Supplementary Figures

Figure S1. POU2F2 mRNA expression is highly expressed only in gastric cancer cells with high metastatic potential.

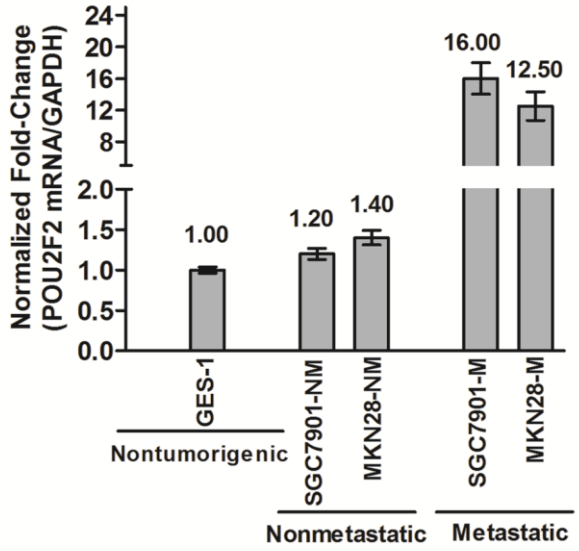

Figure S2. The inhibition efficiency of POU2F2-specific siRNA on POU2F2 proteins was detected by western blot analysis.

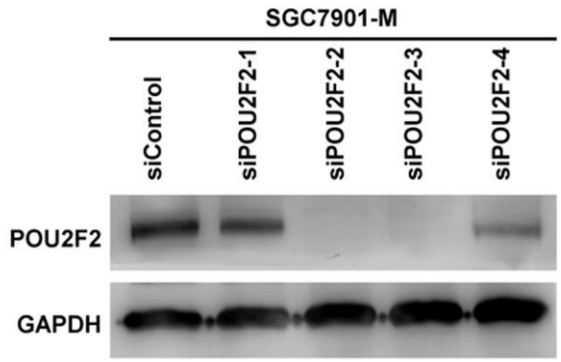

Figure S3. ChIP-PCR sequencing data showed that POU2F2 can bind to the ROBO1 promoter.

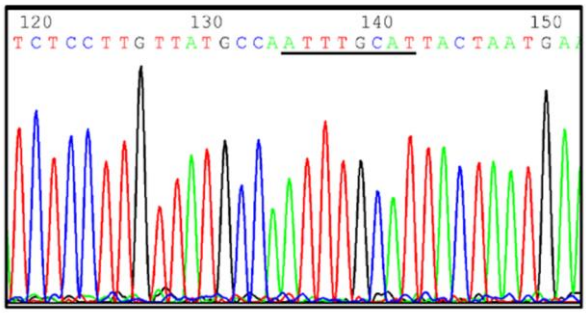

Figure S4. The inhibition efficiency of ROBO1-specific siRNA on ROBO1 proteins was assessed by western blot analysis.

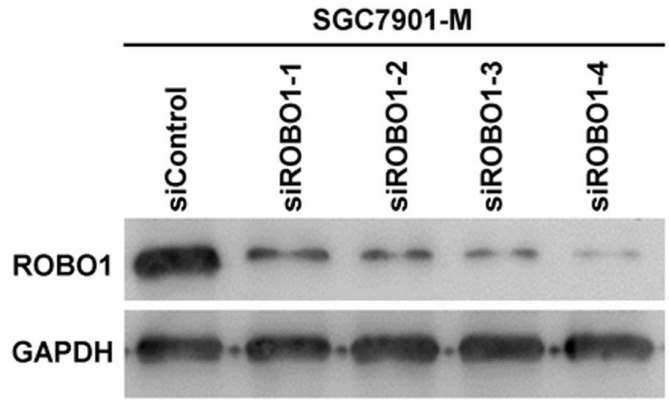

Figure S5. RNAi-mediated silencing of SLIT2 decreased invasion and metastasis driven by ROBO1 overexpression in SGC7901-M (A) and SGC7901-NM-POU2F2 cells (B).

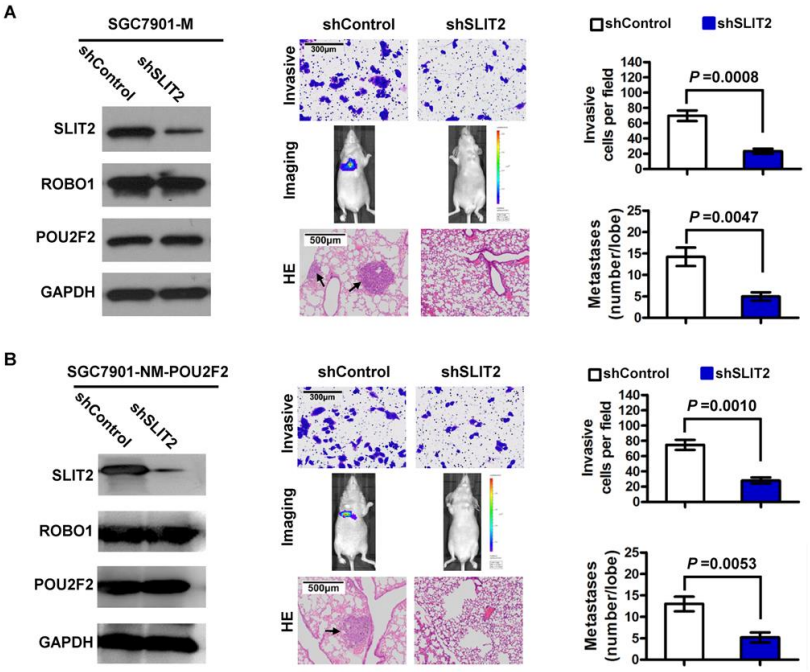

Figure S6. POU2F2 overexpression led to an Epithelial-mesenchymal transition indicated by markers expression. Western blot (A) and Immunofluorescence (B) results showed that POU2F2 overexpression cells characterized by gain of mesenchymal markers (Vimentin, MMP2 and MMP9) and loss of epithelial gene express (ß-catenin, E-cadherin).

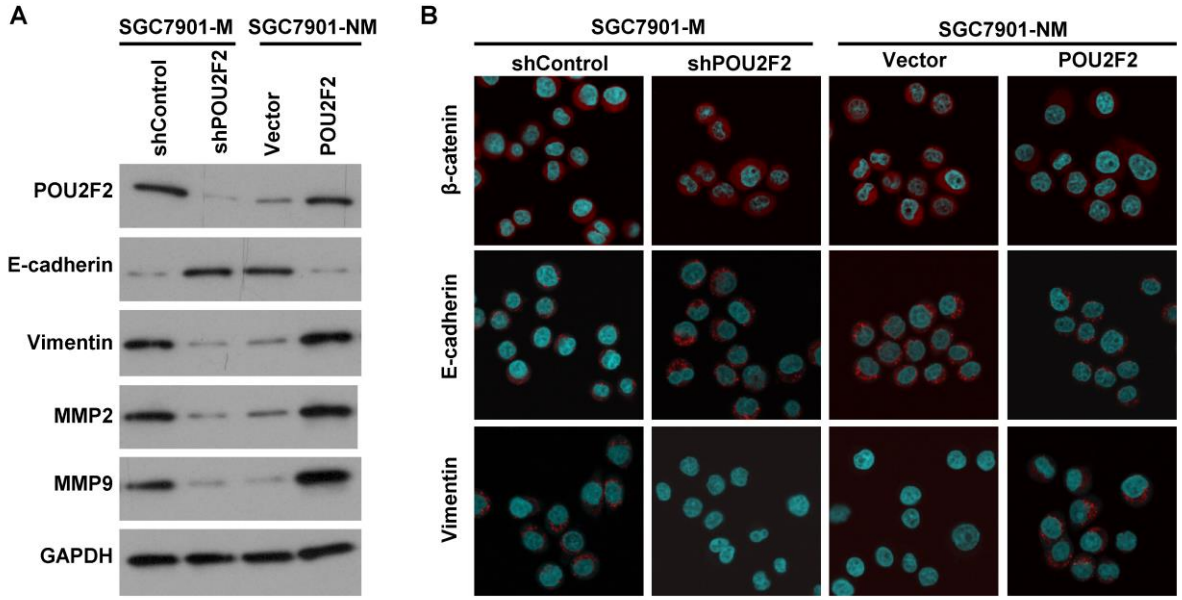

Figure S7. ChIP-PCR sequencing results showed that NF-?B can bind to the POU2F2 promoter.

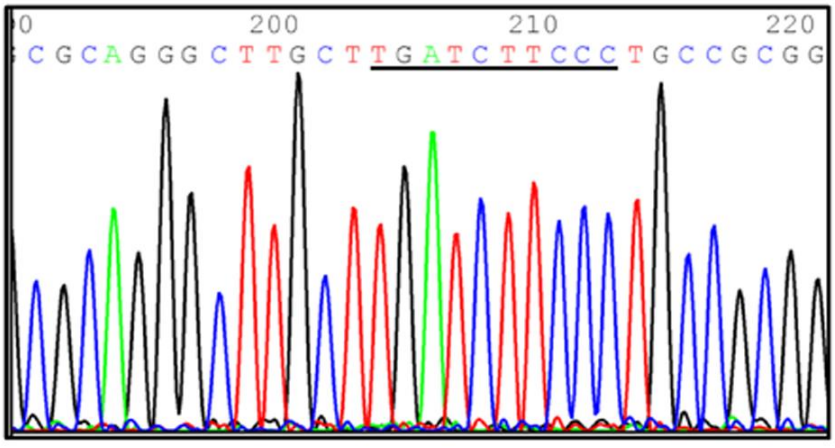

Figure S8. pBabe-Puro-IKBalpha-mut abrogated the LPS-mediated increase in NF-?B activation. SGC7901-NM was transduced with the pBabe-Puro-IKBalpha-mut vector encoding a NF-?B specific inhibitor or an empty vector. Cells were stimulated with 10 μg/ml LPS for 4 h, NF-?B activity was detected by a reporter gene. NF-?B activity was increased in SGC7901-NM-vector cells, whereas there was almost no change in SGC7901-NM-IKBalpha-mut cells.

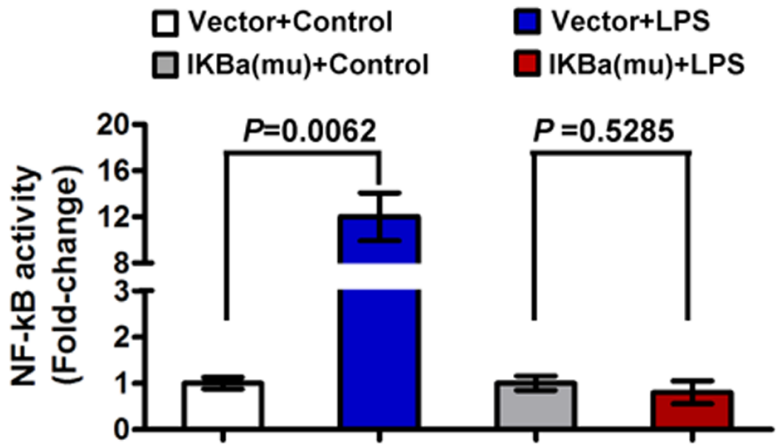

Figure S9. MiR-218 expression levels were analyzed by qPCR.

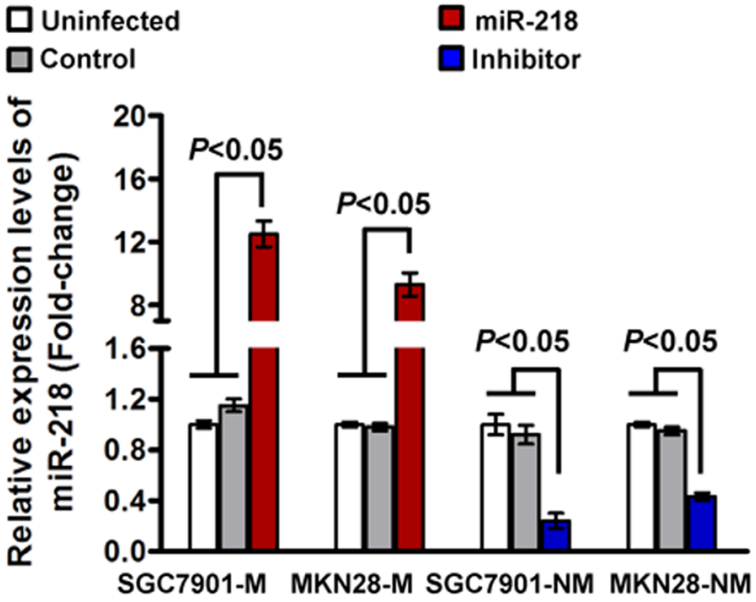

Figure S10. NF-?B activity was detected by a reporter gene.