Supplementary information for

Linifanib (ABT-869) Potentiates the Efficacy of Chemotherapeutic Agents through the Suppression of Receptor Tyrosine Kinase-Mediated AKT/mTOR Signaling Pathways in Gastric Cancer

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Figure S1. The cytotoxicity of ABT-869 in gastric cancer cells. 

**a.** The chemical structure of ABT-869.

**b.** The cytotoxicity of ABT-869 in a broad of gastric cancer cell lines. After the cells were treated with ABT-869 for 48 h, cell viability assays were performed. The half maximal inhibitory concentration (IC<sub>50</sub>) was calculated by Prism 5 (GraphPad Software, La Jolla, CA). Dots, mean of five replicates.
Figure S2. ABT-869 potentiates cell cycle arrest induced by 5-Fu or cisplatin in MGC-803 gastric cancer cells. MGC-803 cells were treated with indicated concentrations of agents (15 μmol/L of 5-FU; 10 μmol/L of cisplatin; 0.05 μmol/L or 0.1 μmol/L of ABT-869) either alone or in combination for 48 h, followed by cell cycle analysis. Columns, mean; bars, standard deviation. **, $P < 0.01$; ***, $P < 0.001$ vs. 5-FU or cisplatin alone group.
Figure S3. ABT-869 potentiates cell cycle arrest induced by 5-Fu or cisplatin in BGC-823 gastric cancer cells. BGC-823 cells were treated with indicated drugs (15 μmol/L of 5-FU, 10 μmol/L of Cisplatin and 0.1 μmol/L of ABT-869) either alone or in combination for 48 h, followed by cell cycle analysis. BGC-823 gastric cancer cells were obtained from the China Center for Type Culture Collection (Shanghai, China).
Figure S4. ABT-869 potentiates apoptosis induced by 5-Fu or cisplatin in AGS and BGC-823 gastric cancer cells. AGS and BGC-823 cells were treated with indicated drugs (15 μmol/L of 5-FU, 10 μmol/L of cisplatin, 0.1 μmol/L of ABT-869) either alone or in combination for 48 h, followed by cell apoptosis determination. AGS gastric cancer cells were obtained from the China Center for Type Culture Collection (Shanghai, China). Columns, mean; bars, standard deviation. *** P < 0.001 vs. 5-FU or cisplatin alone group.