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ORIGINAL ARTICLE

Functions and Bioinformatics Analysis of FGL1 in Gastric Cancer

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SUMMARY

Background: **FGL1 (fibrinogen-like protein 1) is considered to be closely related to cell proliferation and differentiation. The purpose of this study was to explore the value and mechanism of FGL1 as a prognostic indicator for gastric cancer (GC).**
Methods: **Initially, online database was used to analyze the expression of FGL1; surgical paired samples were collected from 10 patients with GC, and FGL1 was detected by RT-qPCR. The relationship between FGL1 and clinicopathological parameters was evaluated, as well as overall survival rate, post-progression survival rate, and relapse-free survival rate (OS, PPS, RFS). FGL1 expression was altered in cell lines to observe its effect on proliferation, migration, invasion, and apoptosis of GC cells (CCK-8 assay, colony formation assay, wound-healing assay, Transwell assay, and apoptosis assay). Finally, differential expression genes (DEGs) of GC were screened by gene chip, Gene ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analyses and PPI networks were performed on DEGs, and Hub genes were identified.**
Results: **FGL1 was highly expressed in GC cell lines and GC tissues. FGL1 expression was significantly correlated with histopathological differentiation, tumor infiltration, and lymph node metastasis of GC ($p = 0.007$; $p = 0.008$; $p = 0.042$); there was no correlation with age, gender, or distant metastasis (all $p > 0.05$). OS, PPS, and RFS of patients in GC with high FGL1 expression were shorter than of patients with low FGL1 expression (HR = 1.38, logrank $p < 0.01$; HR = 1.36, logrank $p < 0.01$; HR = 1.27, logrank $p < 0.01$). Suppressing FGL1 inhibited the proliferation, migration, and invasion ability of SGC7901 cells and arrested cell cycle at the G1 phase. FGL1 silencing did not induce apoptosis of GC cells. In total, 634 GC DEGs were obtained by gene chip analysis. GO analysis found that most of the genes were related to cell membrane and extracellular matrix. KEGG analysis revealed that DEGs were involved in signal pathways such as combined proteoglycan interaction, glycoprotein hormone and peptide hormone biosynthesis, non-integrin membrane-extracellular matrix interaction, and protein digestion and absorption, and the top 20 key genes of GC were identified, including OASL, ISG20, RAB3A, CREM, BIRC7, LHB, etc.**
Conclusions: **FGL1 is a potential biomarker for the diagnosis and prognosis of GC. FGL1-mediated signaling pathways may be a new target for GC therapy in future.**
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Supplementary Data

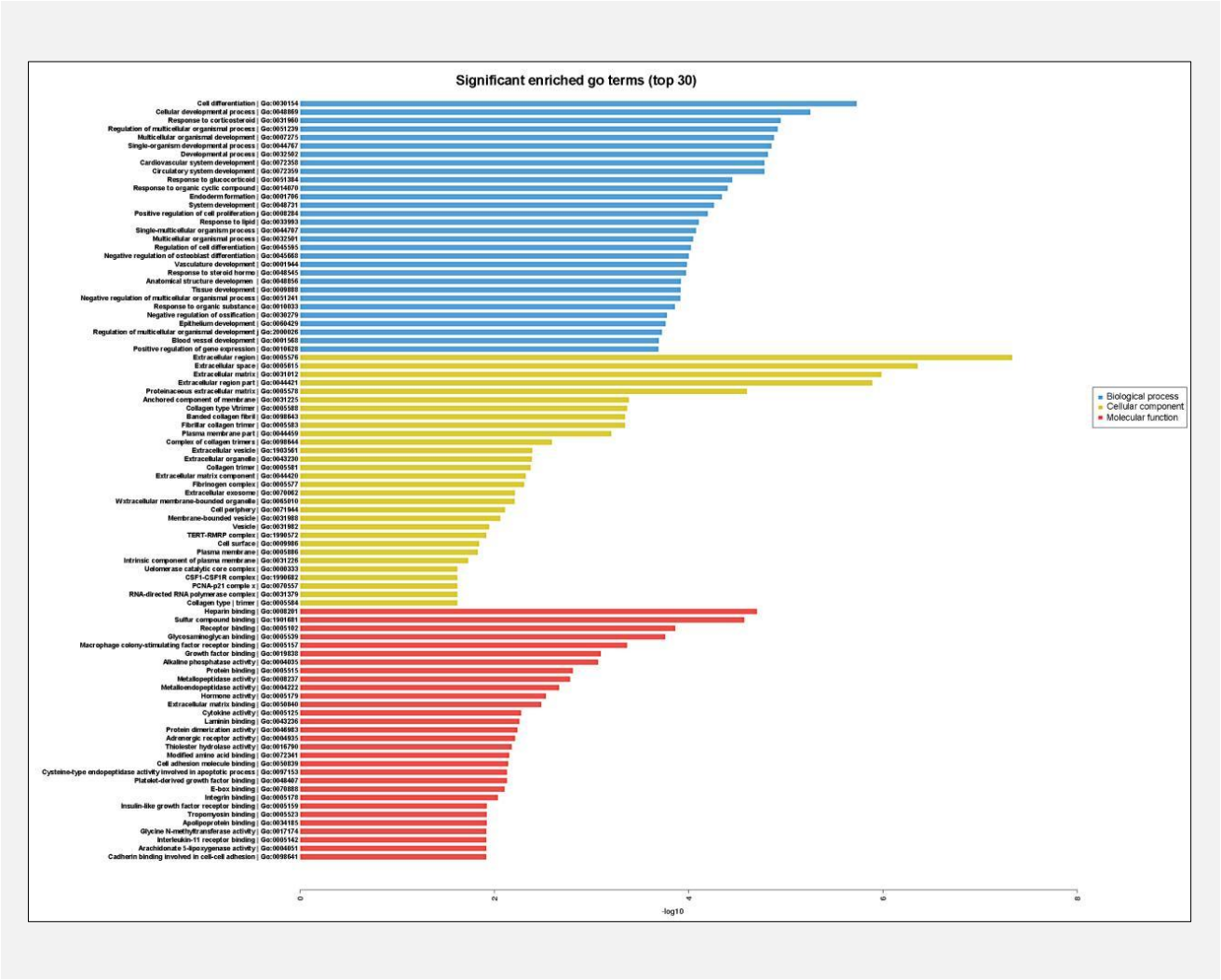

Figure S1. Bar chart of GO analysis in gastric cancer.

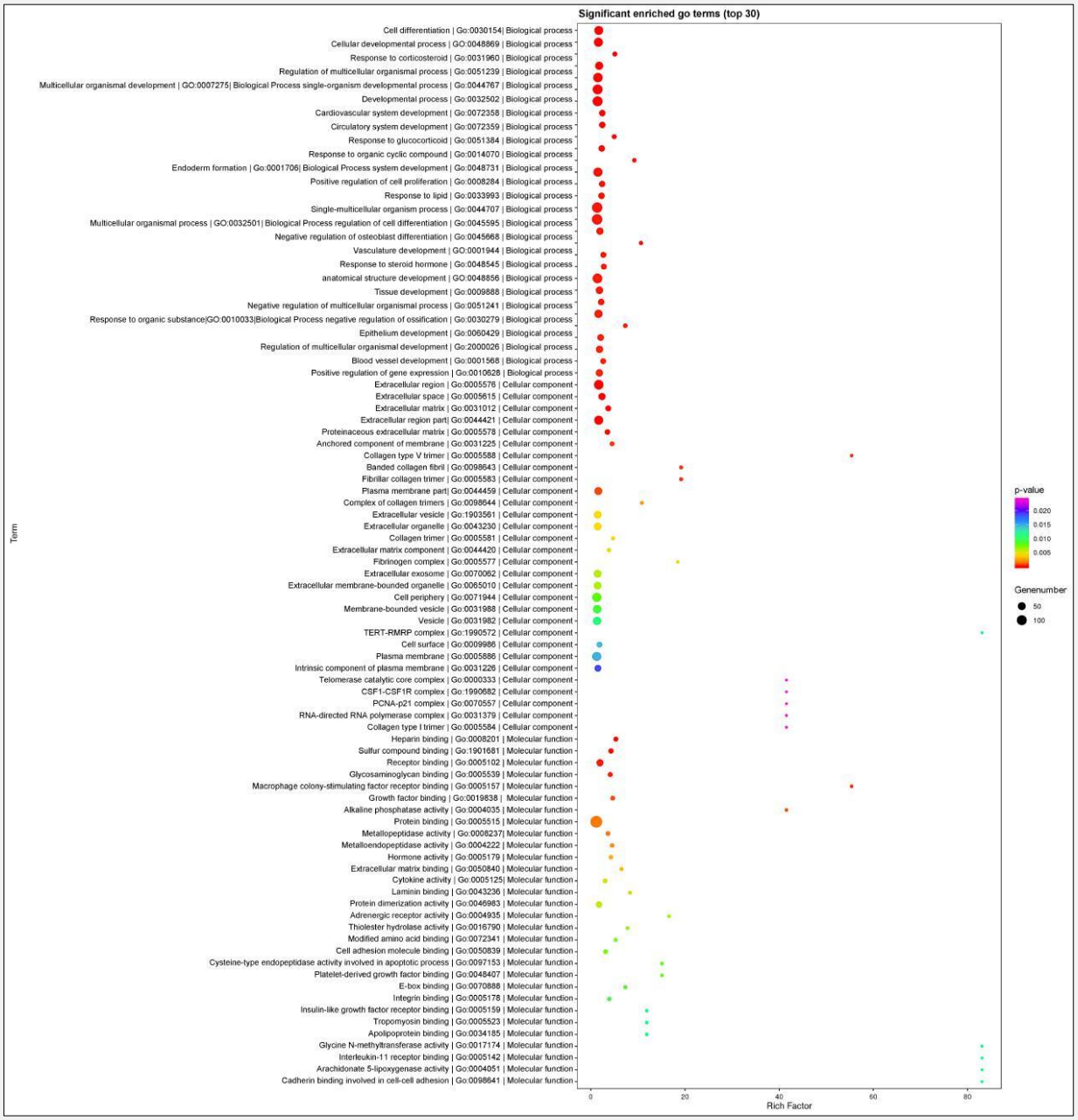

Figure S2. Bubble pattern of GO analysis in gastric cancer.

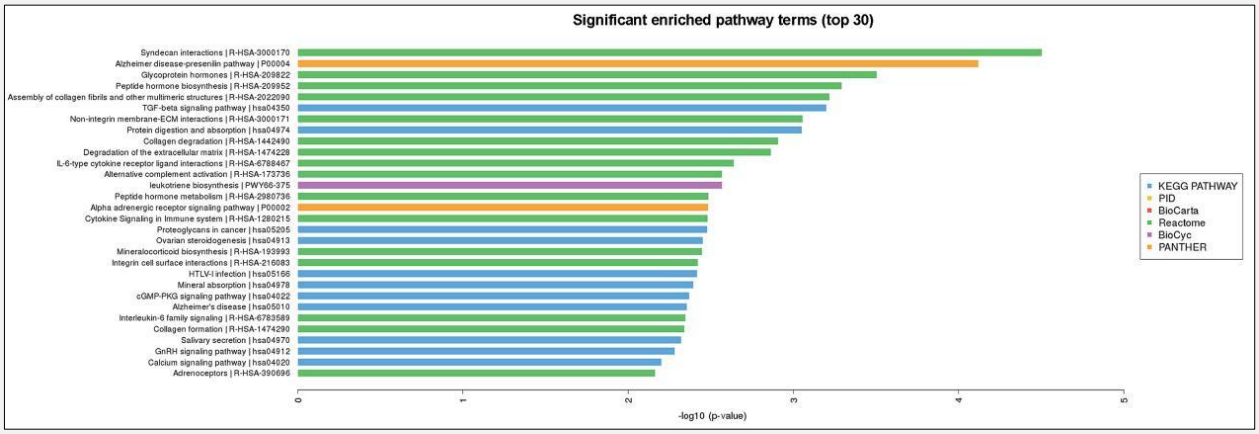

Figure S3. Bar chart of KEGG analysis in gastric cancer.

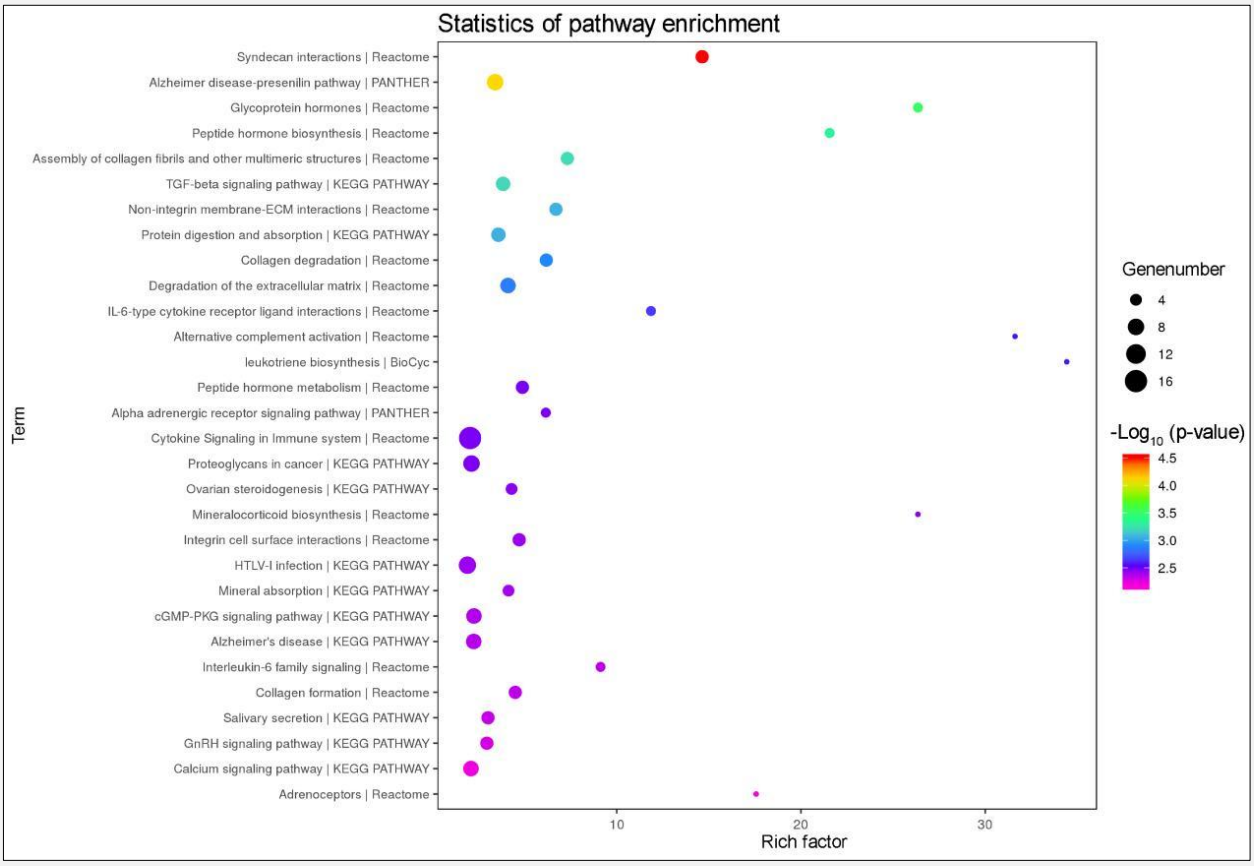

Figure S4. Bubble pattern of KEGG analysis in gastric cancer.

Figure S1 - S4. Enrichment analysis for DEGs of gastric cancer.
DEGs - differential expressed genes, GO - Gene ontology, KEGG - Kyoto Encyclopedia of Genes and Genomes.

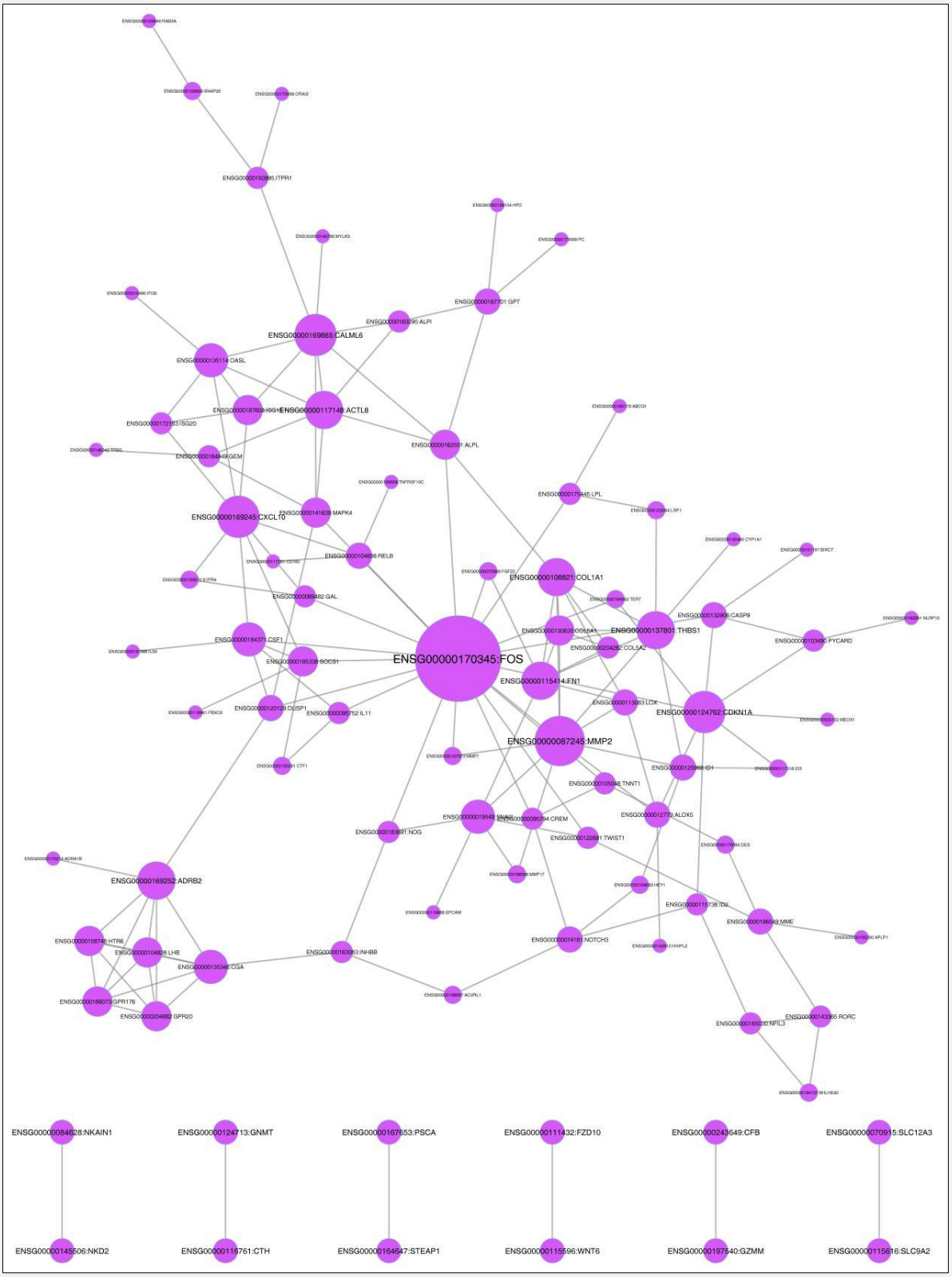

Figure 5. PPI network analysis for DEGs of gastric cancer.

PPI – Protein-protein interaction.