

Figure S1 The landscape of GC prognostic-related gene scores in tumor microenvironment cells in gastric cancer. (A) A UMAP diagram showing the clustering outcomes of the 20 main subtypes derived from the normal and malignant stomach tissues. Different colors represent distinct types of cells. (B) A heatmap displaying the most prevalent marker genes in Figure A's cluster. The magnitude of the dot represents the proportion of cells in each primary cell type that express the marker gene, while the color represents the average expression level of the marker gene in each primary cell type. (C) A heatmap showcases the expression of marker genes in distinct cellular subpopulations based on the enrichment analysis by GSEA. GC, gastric cancer; UMAP, The Uniform Manifold Approximation and Projection for Dimension Reduction; C, cluster; GSEA, gene set variation analysis.

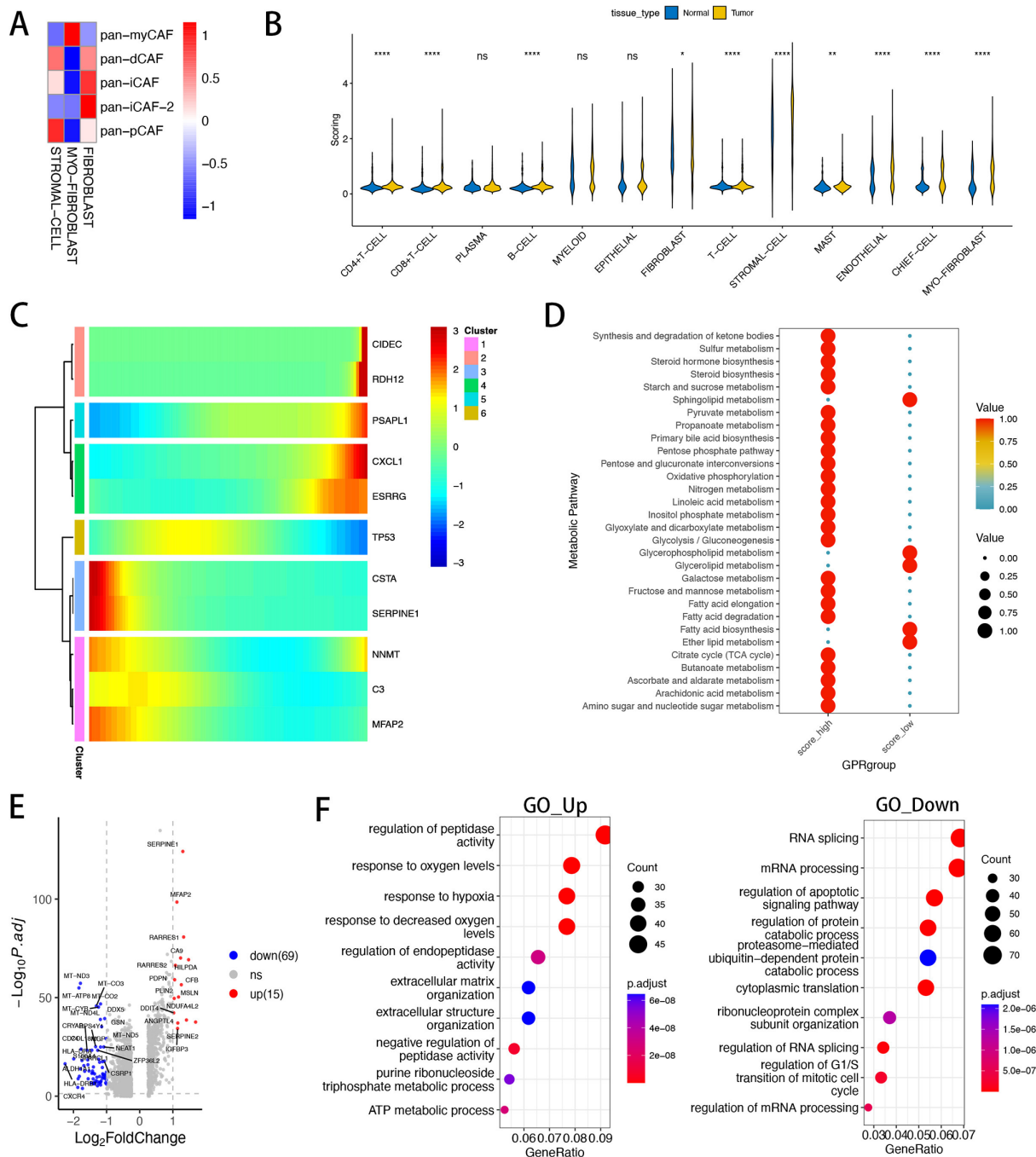


Figure S2 Functional evaluation of prognosis-related genes at the single-cell level. (A) A heatmap showing the correlations between three types of stromal cells and the different CAF subtypes, along with the corresponding levels of gene expression. (B) An accordion chart showing the disparities in GPR scores between 13 subtypes of single cells in tumor and normal tissues (*, $P < 0.01$; ***, $P < 0.001$; ****, $P < 0.0001$; ns: no statistically significant difference). (C) A pseudo-time analysis was conducted to examine the differentiation trajectories of three types of stromal cells and the temporal expression patterns of 11 GPR genes. (D) scMetabolism analysis was used to examine the metabolic differences among the stromal cells across the high_score and low_score groups. (E) A volcano plot showing the DEGs in the stromal cells grouped by different scores, with a significance threshold of \log_2 fold change > 1 and $P < 0.05$. (F) Bubble charts showing the GO enrichment of the DEGs ($P < 0.05$). CAF, cancer-associated fibroblast; GC, gastric cancer; GPR, GC prognostic-related; DEGs, differentially expressed genes; GO, Gene Ontology.

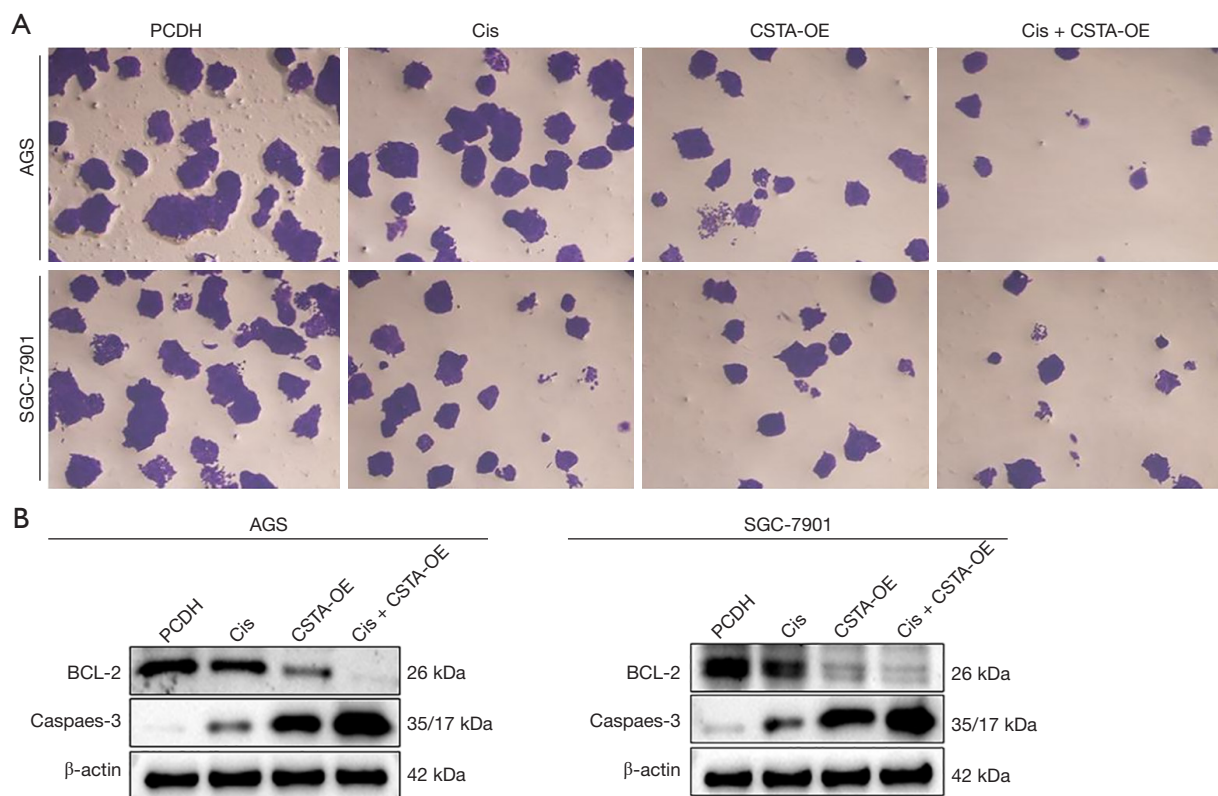


Figure S3 The overexpression of CSTA enhances the caspase-dependent pro-apoptotic effect of cisplatin. (A) Clonogenic assays were used to assess the clonogenic formation ability of the gastric cancer cells in the four experimental groups, through staining with 0.1% crystal violet further microscopy (400 \times magnification). (B) A Western blot analysis was used to measure the expression levels of pro-apoptotic protein Caspase 3 in the four experimental groups. CSTA, Cystatin A; PCDH, pCDH Plasmid vector; OE, overexpression; Cis, cisplatin.