

Supplementary Figure Legends

Supplementary Figure 1.

A. Total IgG concentration (ng/ml) evaluated by ELISA in the serum of 12 PDA patients before or after one or two rounds of CT. B. FACS analysis of CD55 expression in CFPAC-1 and CAPAN-2 cells. CFPAC-1 and CAPAN-2 cells were stained with anti-CD55 monoclonal antibody (blue and red lines, respectively) or isotype-matched control antibody (black and green lines, respectively). C. Coomassie-stained 2-DE electrophoresis of CFPAC-1 cell lysates. Mass spectrometry-identified spots are highlighted and reported in Supplementary Table 1A. D. Analysis of 32 antigens recognized in at least three PDA patients after CT by SERPA. The Venn diagram represents the antigens recognized by autoantibodies after the first and the second round of CT, and those recognized in both rounds of CT.

Supplementary Figure 2. A. Coomassie-stained 2DE electrophoresis of untreated and GEM-treated CFPAC-1 cell lysates. Mass spectrometry-identified spots are highlighted and reported in Supplementary Table 2. B. The Venn diagram shows the number of antigens with an increased autoantibody recognition after CT identified by SERPA, and the number of proteins from CFPAC-1 cells that displayed an enhanced expression after GEM treatment. The proteins in common are listed in the box.

Supplementary Figure 3. Gene expression levels (reported as \log_{10} -transformed FPKM values) in cancer samples from TCGA database of PDA patients. The names of all the genes considered are listed on the right. The 23 genes coding for TAA recognized in at least three patients after CT by SERPA are indicated in italics. The

11 genes coding for TAA recognized by at least seven patients are indicated in red. The overlapping genes are indicated in red and italics.

Supplementary Figure 4. A. Bar plot representing the percentage of proliferative responses ($SI \geq 2$) from 13 PDA patients' PBMC stimulated with ENO1, G3P, K2C8 and FUBP1 before and after one and two CT cycles. B. Ratio between CD8 and Treg (CD4 and FoxP3 double positive) evaluated by flow cytometry on five PDA patients' PBMC stimulated with ENO1, G3P, K2C8 and FUBP1 before (12 responses), after one (9 responses) and two (6 responses) CT rounds.

Supplementary Figure 5. A. Immunohistochemical evaluation of ENO1 expression at different stages of tumorigenesis from PanIN (8 weeks old, upper panel) to advanced tumor lesions (12 months old, bottom panel). B-D. Representative immunohistochemistry of CD4 (B), CD8 (C) and macrophages (D) in tumor lesions of 24 week old treated mice. Black arrows indicate positive cells. Each section was counterstained with hematoxylin. Original magnification A. X20, B-D X10. E-F. CD4+ and CD8+ circulating T cells in mice at 16 (E) and 24 weeks of age (F). G. Spearman correlation of the number of tumor-infiltrating CD4 and CD8 cells in the different treatment groups. H. Anti-ENO1 IgG titer in sera from GEM+ENO1-treated mice undepleted or depleted for CD4, CD8 and B cell subsets.

In all experiments the number of mice per group was between 4 and 7; graphs report the mean \pm SEM values and statistical significance is shown.

Supplementary Figure 6. Number (A) and mean tumor lesion diameter (B) of PanIN and invasive PDA lesions in untreated and treated mice. The experimental groups are indicated as follows: untreated (NT, white bars), GEM (light grey bars), ENO1

(dark grey) and GEM+ENO1 (black bars). Statistical differences are reported on the graph.