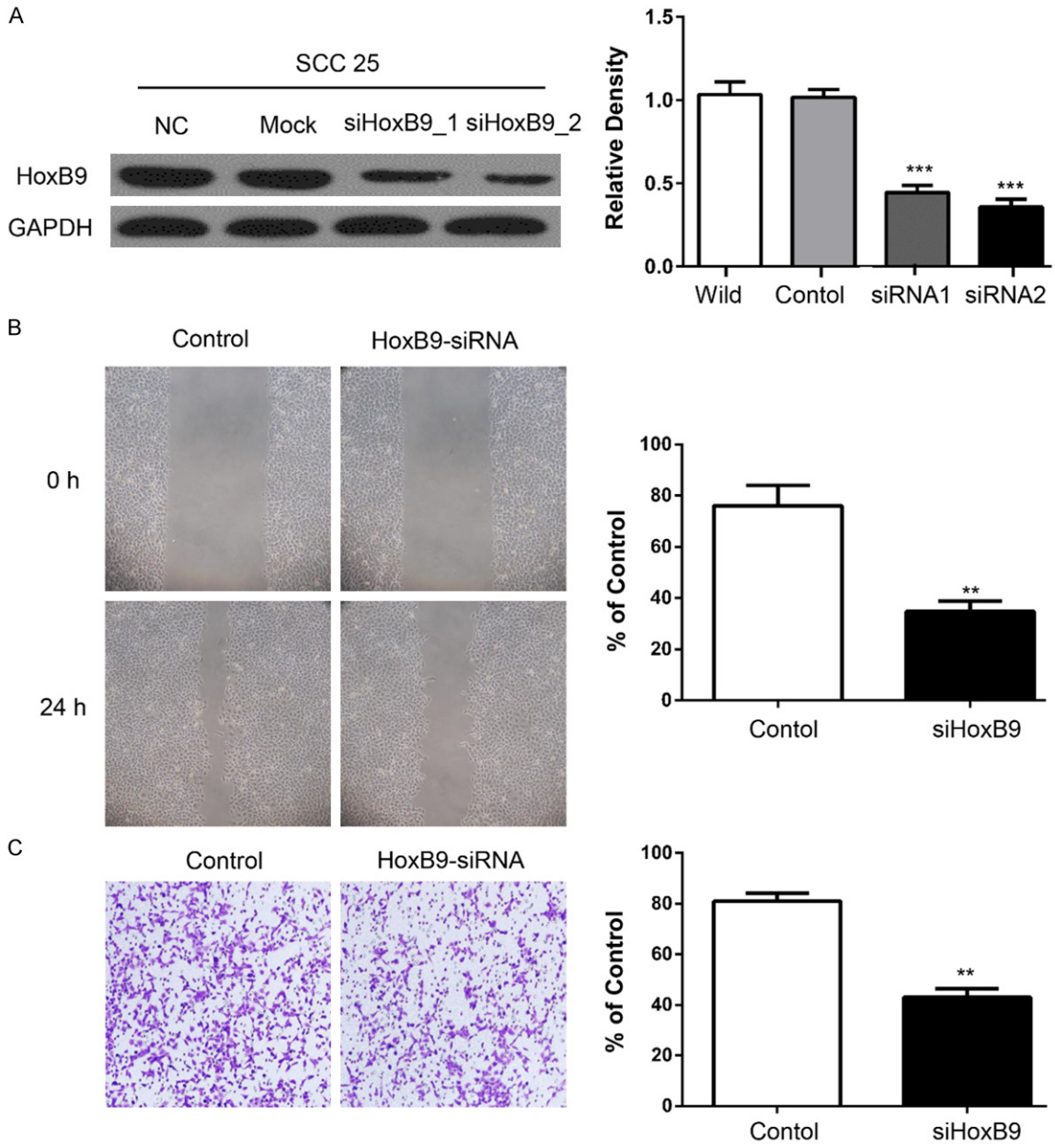
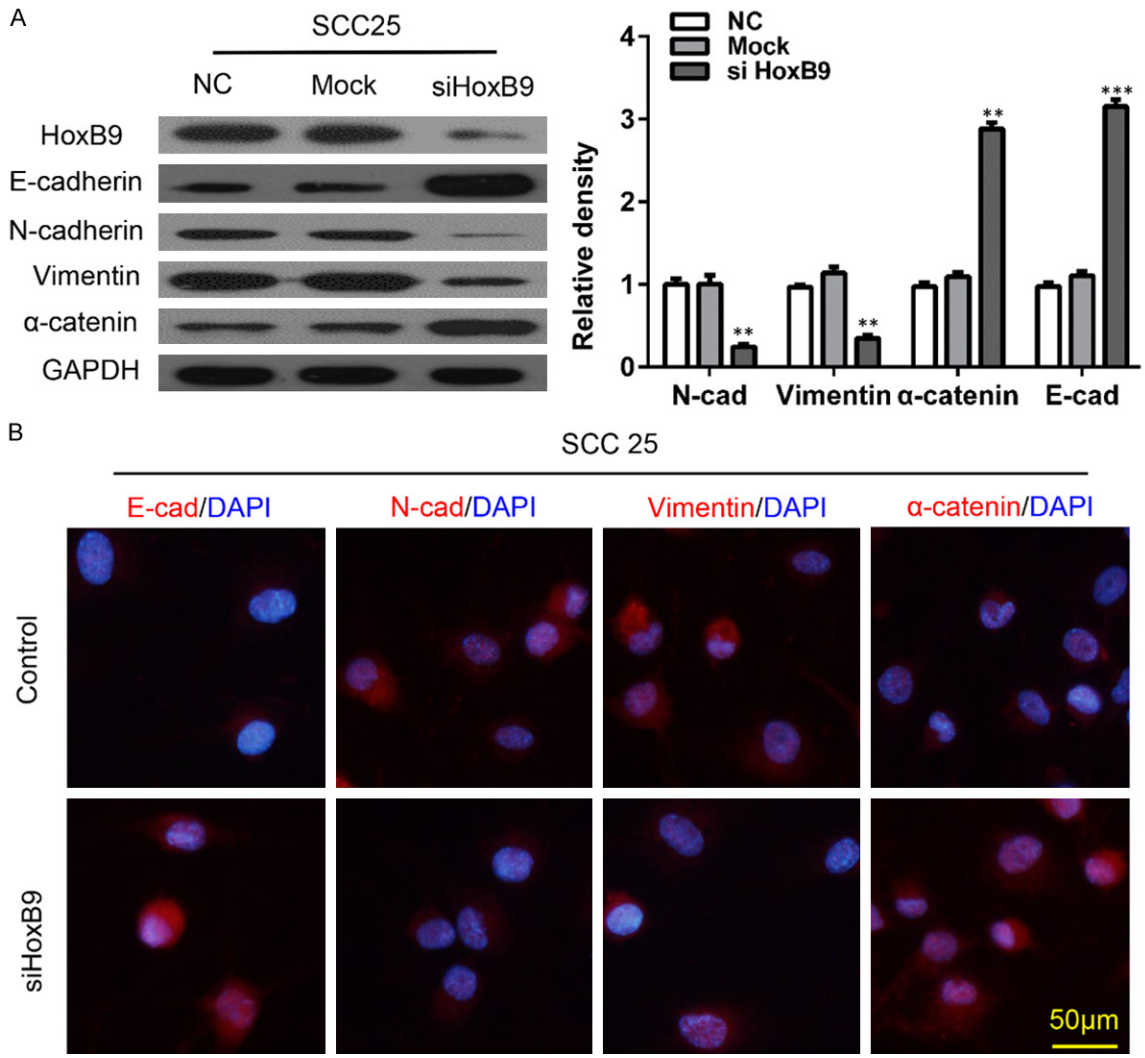
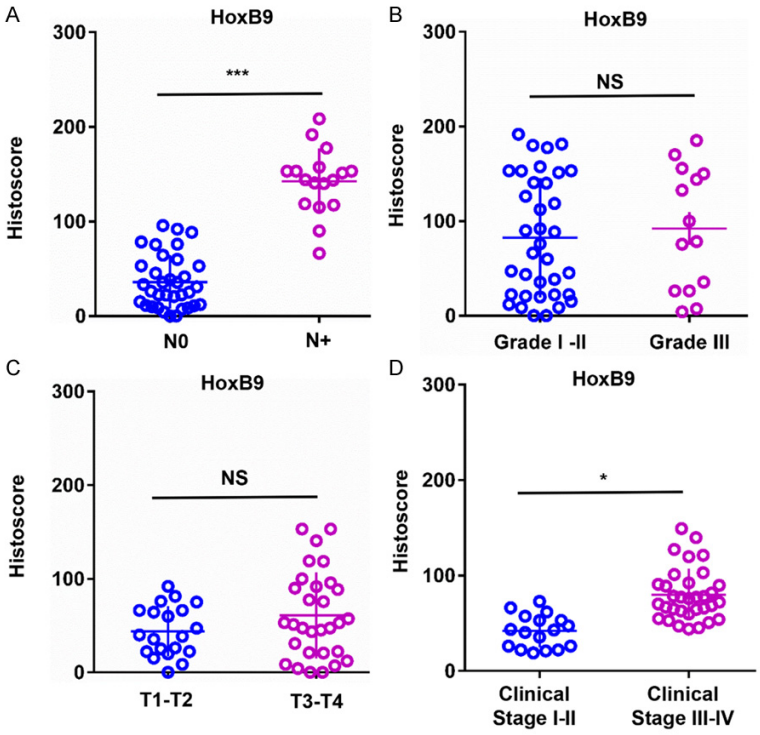


Supplementary Figure 1. Knockdown of HoxB9 decreases migration and invasion of SCC-25. A. Knockdown of HoxB9 by two different siRNA in SCC-25 cell line, GAPDH served as a loading control; Relative density data were calculated by Image J, and the data represented mean of three independent experiments. ***P < 0.001; B. Wound healing assay showed knockdown of HoxB9 suppressed the cell mobility of SCC-25 cell line, and quantification of wound closure shows the statistical significance of the difference (Mean ± SD; **P < 0.01, student t-test with GraphPad Prism 5.0); C. Transwell assay showed the migration abilities of SCC-25 were impaired after knocking down of HoxB9 compared with those of control group, and quantification of cell numbers with Image J "cell counter" module (Mean ± SEM; **P < 0.01, student t-test with GraphPad Prism 5.0 (n = 3).



Supplementary Figure 2. HoxB9 regulates the transition between epithelial and mesenchymal phenotypes in SCC-25 cells. A. SCC-25 cells were treated with siRNA for HoxB9, then the E-cadherin, N-cadherin, Vimentin and α-catenin levels were determined. GAPDH was the internal standard for protein loading. The values are presented as the means ± SEM. One-way ANOVA with post-Dunnett analysis was performed using GraphPad Prism 5.0. **P < 0.01, versus the control group. (n = 3); B. SCC-25 cells were treated with siRNA for HoxB9, the representative immunofluorescence of E-cadherin, N-cadherin, Vimentin and α-catenin were determined (Scale bars = 50 μm).



Supplementary Figure 3. HoxB9 expression in different Grades, T categories and N categories of OSCC. A. HoxB9 expression was correlated with lymph node metastasis of OSCC; B. HoxB9 expression was not correlated with different pathological grades (I-III) of OSCC; C. HoxB9 expression was not correlated with T category of OSCC; D. HoxB9 expression was correlated with clinical stages of OSCC (Mean ± SEM; *P < 0.05, ***P < 0.001, t test).