Long non-coding RNA XIST promotes cell growth and invasion through regulating miR-497/MACC1 axis in gastric cancer

Supplementary Materials



Supplementary Figure S1: XIST down-regulation inhibited HGC-27 cells growth and invasion abilities. (A) LncRNA XIST down-regulation decreased colony formation ability of HGC-27 cells. (B) LncRNA XIST down-regulation led to cell cycle arrest in the G1 phase. (C) The EdU assay revealed that lncRNA XIST down-regulation inhibited HGC-27 cell proliferation. (D) Knockdown of XIST led to an increased rate of apoptosis in HGC-27 cells. (E) The boyden assay showed that lncRNA XIST down-regulation decreased HGC-27 cell invasion.



Supplementary Figure S2: Anti-miR-497 treatment rescued the effect of XIST down-regulation on HGC-27 cell growth and invasion. (A) and (B) MTT and colony formation assays revealed that knockdown of XIST decreased cell proliferation, while anti-miR-497 treatment rescued this effect. (C) The cell cycle distribution altered by XIST down-regulation can be counteracted by anti-miR-497 treatment. (D) Knockdown of XIST increased apoptosis, while anti-miR-497 treatment counteracted the effect of XIST down-regulation on HGC-27 cell invasion.



Supplementary Figure S3: Restoration of MACC1 rescued the effect of XIST knockdown on HGC-27 cell growth, cell cycle distribution, apoptosis and invasion. (A) and (B) MTT and colony formation assays revealed that knockdown of XIST decreased HGC-27 cell proliferation, while restoration of MACC1 rescued this effect. (C) The cell cycle distribution altered by XIST down-regulation can be counteracted by MACC1 restoration. (D) Knockdown of XIST increased apoptosis, while overexpression of MACC1 counteracted this effect. (E) MACC1 overexpression counteracted the effect of XIST down-regulation on invasion. (F) The expression levels of lncRNA XIST, miR-497, and MACC1 in clinical samples.