

Figure S1. Expression of tRF-Glu49 after mimics or inhibitor were transfected in cervical carcinoma cells. \*\* $P < 0.01$ , tRF-Glu49 inhibitor group vs. Caski cells transfected with inhibitor to NC, tRF-Glu49 mimics group vs. HeLa cells transfected with mimics NC tRF, tRNA-derived fragment; NC, negative control.

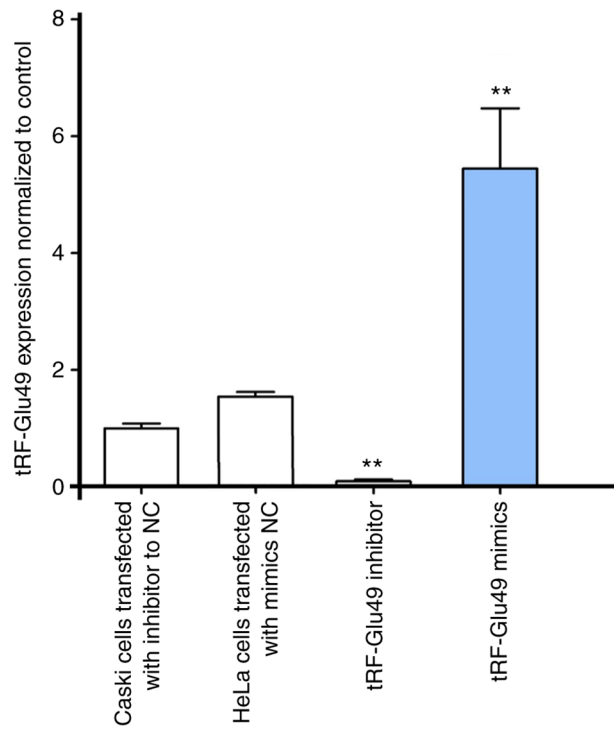


Figure S2. Gene ontology and Kyoto Encyclopedia of Genes and Genomes pathway enrichment analyses for pathways selection.

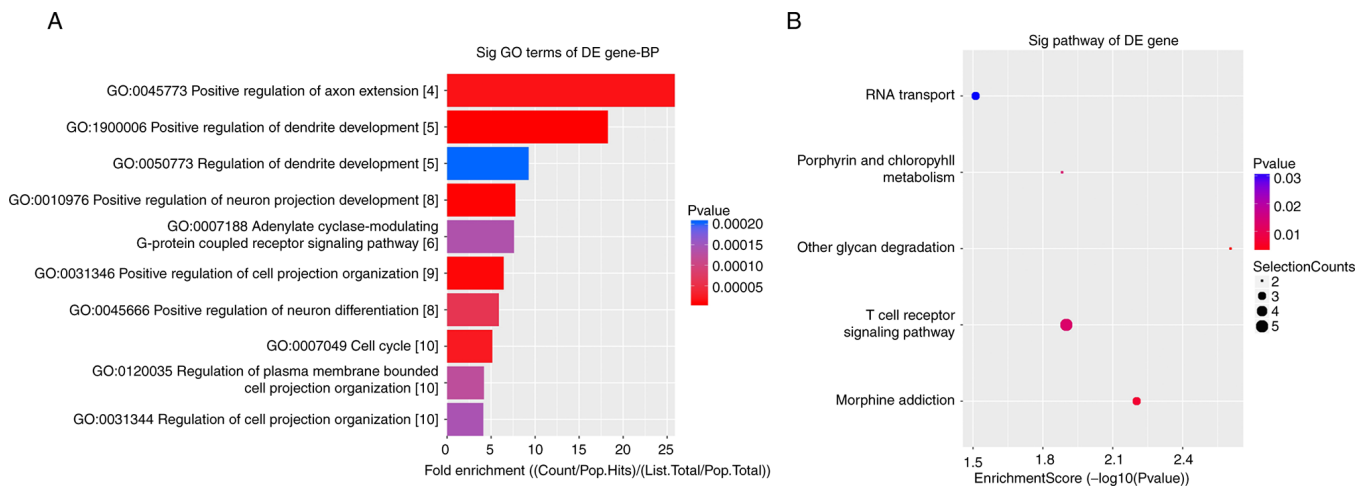


Figure S3. FGL1 eliminates biological functions of tRF-Glu49. (A and B) Effects of tRF-Glu49 and FGL1 on proliferative capacity of (A) Caski and (B) HeLa cells evaluated by Cell Counting Kit-8 assay. (C and D) Effects of tRF-Glu49 and FGL1 on migration and invasion capacity of (C) Caski and (D) HeLa cells evaluated by Transwell and Matrigel assays. \*\* $P < 0.01$  vs. Control group. FGL1, fibrinogen-like protein-1; tRF, tRNA-derived fragment; si-, small interfering.

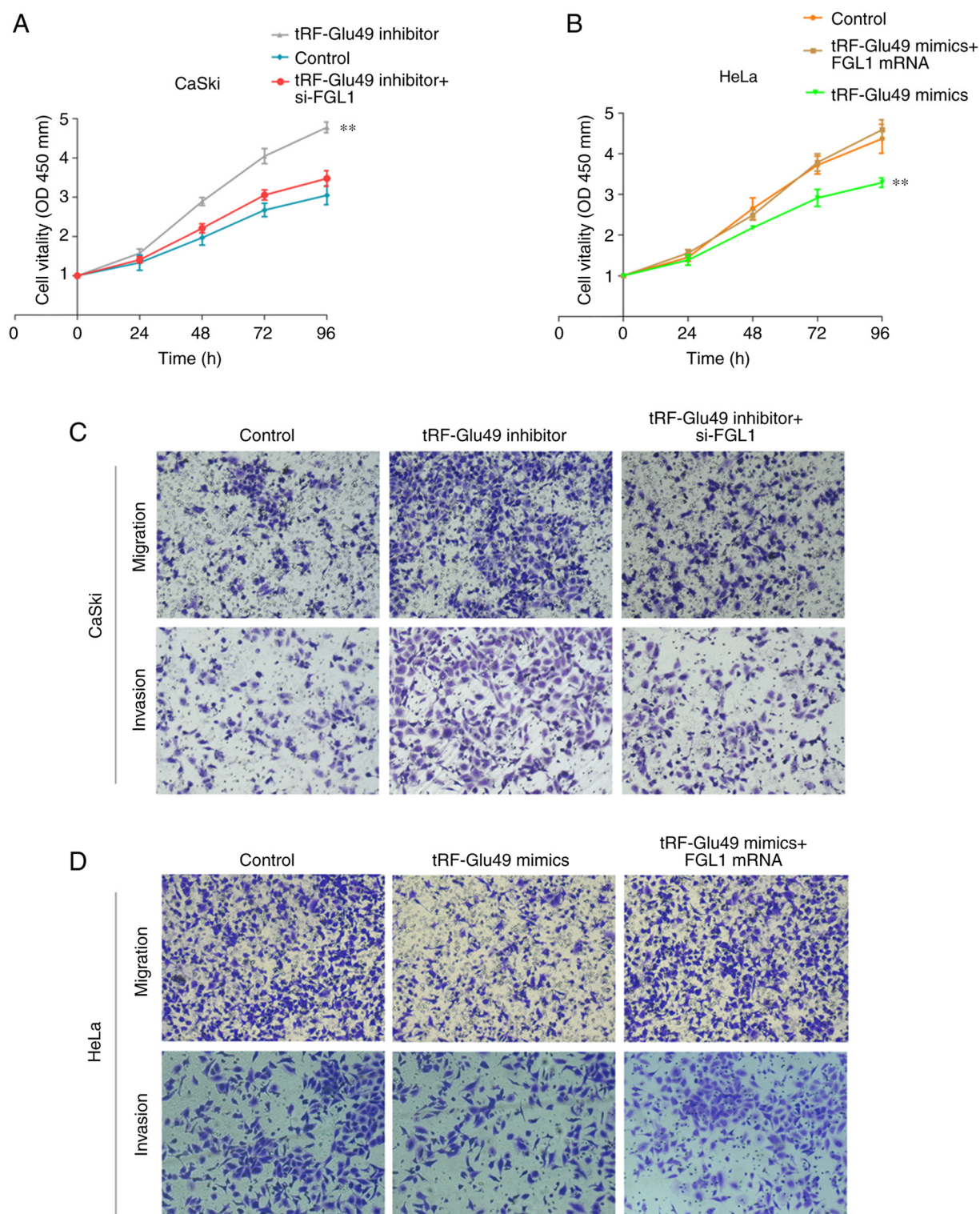


Figure S4. Proof of transfection of si-FGL1 and FGL1 overexpression vector in Caski and HeLa cells. (A) Downregulation of FGL1 in Caski cells transfected with si-FGL1 compared with Caski cells transfected with negative control siRNA. (B) Upregulation of FGL1 in HeLa cells transfected with the FGL1 overexpression vector compared with HeLa cells transfected with the corresponding negative control. \*\*P<0.01 vs. Control group. FGL1, fibrinogen-like protein-1; si-, small interfering.

