Figure S1. Expression of MIAT was positively correlated with the cell proliferation and metastasis ability. (A) reverse transcription-quantitative PCR analysis of MIAT expression in AGS and HGC-27 cells. (B) AGS and HGC-27 cells proliferation ability was assessed by Cell Counting Kit-8 (CCK-8) assay. (C) AGS and HGC-27 cells metastasis ability was assessed by Transwell assay (magnification, x100). n=3. *P<0.05, **P<0.01 vs. the AGS group. GC, gastric cancer; MIAT, myocardial infarction associated transcript.

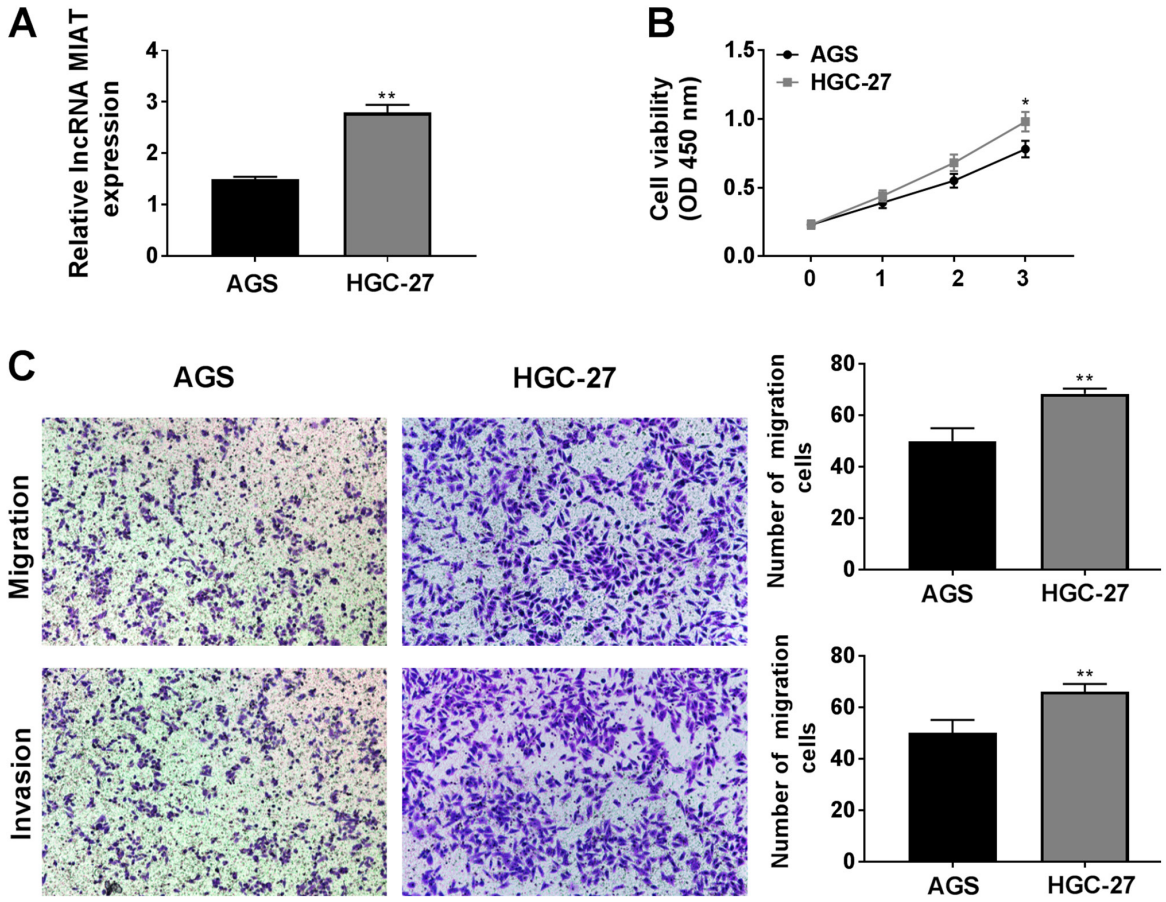

Figure S2. Cell proliferation in serum-free medium has little effect on wound healing (cell migration) and invasion assay. The AGS and HGC-27 cells without transfection ($5x10^3$ cells/well) were seeded into 96-well plates. The cell proliferation in serum-free medium or medium with 10% FBS was assessed by Cell Counting Kit-8 assay. n=3. $^{**}P<0.01$ vs. the serum-free medium group.

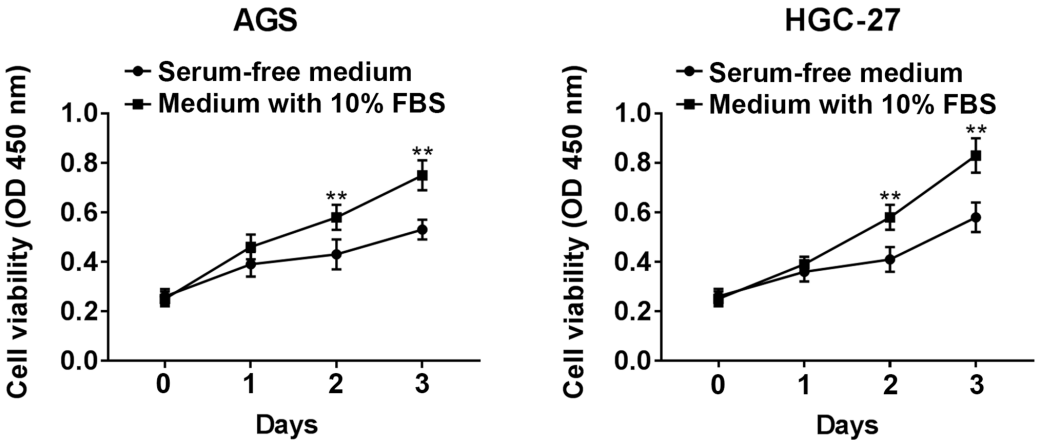

Figure S3. Functions of MIAT knockdown regulating cell proliferation and invasion were reversed by miR-331-3p inhibitor in gastric cancer cells. (A) Cell proliferation ability was assessed by Cell Counting Kit-8 assay. (B) Cell invasion capacity was evaluated by Transwell invasion assay. n=3. *P<0.05, **P<0.01 vs. the blank or si-NC + inhibitor NC group; $^{\#\#}$P<0.01 vs. the si-MIAT-1 + inhibitor NC group; $^{\&\&}$P<0.01 vs. the si-NC + miR-331-3p inhibitor group. MIAT, myocardial infarction associated transcript; NC, negative control; miR, microRNA; si, small interfering.