Supplementary Figure 1. Neutrophil percentage and its potential correlations with clinical parameters. Neutrophil percentage in $CD45^+$ leukocytes was analyzed for correlations with clinical pathological parameters. *p<0.05, **p<0.01, n.s p>0.05 for groups connected by horizontal lines. Each dot represents one patient. CEA, carcinoembryonic antigen; *H.pylori* Ab, *Helicobacter pylori* antibody.

Supplementary Figure 2. Tumour environment sustains neutrophil survival. (A and B) Detection of deoxyuridine triphosphate nucleotides $(dUTP)^-$ viable non-apoptotic neutrophils exposed to 50% TTCS and 50% NTCS from autologous GC patients (A); or neutrophils to exposed to 20%, 40% or 80% TTCS from GC patients for 16 h (B). (C) Expression of HLA-DR on neutrophils exposed to 50% TTCS and 50% NTCS from autologous GC patients (3 pairs of TTCS and NTCS from 3 independent GC patients) for 12 h. black, isotype control. (D) Tumour-infiltrating $PD\text{-}L1^+CD54^+$ neutrophil percentage in neutrophils among TNM stages was compared. **p<0.01 for groups connected by horizontal lines.

Supplementary Figure 3. Tumour-infiltrating neutrophils interact with T cells in GC tumours. (A) Representative analysis of $CD15^+$ (green) neutrophil and $CD3^+$ (red) T cell interactions in tumour tissues of GC patients by immunofluorescence. Scale bars: 20 microns. (B) CFSE-labelled peripheral $CD3^+$ T cells of patients with GC were co-cultured for 5 days with autologous neutrophils from non-tumour or tumour tissues with or without anti-PD-L1 antibody. Representative data and statistical analysis of T cell proliferation were shown (n=5). (C) Wright staining of sorted tumour-infiltrating neutrophils was shown. (D) CFSE-labelled peripheral $CD3^+$ T cells of donors were co-cultured for 5 days with TTCS-conditioned allogeneic blood neutrophils with or without PD-L1 neutralizing antibody, nor-NOHA, or 1400W. Representative data and statistical analysis of T cell proliferation and IFN-γ production were shown (n=5). *p<0.05, **p<0.01 for groups connected by horizontal lines.

Supplementary Figure 4. Tumour-conditioned neutrophils suppress $CD4^+$ and $CD8^+$ T cell immunity through PD-L1. (A and B) CFSE-labelled peripheral $CD4^+$ T cells of donors were co-cultured for 5 days with TTCS-, or NTCS-conditioned autologous (A) or allogeneic (B) blood neutrophils with or without anti-PD-L1 antibody. (C and D) CFSE-labelled peripheral $CD8^+$ T cells of donors were co-cultured for 5 days with TTCS-, or NTCS-conditioned autologous (C) or allogeneic (D) blood neutrophils with or without anti-PD-L1 antibody. Representative data and statistical analysis of T cell proliferation and IFN-γ production were shown (n=5). *$p<0.05$, **$p<0.01$ for groups connected by horizontal lines.

Supplementary Figure 5. Expression of PD-L1 on neutrophils after stimulation. (A) Clustering of microarray data for the expression of 50 pro-inflammatory cytokine genes in human tumour tissues from 10 GC patients. (B) Expression of PD-L1 on neutrophils exposed to M-CSF, G-CSF, IFN-γ, TGF-β, IL-1β, IL-6, IL-17A, IL-23, IL-22, IL-10 (100 ng/ml) for 12 h. black, isotype control. (C) Expression of PD-L1 on neutrophils exposed to 50% TTCS with or without BAY 11-7082 (an lκBα inhibitor), SP600125 (a JNK inhibitor), SB203580 (a MAPK inhibitor), or Wortmannin (a PI3K inhibitor) for 12 h. black, isotype control. (D) Expression of GM-CSF receptor (GM-CSFR) on tumour-infiltrating neutrophils. black, isotype control. (E) Representative analysis of GM-CSF-expressing (green) $EpCam^+$ tumour cells (red) in tumour tissues of GC patients by immunofluorescence. Scale bars: 20 microns. (F) The correlations between GM-CSF and neutrophils in human tumours were analyzed. Results are expressed as percentage of neutrophils in $CD45^+$ cells or the number of neutrophils per million total cells and GM-CSF concentration in tumour tissues.

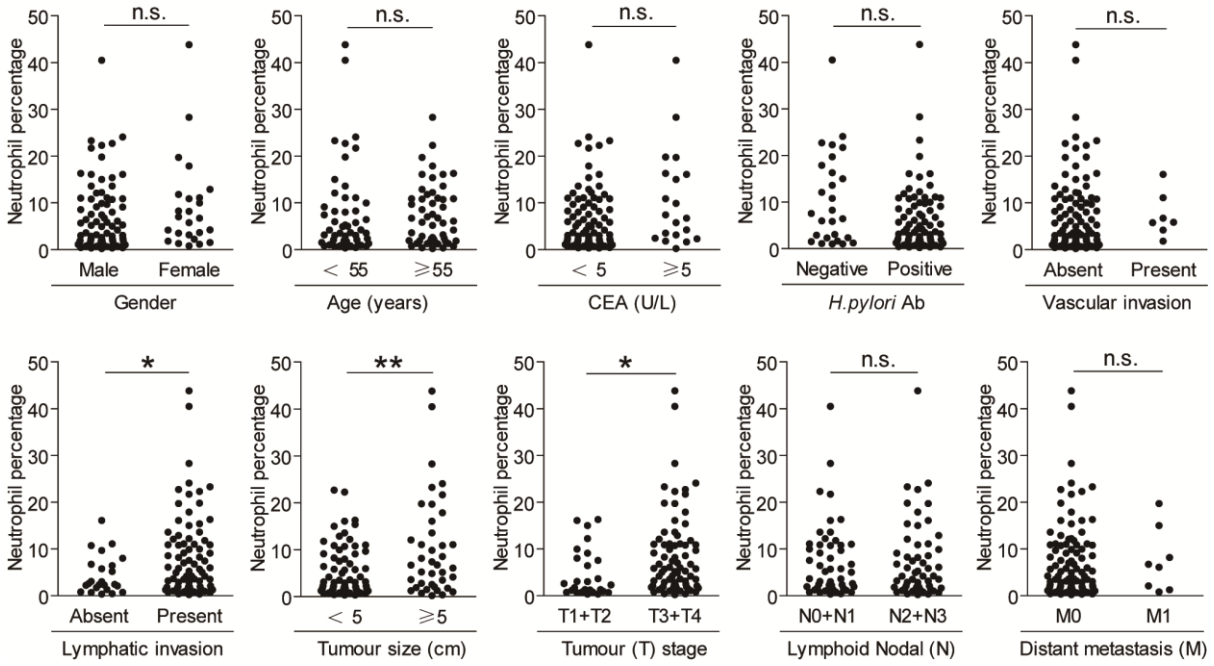
Supplementary Figure 6. Blockade of neutrophil-associated PD-L1 on T cell immunity inhibits tumour growth and GC progression *in vivo*. (A and B) The expression (A) and the production (B) of anti-tumour molecules perforin and granzyme B in tumours of mice injected with T cells in

combination with TTCS-conditioned neutrophils (TCN), or TCN pre-treated with an anti-PD-L1 antibody or a control IgG on day 24 after tumour cell injection were compared. *p<0.05, **p<0.01 for groups connected by horizontal lines.

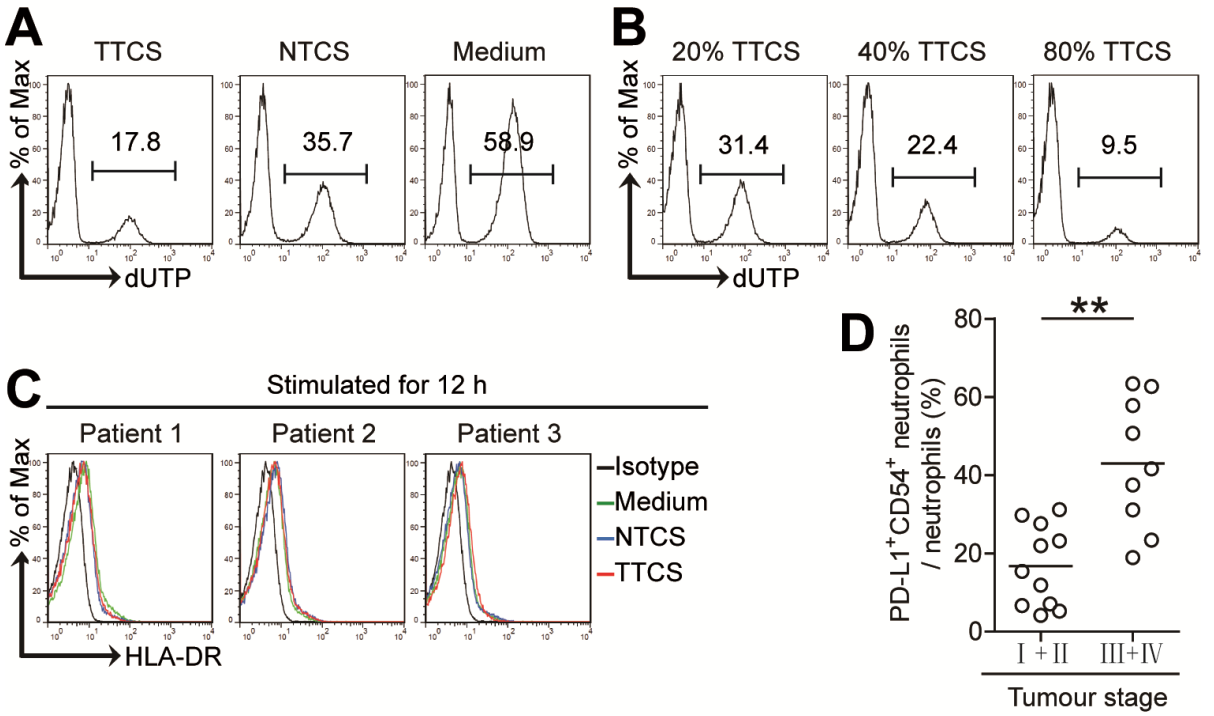
Supplementary Figure 7. Tumour-activated neutrophils suppress T cell immunity through PD-L1-PD-1 interaction. (A and B) CFSE-labelled FACS-sorted peripheral PD-1$^-$ T cells of donors were co-cultured for 5 days with TTCS-, or NTCS-conditioned autologous (A) or allogeneic (B) blood neutrophils with or without anti-PD-L1 antibody. Representative data and statistical analysis of T cell proliferation and IFN-γ production were shown (n=3). (C) PD-L1 expression on tumour-infiltrating neutrophils among TNM stages was compared. MFI: mean fluorescence intensity. **p<0.01 for groups connected by horizontal lines.

Supplementary Figures

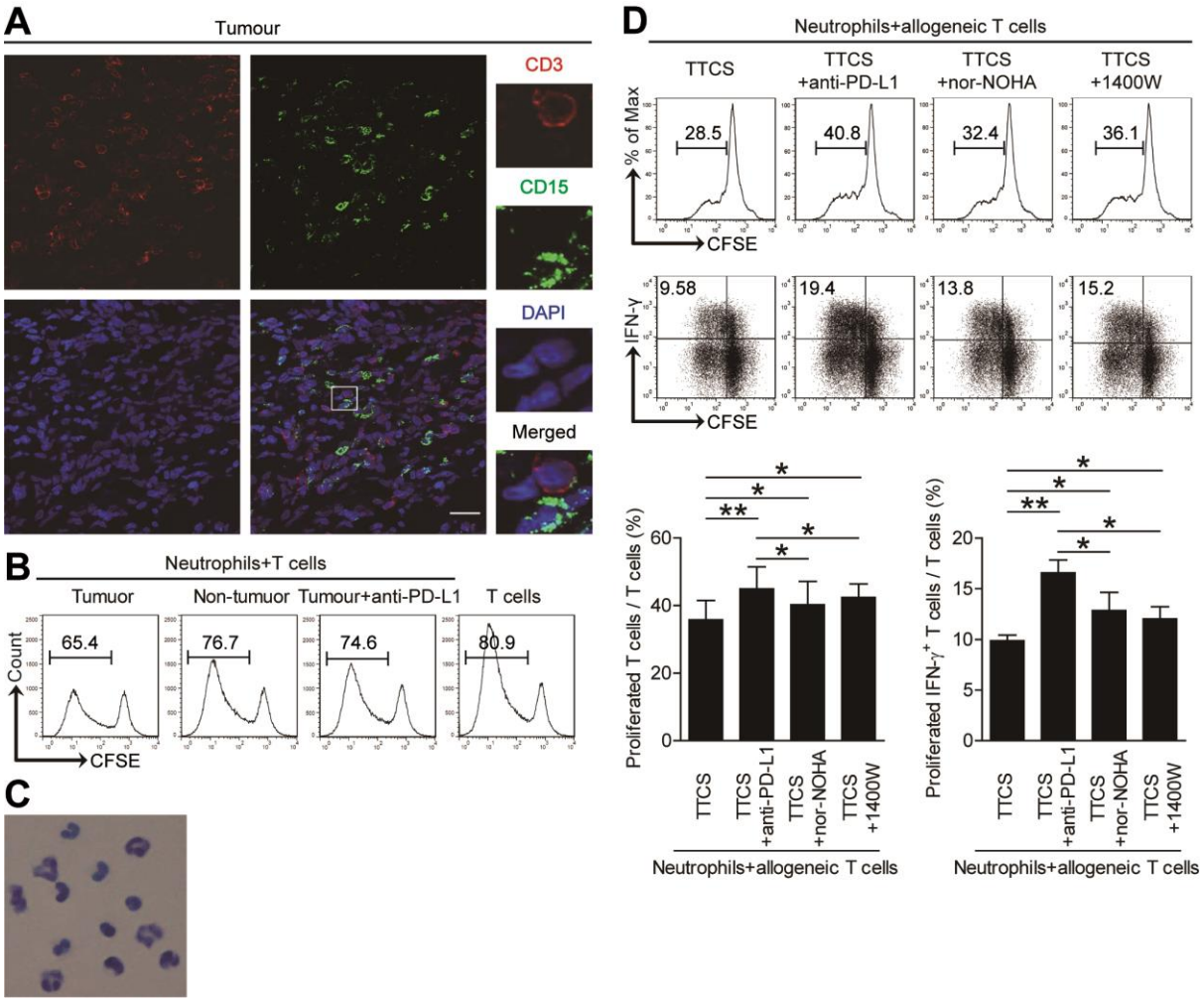
Supplementary Figure 1



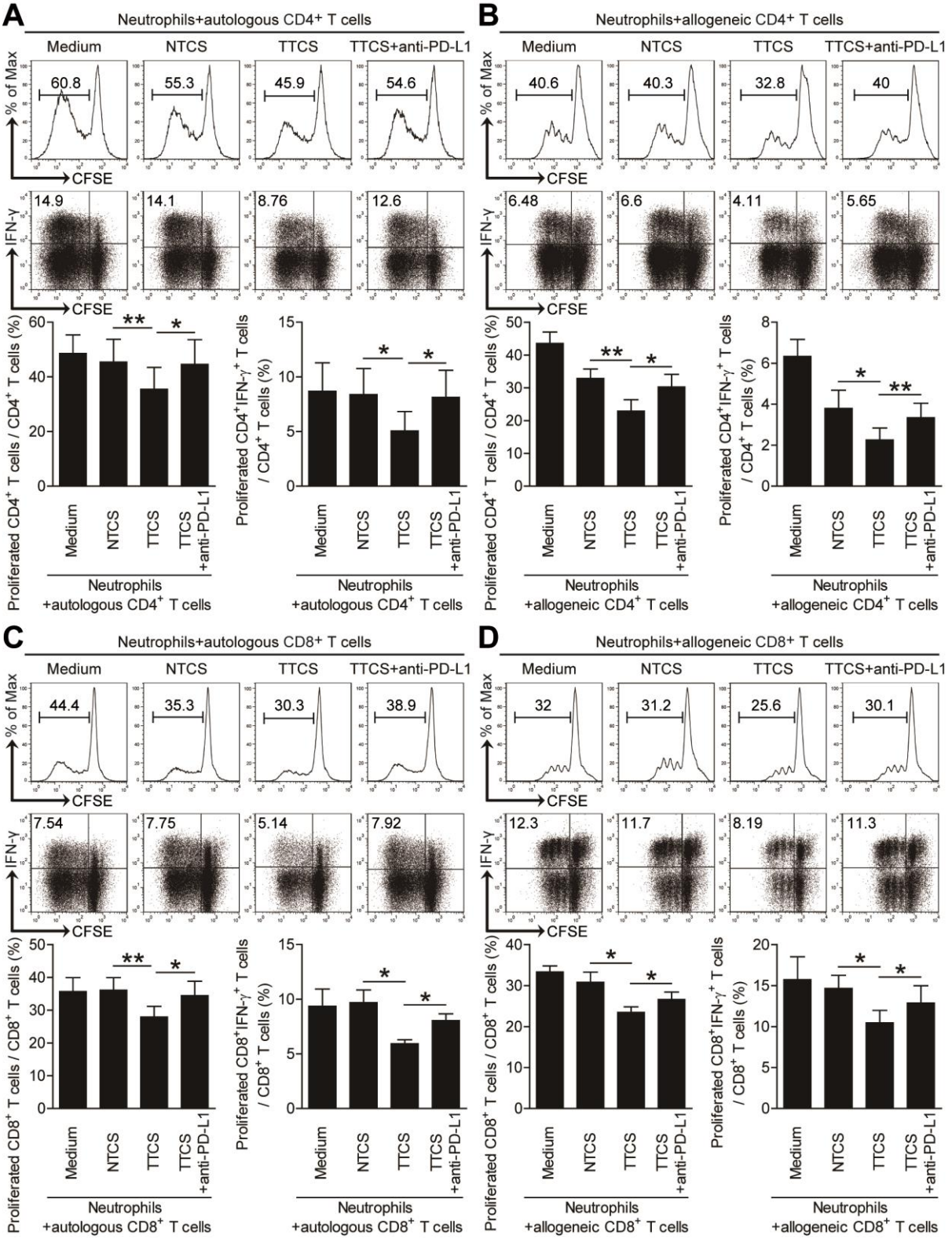
Supplementary Figure 2



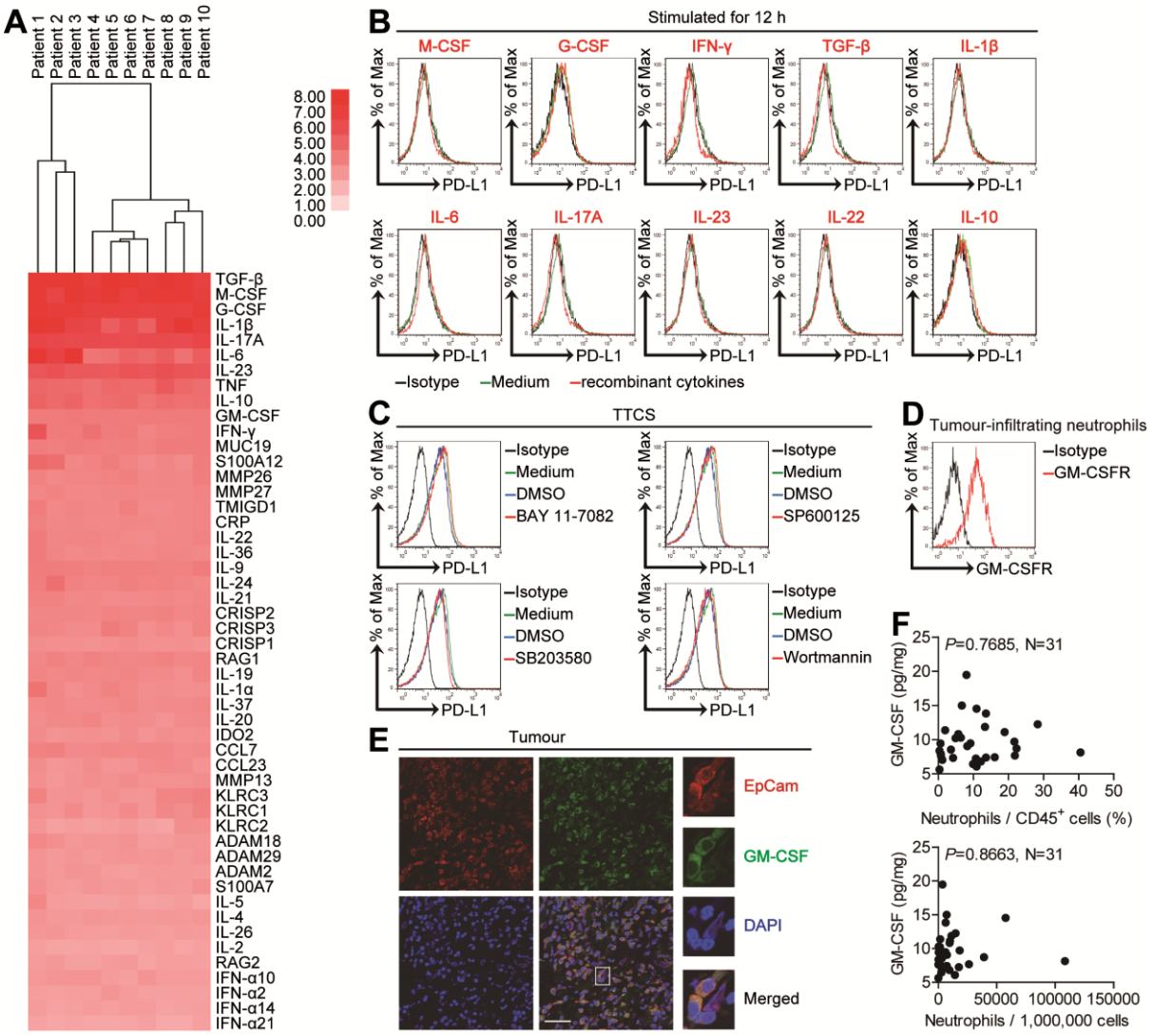
Supplementary Figure 3



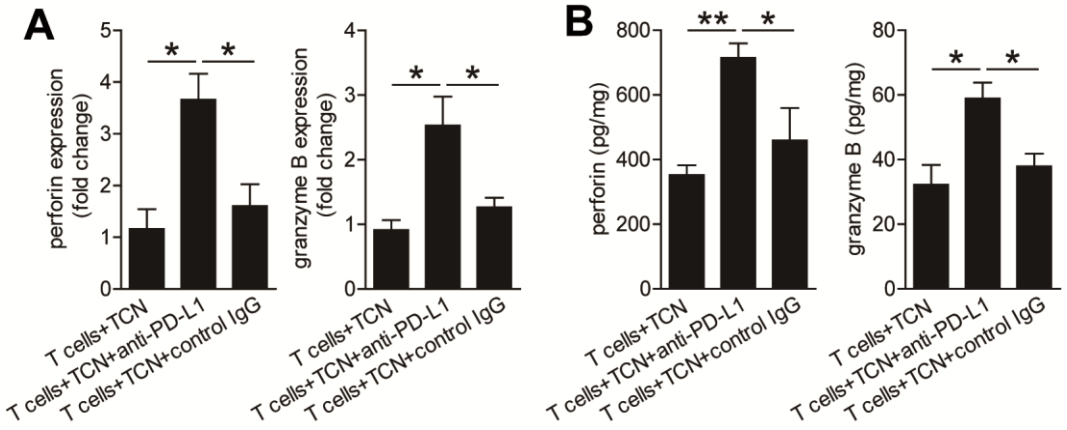
Supplementary Figure 4



Supplementary Figure 5



Supplementary Figure 6



Supplementary Figure 7