Figure S1. AMOTL2 inhibits the proliferation, migration and invasion of glioma cells. (A and B) The U251 cell line was transfected with lentiviral vectors of AMOTL2 shRNA and overexpressed plasmids of AMOTL2. Reverse transcription-quantitative PCR and western blotting were performed to examine the AMOTL2 expression level. (C) Colony formation assays were conducted on the U251 cell line to examine the effect of the expression of AMOTL2 in cell colony formation (n=3). (D and E) Transwell migration and Matrigel® invasion assays were conducted to investigate the effect of AMOTL2 knockdown on U251 cell migration and invasion (n=5). (F) The migrating ability of U251 cells was detected by wound healing assay (n=5). Results are presented as the mean  $\pm$  SD. \*P<0.05, \*\*P<0.01, \*\*\*P<0.001 and \*\*\*\*P<0.0001. AMOTL2, angiomotin-like 2; NC, negative control; sh-, short hairpin; oe, overexpression.

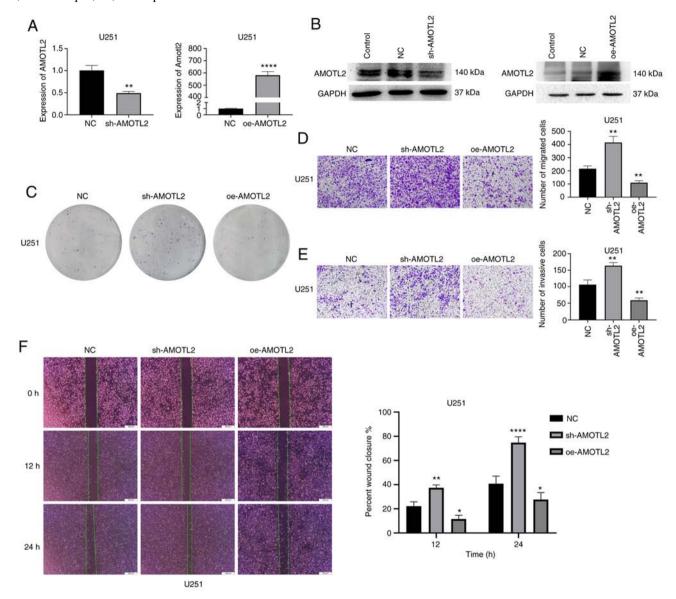


Figure S2. AMOTL2-knockdown can promote the proliferation, migration and invasion of glioma through the Wnt/β-catenin signaling pathway. (A) U251 cells transfected with lentiviral vectors of AMOTL2 shRNA were treated with Wnt pathway inhibitor XAV939 for 24 h. Western blotting was performed to examine the effect of the inhibition. (B) Colony formation assays were conducted on the U251 cell line (n=3). (C) CKK-8 assay was used to detect cell growth in U251 cells at 1, 2, 3, 4, 5 and 6 days (n=3). (D) The migration of U251 cells was detected by wound healing assay (n=5). (E) Transwell migration (upper images) and Matrigel® invasion assays (lower images) were conducted to examine migration and invasion (n=5). Results are presented as mean ± SD. \*\*P<0.01 and \*\*\*P<0.001. AMOTL2, angiomotin-like 2; CKK-8, Cell Counting Kit-8; NC, negative control; sh-, short hairpin; ns, not significant.

