Figure S1. Downregulation of miR-302c-3p and miR-520a-3p increases proliferation and decreases apoptosis in CC cells. (A) Reverse transcription-quantitative PCR assays to detect the transfection efficiencies of miR-302c-3p and miR-520a-3p inhibitors. Inhibitor NC represents inhibitor control. Compared with blank group. (B) Cell counting kit-8 assays of HeLa-S3 (left) and C-33A (right) cell proliferation after transfection with miR-302c-3p/miR-520a-3p inhibitor. (C) Changes in CC cell proliferation were also determined by EdU staining (EdU/DAPI). Decreased cell proliferation by upregulating miR-302c-3p or/and miR-520a-3p was observed in HeLa-S3 (top) and C-33A (bottom) cells. Compared with inhibitor NC group. (D) The changes in apoptosis of CC cells were determined by flow cytometry. Compared with inhibitor NC group. *P<0.05; **P<0.01; ***P<0.001. CC, cervical carcinoma; miR, microRNA.

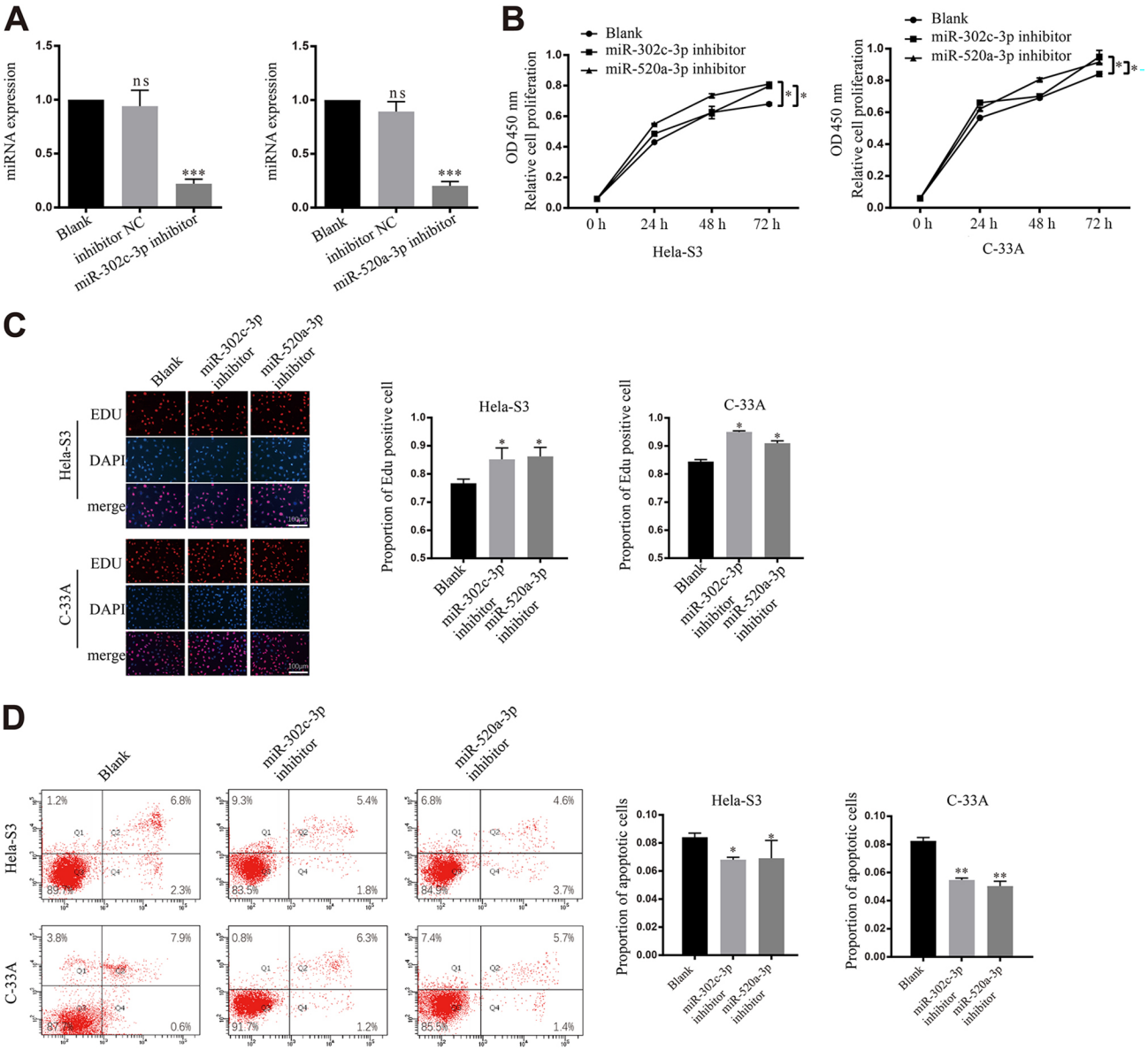

Figure S2. CXCL8 is identified as a common target gene of miR-302c-3p and miR-520a-3p. The expression levels of CXCL8 vs. miR-302c-3p/miR-520a-3p were displayed based on the reverse transcription-quantitative PCR results. **P<0.01. miR, microRNA; CXCL8, C-X-C motif ligand 8.

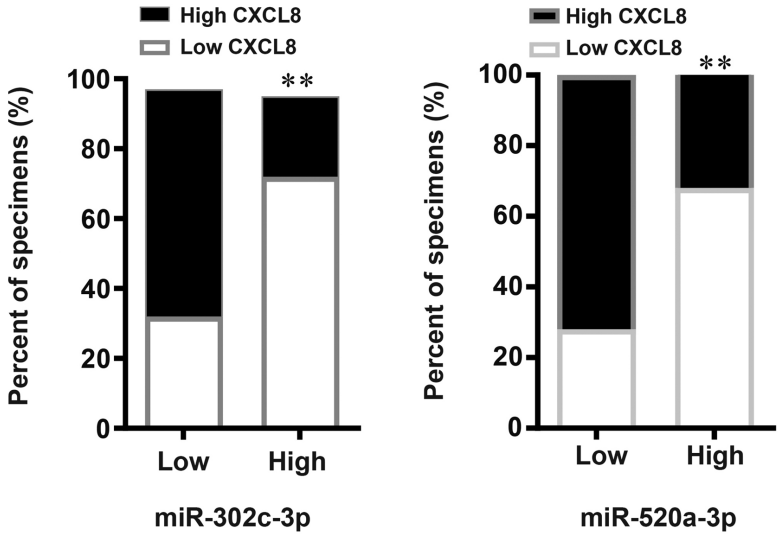

Figure S3. Proliferation-promoting and apoptosis-suppressive effects of CXCL8 in cervical carcinoma cells. (A) EdU assay of HeLa-S3 and C-33A cells transfected with pcDNA3.1-CXCL8. (B) Flow cytometry was used to determine the apoptosis of HeLa-S3 and C-33A cells transfected with pcDNA3.1-CXCL8. CXCL8 siRNA was compared with si-NC. *P<0.05; **P<0.01; ***P<0.001. CXCL8, C-X-C motif ligand 8; si-NC, siRNA negative control.