, number of patients =22; (esophageal carcinoma, n=7, gastric carcinoma, n=8, colorectal carcinoma, n=7). Horizontal line represents median; interval represent 95% confidence. \* indicates p-value<0.05, \*\* p-values<0.01, \*\*\* p-values<0.001, ns, not significant. p-values were calculated using either one-way ANOVA or two-way ANOVA with Tukey post hoc analysis. Correlation analysis was performed by using non-parametric Spearman test. Data represent with medians.e.m.

**Supplementary Figure S4. Characterisation of NKG2a+ CD8+ T cells.** Gating strategy to identify co-expression of immune checkpoints (PD-1, KLRG-1 and BTLA) and of tissue-resident marker (CD103) on NKG2a+ CD8+ T cells from paired tumor, paratumor and PBMC.

**Supplementary Figure S5. *In vitro* functional setting and flow analysis of CD94/NKG2a+ and CD94/NKG2a- tumor-specific T cells.** (A) Histogram plot of HLA-E expression on HCT116, HLA-Ehigh-expressing BCL and HLA-EWT BCL. (B) Representative contour plots on IFNγ, TNFα and IL-2 expression on CD94/NKG2a+ and CD94/NKG2a- populations of HLA-A2-restricted TAA-specific CD8+ T cells following either aCD94/aNKG2a antibody blocking, isotype blocking or no blocking treatments, with or without 1μM antigen stimulation of HCT116.

**Supplementary Figure S6. Cytokine responses of CD94/NKG2a+ and CD94/NKG2a- tumor-specific T cells *in vitro* following HCT116 co-culture.** The proportion of TNFα+ cells of (A) CD94/NKG2a+ and CD94/NKG2a- populations of TAA-specific T cells, and of (B) CD94/NKG2a+ population of PBMC-derived TAA-specific T cells and tumor-derived HLA-A2-restricted CMV-specific T cells following antigen stimulation and after treatment with either aCD94/NKG2a blocking, isotype or no blocking treatments. (C) TNFα production following treatment for 48 hours on both CD94/NKG2a+ antigen-specific T cell lines. The proportion of MIP-1β+ cells of (D) CD94/NKG2a+ and CD94/NKG2a- populations of TAA-specific T cells, and of (E) CD94/NKG2a+ population of PBMC-derived TAA-specific T cells and tumor-derived HLA-A2-restricted CMV-specific T cells following antigen stimulation and after treatment with either aCD94/NKG2a blocking, isotype or no blocking treatments. The CCL-5 production of (F) CD94/NKG2a+ and CD94/NKG2a- populations of TAA-specific T cells, and of (G) CD94/NKG2a+ population of PBMC-derived TAA-specific T cells and tumor-derived HLA-A2-restricted CMV-specific T cells following antigen stimulation and after treatment with either aCD94/NKG2a blocking, isotype or no blocking treatments. Data were obtained from three independent experiments n=3. \* indicates p-value<0.05, \*\* p-values<0.01, \*\*\* p-values<0.001, ns, not significant. p-values were calculated using one-way ANOVA with Tukey post hoc analysis. Data represent with medians.e.m.

**Supplementary Figure S7. *Ex vivo* functional analysis of CD94/NKG2a-confirmed CD8+ TILs cell mixture.** Contour plots on IFNγ, TNFα and IL-2 expression on CD8+ TILs cell mixture following either aCD94/aNKG2a antibody blocking, isotype blocking or no blocking treatments, with or without 0.1μM SEB stimulation, data representation from one cancer patient.