

Supplementary Fig.1.

(A) Linc00261 expression pattern was examined in breast cancer cell lines (MCF7, ZR-75-30, BT474, SKBR-3, MDA-MB-231) via RT-qPCR. (B) Relative expression levels of Linc00261, measured by qPCR, in MCF7 cell after siRNA-mediated knockdown of Linc00261. (C) Relative expression levels of Linc00261, measured by qPCR, in MCF7 cell after shRNA-mediated knockdown of Linc00261. (D) Relative expression levels of Linc00261, measured by qPCR, in MDA-MB-231 cell after overexpression of Linc00261. (E) Relative expression levels of NME1, measured by qPCR, in MCF7 cell after overexpression of NME1. (F) Relative expression levels of NME1, measured by qPCR, in MDA-MB-231 cell after siRNA-mediated knockdown of NME1. Data are reported as means \pm SD. *P<0.05; **P<0.01; ***P<0.001.



Supplementary Fig.2.

(A) The stability of NME1 and β -actin mRNA over time was measured by qRT-PCR relative to time 0 after blocking new RNA synthesis with a-amanitin (50 mM) in breast cancer cells and normalized to 18S rRNA (a product of RNA polymerase I that is unchanged by a-amanitin) by silencing linc00261 expression. (B) Nascent NME1 mRNA levels in siLinc00261 and control cells were quantified by qRT-PCR. (C) The relationship between mRNA expressions of linc00261 and NME1 in 60 breast carcinoma tissues. Data are reported as means ± SD. **P<0.01.



Supplementary Fig.3.

(A) Silencing NME1 could rescue cell proliferation capacity caused by Linc00261 overexpression via MTT assay. (B) Silencing NME1 could rescue EMT phenotype after linc00261 overexpression by qRT-PCR. Data are reported as means \pm SD. *P<0.05; **P<0.01; ***P<0.001.